Scientific Report of the 2015 Dietary Guidelines Advisory Committee

Advisory Report to the Secretary of Health and Human Services and the Secretary of Agriculture

First Print
February 2015

Contents

DEPARTMENT OF HEALTH AND HUMAN SERVICES

DEPARTMENT OF AGRICULTURE

January 28, 2015

The Honorable Sylvia Mathews Burwell
Secretary of Health and Human Services
200 Independence Avenue, SW
Washington DC, 20201

The Honorable Thomas J. Vilsack
Secretary of Agriculture
1400 Independence Avenue, SW
Washington DC, 20250

Dear Secretaries Burwell and Vilsack,

It is my great honor to present to you the final Scientific Report of the 2015 Dietary Guidelines Advisory Committee (DGAC). When appointed a year and a half ago, our Committee accepted the charge of examining where sufficient "new scientific evidence is likely to be available that may inform revisions to the current guidance or suggest new guidance." We recognized the importance and key function of the U.S. *Dietary Guidelines* in forming the basis of Federal nutrition policy and programs and in providing a critical framework for local, state, and national health promotion and disease prevention strategies. We also understood the influence of the *Guidelines* in shaping policies, standards, and initiatives across the public and private sectors, including public health and health care, education, business, and the food industry and retailers. As such, we approached our review with a broad scope to address the many issues that may be relevant as the government creates the *2015 Dietary Guidelines for Americans*.

In carrying out our charge, the 2015 DGAC formulated a set of overarching goals. In brief, we planned to determine the current composition and quality of the American diet and areas of public health concern; trends in the Nation's leading diet- and lifestyle-related health problems; the established, measurable impact of overall dietary patterns and physical activity on short- and long-term health outcomes; the most effective methods of improving dietary patterns and physical activity to achieve favorable health outcomes in Americans 2 years and older; and sound strategies to help promote a healthy, safe, affordable, and sustainable food supply. We also were intent on identifying the Nation's major diet- and lifestyle-related health disparities and levels of food insecurity in underserved populations. Recognizing the dynamic interplay between individuals, their families and communities, and the environment, we laid out an ecological, systems-based conceptual framework to guide our deliberative processes and then evaluated almost 100 primary and many ancillary research questions.

Over the past 18 months, the 2015 DGAC was extremely privileged to work with the outstanding Federal support staff of the U.S. Departments of Agriculture and Health and Human Services. We wish to acknowledge these individuals and their invaluable assistance as we developed our Report. We will be forever grateful for their dedication to working with our expert Committee to create the most productive and wonderfully collegial environment for our deliberations. With their extraordinarily capable assistance, we were able to develop a current and sound evidence base using many complex sources, including an abundance of original peer-reviewed literature compiled by USDA's Nutrition Evidence Library and its national network of research volunteers, the national nutrition and health data monitoring systems, the National Health and Nutrition Examination Survey, and the USDA food pattern modeling process.

Our Report highlights the major diet-related health problems we face as a Nation and must reverse. About half of all American adults—117 million individuals—have one or more *preventable* chronic diseases that relate to poor quality dietary patterns and physical inactivity, including cardiovascular diseases, hypertension, type 2 diabetes, and diet-related cancers. More than two-thirds of adults and nearly one-third of children and youth are overweight or obese. These devastating health problems have persisted for decades, strained U.S. health care costs, and focused the attention of our health care system on disease treatment rather than prevention. They call for bold action and sound, innovative solutions.

The dietary patterns of the American public are suboptimal and are causally related to poor individual and population health and higher chronic disease rates. Unfortunately, few improvements in consumers' food choices have occurred in recent decades. On average, the U.S. diet is low in vegetables, fruit, and whole grains and too high in calories, saturated fat, sodium, refined grains, and added sugars. Under-consumption of vitamin D, calcium, potassium, and fiber are of public health concern for the majority of the U.S. population. Furthermore, more than 49 million people in the United States, including nearly 9 million children, live in food insecure households. Creative, evidence-based strategies are needed to reverse these alarming trends.

The economic and social costs of preventable chronic diseases, health disparities, and food insecurity are enormous, and the Nation's adverse dietary pattern and physical activity trends must be reversed. The 2015 DGAC hopes that its Report will aid in developing public policies that aim to establish a "culture of health" at individual and population levels and, in so doing, make healthy lifestyle choices easy, accessible, affordable and normative—both at home and away from home. Dramatic paradigm shifts are needed to help individuals and families take more active roles in their personal health and to incentivize health care and public health services, programs, and research to focus more on prevention and personal diet and lifestyle management. We hope our Report will also lead to public policies that align the public and private sectors on common ground to work collaboratively to develop and offer healthier food products and choices, expanded nutrition programs and services focused on prevention, and greater opportunities for increased physical activity. We urge the development and implementation of nutrition and related policies, standards, programs, and services that promote population-wide healthy dietary patterns and physical activity. Our Report also recommends key research areas where priority attention is needed. That said, the Committee wishes to emphasize that the current evidence base has never been stronger and provides a sound basis to guide the development of public policies and effective nutrition and physical activity interventions to promote health and prevent disease at individual and population levels. Establishing the policy framework to achieve these aims is of paramount importance. We look forward to the translation of this Report into future recommendations in the *2015 Dietary Guidelines for Americans*.

Respectfully and sincerely yours,

Barbara E. Millen

Barbara E. Millen, DrPH, RD, FADA
Chair, 2015 Dietary Guidelines Advisory Committee

2015 Dietary Guidelines Advisory Committee Membership

Chair

Barbara Millen, DrPH, RD
Millennium Prevention
Westwood, MA

Vice Chair

Alice H. Lichtenstein, DSc
Tufts University
Boston, MA

Members

Steven Abrams, MD
Baylor College of Medicine
Houston, TX

Lucile Adams-Campbell, PhD
Georgetown University Medical Center
Washington, DC

Cheryl Anderson, PhD, MPH
University of California, San Diego
La Jolla, CA

J. Thomas Brenna, PhD
Cornell University
Ithaca, NY

Wayne Campbell, PhD
Purdue University
West Lafayette, IN

Steven Clinton, MD, PhD
The Ohio State University
Columbus, OH

Gary Foster, PhD *(May – August 2013)*[i]
Temple University
Philadelphia, PA

Frank Hu, MD, PhD, MPH
Harvard School of Public Health
Boston, MA

Miriam Nelson, PhD
Tufts University
Boston, MA

Marian Neuhouser, PhD, RD
Fred Hutchinson Cancer Research Center
Seattle, WA

Rafael Pérez-Escamilla, PhD
Yale School of Public Health
New Haven, CT

Anna Maria Siega-Riz, PhD, RD
The University of North Carolina at Chapel Hill
Chapel Hill, NC

Mary Story, PhD, RD
Duke University
Durham, NC

[i] Dr. Gary Foster assumed a new position shortly after being appointed as a member of the 2015 DGAC. Due to the significant demands of the new position, it became necessary for Dr. Foster to resign his appointment to the 2015 DGAC in August 2013.

Consultant Subcommittee Members

Timothy S. Griffin, PhD
Tufts University
Boston, MA

Michael W. Hamm, PhD
Michigan State University
East Lansing, MI

Michael G. Perri, PhD, ABPP
University of Florida
Gainesville, FL

Co-Executive Secretaries

Richard Olson, MD, MPH
Designated Federal Officer
Co-Executive Secretary
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Kellie Casavale, PhD, RD
Alternate Designated Federal Officer
Co-Executive Secretary
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Colette Rihane, MS, RD
Lead USDA Co-Executive Secretary
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Shanthy Bowman, PhD
Co-Executive Secretary
Food Surveys Research Group
Agricultural Research Service
USDA

Policy Officials

Don Wright, MD, MPH
Deputy Assistant Secretary for Health
Director
Office of Disease Prevention and Health Promotion
HHS

Angela Tagtow, MS, RD, LD
Executive Director *(beginning July 2014)*
Center for Nutrition Policy and Promotion
USDA

Jackie Haven, MS, RD
Acting Executive Director
(October 2013 to July 2014)
Center for Nutrition Policy and Promotion
USDA

Robert Post, PhD, MEd, MSc
Acting Executive Director
(August to October 2013)
Center for Nutrition Policy and Promotion
USDA

Rajen Anand, DVM, PhD
Executive Director *(February 2013 to July 2013)*
Center for Nutrition Policy and Promotion
USDA

Dietary Guidelines Management Team

Jean Altman, MS
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Patricia Britten, PhD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Janet de Jesus, MS, RD
National Heart, Lung, and Blood Institute
National Institutes of Health
HHS

Rachel Fisher, MS, MPH, RD
Division of Nutrition Research Coordination
National Institutes of Health
HHS

Stephanie Goodwin, PhD, RD
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Patricia Guenther, PhD, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Angela Hutson, RN
LCDR, US Public Health Service
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Kristin Koegel, MBA, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Kevin Kuczynski, MS, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Margaret McDowell, PhD, MPH, RD
Division of Nutrition Research Coordination
National Institutes of Health
HHS

Holly McPeak, MS
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Amber Mosher, MPH, RD
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Katrina Piercy, PhD, RD
LT, US Public Health Service
Division of Prevention Science
Office of Disease Prevention and Health Promotion
HHS

Elizabeth Rahavi, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Eve Essery Stoody, PhD
Nutrition Guidance Team Lead
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Elaine Trujillo, MS, RD
National Cancer Institute
National Institutes of Health
HHS

Essie Yamini, PhD, RD
Center for Food Safety and Applied Nutrition
Food and Drug Administration
HHS

Nutrition Evidence Library Team

Joanne Spahn, MS, RDN, FADA
Nutrition Evidence Library Team Lead
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Jean Altman, MS
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Donna Blum-Kemelor, MS, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Eric Calloway, PhD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Thomas V. Fungwe, PhD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Joan M.G. Lyon, MS, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Patricia Carrera MacNeil, MS, LN, CNS
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Mary M. McGrane, PhD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Julie E. Obbagy, PhD, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Tricia Psota, PhD, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Maureen Spill, PhD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Nancy Terry, MSLS
Division of Library Services
National Institutes of Health
HHS

Yat Ping Wong, MLS, MPH
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Data Analysis Team

Shanthy Bowman, PhD
Data Analysis Team Lead
Food Surveys Research Group
Agricultural Research Service
USDA

Namanjeet Ahluwalia, PhD, DSc, FACN
National Center for Health Statistics
Centers for Disease Control and Prevention
HHS

Patricia Britten, PhD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Kirsten Herrick, MSc, PhD
National Center for Health Statistics
Centers for Disease Control and Prevention
HHS

WenYen Juan, PhD
Center for Food Safety and Applied Nutrition
Food and Drug Administration
HHS

Brian Kit, MD, MPH
National Center for Health Statistics
Centers for Disease Control and Prevention
HHS

Susan M. Krebs-Smith, PhD, MPH, RD
Risk Factor Monitoring and Methods Branch
National Cancer Institute
HHS

Amy Lando, MPP
Center for Food Safety and Applied Nutrition
Food and Drug Administration
HHS

Alanna Moshfegh, MS, RD
Food Surveys Research Group
Agricultural Research Service
USDA

Melissa Nickle, MPH
Nutrient Data Laboratory
Agricultural Research Service
USDA

Cynthia Ogden, PhD, MRP
National Center for Health Statistics
Centers for Disease Control and Prevention
HHS

Pamela Pehrsson, PhD
Nutrient Data Laboratory
Agricultural Research Service
USDA

Jill Reedy, PhD, MPH, RD
Risk Factor Monitoring and Methods Branch
National Cancer Institute
HHS

Donna Rhodes, MS, RD
Food Surveys Research Group
Agricultural Research Service
USDA

Colette Rihane, MS, RD
Office of Nutrition Guidance and Analysis
Center for Nutrition Policy and Promotion
USDA

Jennifer Seymour, PhD
Division of Nutrition, Physical Activity, and Obesity
Centers for Disease Control and Prevention
HHS

Jessica E. Todd, PhD
Economic Research Service
USDA

Paula Trumbo, PhD
Center for Food Safety and Applied Nutrition
Food and Drug Administration
HHS

Science Writer/Editor

Anne Brown Rodgers
Falls Church, Virginia

Part A. Executive Summary

The 2015 Dietary Guidelines Advisory Committee (DGAC) was established jointly by the Secretaries of the U.S. Department of Health and Human Services (HHS) and the U.S. Department of Agriculture (USDA). The Committee was charged with examining the *Dietary Guidelines for Americans*, 2010 to determine topics for which new scientific evidence was likely to be available with the potential to inform the next edition of the Guidelines and to place its primary emphasis on the development of food-based recommendations that are of public health importance for Americans ages 2 years and older published since the last DGAC deliberations.

The 2015 DGAC's work was guided by two fundamental realities. First, about half of all American adults—117 million individuals—have one or more preventable, chronic diseases, and about two-thirds of U.S. adults—nearly 155 million individuals—are overweight or obese. These conditions have been highly prevalent for more than two decades. Poor dietary patterns, overconsumption of calories, and physical inactivity directly contribute to these disorders. Second, individual nutrition and physical activity behaviors and other health-related lifestyle behaviors are strongly influenced by personal, social, organizational, and environmental contexts and systems. Positive changes in individual diet and physical activity behaviors, and in the environmental contexts and systems that affect them, could substantially improve health outcomes.

Recognizing these realities, the Committee developed a conceptual model based on socio-ecological frameworks to guide its work (see ***Part B. Chapter 1: Introduction***) and organized its evidence review to examine current status and trends in food and nutrient intakes, dietary patterns and health outcomes, individual lifestyle behavior change, food and physical activity environments and settings, and food sustainability and safety.

The remainder of this Executive Summary provides brief synopses of the DGAC's topic-specific evidence review chapters. Each of these chapters ends with a list of research recommendations (see ***Appendix E-1: Needs for Future Research*** for a compilation of these recommendations). The Committee integrated its findings and conclusions into several key themes and articulated specific recommendations for how the report's findings can be put into action at the individual, community, and population levels. The Executive Summary ends with a brief summary of this chapter.

TOPIC-SPECIFIC FINDINGS AND CONCLUSIONS

Food and Nutrient Intakes, and Health: Current Status and Trends

The DGAC conducted data analyses to address a series of questions related to the current status and trends in the Nation's dietary intake. The questions focused on: intake of specific nutrients and food groups; food categories (i.e., foods as consumed) that contribute to intake; eating behaviors; and the composition of various dietary patterns shown to have health benefits. These topics were addressed using data from the What We Eat in America dietary survey, which is the dietary intake component of the ongoing National Health and Nutrition Examination Survey. Food pattern modeling using the USDA Food Pattern food groups also was used to address some questions. In addition, the DGAC examined the prevalence and trends of health conditions that may have a nutritional origin, or where the course of disease may be influenced by diet.

The DGAC found that several nutrients are underconsumed relative to the Estimated Average Requirement or Adequate Intake levels set by the Institute of Medicine (IOM) and the Committee characterized these as shortfall nutrients: vitamin A, vitamin D, vitamin E, vitamin C, folate, calcium, magnesium, fiber, and potassium. For adolescent and premenopausal females, iron also is a shortfall nutrient. Of the shortfall nutrients, calcium, vitamin D, fiber, and potassium also are classified as nutrients of public health concern because their underconsumption has been linked in the scientific literature to adverse health outcomes. Iron is included as a shortfall nutrient of public health concern for adolescent females and adult females who are premenopausal due to the increased risk of iron-deficiency in these groups. The DGAC also found that two nutrients—sodium and saturated fat—are overconsumed by the U.S. population relative to the Tolerable Upper Intake Level set by the IOM or other maximal standard and that the overconsumption poses health risks.

In comparison to recommended amounts in the USDA Food Patterns, the majority of the U.S. population has low intakes of key food groups that are important sources of the shortfall nutrients, including vegetables, fruits, whole grains, and dairy. Furthermore, population intake is too high for refined grains and added sugars. The data suggest cautious optimism about dietary intake of the youngest members of the U.S. population because many young children ages 2 to 5 years consume recommended amounts of fruit and dairy. However, a better understanding is needed on how to maintain and encourage good habits that are started early in life. Analysis of data on food categories, such as burgers, sandwiches, mixed dishes, desserts, and beverages, shows that the composition of many of these items could be improved so as to increase population intake of vegetables, whole grains, and other underconsumed food groups and to lower population intake of the nutrients sodium and saturated fat, and the food component refined grains. Improved beverage selections that limit or remove sugar-sweetened beverages and place limits on sweets and desserts would help lower intakes of the food component, added sugars.

The U.S. population purchases its food in a variety of locations, including supermarkets, convenience stores, schools, and the workplace. The DGAC found that although diet quality varies somewhat by the setting where food is obtained, overall, no matter where the food is obtained, the diet quality of the U.S. population does not meet recommendations for vegetables, fruit, dairy, or whole grains, and exceeds recommendations, leading to overconsumption, for the nutrients sodium and saturated fat and the food components refined grains, solid fats, and added sugars.

Obesity and many other health conditions with a nutritional origin are highly prevalent. The Nation must accelerate progress toward reducing the incidence and prevalence of overweight and obesity and chronic disease risk across the U.S. population throughout the lifespan and reduce the disparities in obesity and chronic disease rates that exist in the United States for certain ethnic and racial groups and for those with lower incomes.

The DGAC had enough descriptive information from existing research and data to model three dietary patterns and to examine their nutritional adequacy. These patterns are the Healthy U.S.-style Pattern, the Healthy Mediterranean-style Pattern, and the Healthy Vegetarian Pattern. These patterns include the components of a dietary pattern associated with health benefits.

Dietary Patterns, Foods and Nutrients, and Health Outcomes

A major goal of the DGAC was to describe the common characteristics of healthy diets, and the Committee focused on research examining dietary patterns because the totality of diet—the combinations and quantities in which foods and nutrients are consumed—may have synergistic and cumulative effects on health and disease. The Committee focused on providing a qualitative description of healthy dietary patterns based on scientific evidence for several health outcomes.

The DGAC found remarkable consistency in the findings and implications across its conclusion statements for the questions examining dietary patterns and various health outcomes. When reviewing the evidence, the Committee attempted to adhere to the language used by the study authors in describing food groupings. There was variability across the food groupings, and this was particularly apparent in the meat group. For example, "total meat" may have been defined as "meat, sausage, fish, and eggs," "red meat, processed meat, and poultry," or various other combinations of meat. Similarly, "vegetables" seemed to most often exclude potatoes, but some studies included potatoes, yet those that mentioned potatoes rarely provided information on how the potatoes were consumed (e.g., fried versus baked). When reported in the studies, the Committee considered these definitions in their review. However, the Committee provided a general label for the food groupings in its conclusion statements.

The overall body of evidence examined by the 2015 DGAC identifies that a healthy dietary pattern is higher in vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes, and nuts; moderate in alcohol (among adults); lower in red and processed meat;[1] and low in sugar-sweetened foods and drinks and refined grains. Vegetables and fruit are the only characteristics of the diet that were consistently identified in every conclusion statement across the health outcomes. Whole grains were identified slightly less consistently compared to vegetables and fruits, but were identified in every conclusion with moderate to strong evidence. For studies with limited evidence, grains were not as consistently defined and/or they were not identified as a key characteristic. Low- or non-fat dairy, seafood, legumes, nuts, and alcohol were identified as beneficial characteristics of the diet for some, but not all, outcomes. For conclusions with moderate to strong evidence, higher intake of red and processed meats was identified as detrimental compared to lower intake. Higher consumption of sugar-sweetened foods and beverages as well as refined grains was identified as detrimental in almost all conclusion statements with moderate to strong evidence.

Regarding alcohol, the Committee confirmed several conclusions of the 2010 DGAC, including that moderate alcohol intake can be a component of a healthy dietary pattern, and that if alcohol is consumed, it should be consumed in moderation and only by adults. However, it is not recommended that anyone begin drinking or drink more frequently on the basis of potential health benefits, because moderate alcohol intake also is associated with increased risk of violence, drowning, and injuries from falls and motor vehicle crashes. Women should be aware of a moderately increased risk of breast cancer even with moderate alcohol intake. In addition, there are many circumstances in which people should not drink alcohol, including during pregnancy. Because of the substantial evidence clearly demonstrating the health benefits of breastfeeding, occasionally consuming an alcoholic drink does not warrant stopping breastfeeding. However, women who are breastfeeding should be very cautious about drinking alcohol, if they choose to drink at all.

Following a dietary pattern associated with reduced risk of CVD, overweight, and obesity also will have positive health benefits beyond these categories of health outcomes. Thus, the U.S. population should be encouraged and guided to consume dietary patterns that are rich in vegetables, fruit, whole grains, seafood, legumes, and nuts; moderate in low- and non-fat dairy products and alcohol (among adults); lower in red and processed meat; and low in sugar-sweetened foods and beverages and refined grains. These dietary patterns can be achieved in many ways and should be tailored to the individual's biological and medical needs as well as socio-cultural preferences.

[1] As lean meats were not consistently defined or handled similarly between studies, they were not identified as a common characteristic across the reviews. However, as demonstrated in the food pattern modeling of the Healthy U.S.-style and Healthy Mediterranean-style patterns, lean meats can be a part of a healthy dietary pattern.

The dietary pattern characteristics being recommended by the 2015 DGAC reaffirm the dietary pattern characteristics recommended by the 2010 DGAC. Additionally, these characteristics align with recommendations from other groups, including the American Institute for Cancer Research (AICR) and the American Heart Association (AHA). The majority of evidence considered by the Committee focused on dietary patterns consumed in adulthood. Very little evidence examined dietary patterns during childhood. However, the healthy dietary pattern components described above also apply to children and are reaffirmed with the USDA Food Patterns, which are designed to meet nutrient needs across the lifespan.

Individual Diet and Physical Activity Behavior Change

The individual is at the innermost core of the social-ecological model. In order for policy recommendations such as the *Dietary Guidelines for Americans* to be fully implemented, motivating and facilitating behavioral change at the individual level is required. This chapter suggests a number of promising behavior change strategies that can be used to favorably affect a range of health-related outcomes and to enhance the effectiveness of interventions. These include reducing screen time, reducing the frequency of eating out at fast food restaurants, increasing frequency of family shared meals, and self-monitoring of diet and body weight as well as effective food labeling to target healthy food choices. These strategies complement comprehensive lifestyle interventions and nutrition counseling by qualified nutrition professionals.

For this approach to work, it will be essential that the food environments in communities available to the U.S. population, particularly to low-income individuals, facilitate access to healthy and affordable food choices that respect their cultural preferences. Similarly, food and calorie label education should be designed to be understood by audiences with low health literacy, some of which may have additional English language fluency limitations. Although viable approaches are available now, additional research is necessary to improve the scientific foundation for more effective guidelines on individual-level behavior change for all individuals living in the United States, taking into account the social, economic, and cultural environments in which they live.

The evidence reviewed in this chapter also indicates that the social, economic, and cultural context in which individuals live may facilitate or hinder their ability to choose and consume dietary patterns that are consistent with the Dietary Guidelines. Specifically, household food insecurity hinders the access to healthy diets for millions of Americans. In addition, immigrants are at high risk of losing the healthier dietary patterns characteristic of their cultural background as they acculturate into mainstream America. Furthermore, preventive nutrition services that take into account the social determinants of health are largely unavailable in the U.S. health system to systematically address nutrition-related health problems, including overweight and obesity, cardiovascular disease, type 2 diabetes, and other health outcomes.

This chapter calls for: a) stronger Federal policies to help prevent household food insecurity and to help families to cope with food insecurity if it develops, b) food and nutrition assistance programs to take into account the risk that immigrants have of giving up their healthier dietary habits soon after arriving in the United States, and c) efforts to provide all individuals living in the United States with the environments, knowledge, and tools needed to implement effective individual- or family-level behavioral change strategies to improve the quality of their diets and reduce sedentary behaviors. These goals will require changes at all levels of the social-ecological model through coordinated efforts among health care and social and food systems from the national to the local level.

Food Environment and Settings

Environmental and policy approaches are needed to complement individual-based efforts to improve diet and reduce obesity and other diet-related chronic diseases. These approaches have the potential for broad and sustained impact at the population level because they can become incorporated into organizational structures and systems and lead to alterations in sociocultural and societal norms. Both policy and environmental changes also can help reduce disparities by improving access to and availability of healthy food in underserved neighborhoods and communities. Federal nutrition assistance programs, in particular, play a vital role in achieving this objective through access to affordable foods that help millions of Americans meet Dietary Guidelines recommendations.

The DGAC focused on physical environments (settings) in which food is available. Its aim was to better understand the impact of the food environment to promote or hinder healthy eating in these settings and to identify the most effective evidence-based diet-related approaches and policies to improve diet and weight status. The DGAC focused on four settings—community food access, child care, schools, and worksites—and their relationships to dietary intake and quality and weight status.

The DGAC found moderate and promising evidence that multi-component obesity prevention approaches implemented in child care settings, schools, and worksites improve weight-related outcomes; strong to moderate evidence that school and worksite policies are associated with improved dietary intake; and moderate evidence that multi-component school-based and worksite approaches increase vegetable and fruit consumption. For the questions on community food access addressing the relationship between food retail settings and dietary intake and quality and weight status, the evidence was too limited or insufficient to assign grades. To reduce the disparity gaps that currently exist in low resource and underserved communities, more solution-oriented strategies need to be implemented and evaluated on ways to increase access to and procurement of healthy affordable foods and beverages, and also to reduce access to energy-dense, nutrient-poor foods and beverages. Although several innovative approaches are taking place now throughout the country, they generally lack adequate evaluation efforts.

The Committee's findings revealed the power of multi-component approaches over single component interventions. For obesity prevention, effective multi-component interventions incorporated both nutrition and physical activity using a variety of strategies, such as environmental policies to improve the availability and provision of healthy foods and beverages; increasing opportunities for physical activity; increased parent engagement (in child care and school settings); and educational approaches, such as a school nutrition curriculum. For multi-component dietary interventions (e.g., to increase consumption of vegetables and fruit) the most effective strategies included nutrition education, parent engagement (in school and child care settings), and environmental modifications (e.g., policies for nutrition standards, food service changes, point of purchase information).

Collaborative partnerships and strategic efforts are needed to translate this evidence into action. Further work on restructuring the environment to facilitate healthy eating and physical activity, especially in high risk populations, is needed to advance evidence-based solutions that can be scaled up.

Food Sustainability and Safety

Access to sufficient, nutritious, and safe food is an essential element of food security for the U.S. population. A sustainable diet ensures this access for both the current population and future generations.

The major findings regarding sustainable diets were that a diet higher in plant-based foods, such as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in calories and animal-based foods is more health promoting and is associated with less environmental impact than is the current U.S. diet. This pattern of eating can be achieved through a variety of dietary patterns, including the Healthy U.S.-style Pattern, the Healthy Mediterranean-style Pattern, and the Healthy Vegetarian Pattern. All of these dietary patterns are aligned with lower environmental impacts and provide options that can be adopted by the U.S. population. Current evidence shows that the average U.S. diet has a larger environmental impact in terms of increased greenhouse gas emissions, land use, water use, and energy use, compared to the above dietary patterns. This is because the current U.S. population intake of animal-based foods is higher and plant-based foods are lower, than proposed in these three dietary patterns. Of note is that no food groups need to be eliminated completely to improve sustainability outcomes over the current status.

A moderate amount of seafood is an important component of two of three of these dietary patterns, and has demonstrated health benefits. The seafood industry is in the midst of rapid expansion to meet worldwide demand. The collapse of some fisheries due to overfishing in the past decades has raised concern about the ability to produce a safe and affordable supply. In addition, concern has been raised about the safety and nutrient content of farm-raised versus wild-caught seafood. To supply enough seafood to support meeting dietary recommendations, both farm-raised and wild caught seafood will be needed. The review of the evidence demonstrated, in the species evaluated, that farm-raised seafood has as much or more EPA and DHA per serving as wild caught. It should be noted that low-trophic seafood, such as catfish and crawfish, regardless of whether wild caught or farm-raised seafood, have less EPA and DHA per serving than high-trophic seafood, such as salmon and trout.

Regarding contaminants, for the majority of wild caught and farmed species, neither the risks of mercury nor organic pollutants outweigh the health benefits of seafood consumption. Consistent evidence demonstrated that wild caught fisheries that have been managed sustainably have remained stable over the past several decades; however, wild caught fisheries are fully exploited and their continuing productivity will require careful management nationally and internationally to avoid long-term collapse. Expanded supply of seafood nationally and internationally will depend upon the increase of farm-raised seafood worldwide.

The impact of food production, processing, and consumption on environmental sustainability is an area of research that is rapidly evolving. As further research is conducted and best practices are evaluated, additional evidence will inform both supply-side participants and consumers on how best to shift behaviors locally, nationally, and globally to support sustainable diets. Linking health, dietary guidance, and the environment will promote human health and the sustainability of natural resources and ensure current and long-term food security.

In regard to food safety, updated and previously unexamined areas of food safety were studied. Currently, strong evidence shows that consumption of coffee within the moderate range (3 to 5 cups per day or up to 400 mg/d caffeine) is not associated with increased long-term health risks among healthy individuals. In fact, consistent evidence indicates that coffee consumption is associated with reduced risk of type 2 diabetes and cardiovascular disease in adults. Moreover, moderate evidence shows a protective association between caffeine intake and risk of Parkinson's disease. Therefore, moderate coffee consumption can be incorporated into a healthy dietary pattern, along with other healthful behaviors. However, it should be noted that coffee as it is normally consumed can contain added calories from cream, milk, and added sugars. Care should be taken to minimize the amount of calories from added sugars and high-fat dairy or dairy substitutes added to coffee.

The marketing and availability of high-caffeine beverages and products is on the rise. Unfortunately, only limited evidence is currently available to ascertain the safety of high caffeine intake (greater than 400 mg/day for adults and undetermined for children and adolescents) that may occur with rapid consumption of large-sized energy drinks. Limited data suggest adverse health outcomes, such as caffeine toxicity and cardiovascular events. Concern is heightened when caffeine is combined with alcoholic beverages. Limited or no consumption of high caffeine drinks, or other products with high amounts of caffeine, is advised for children and adolescents. Energy drinks with high levels of caffeine and alcoholic beverages should not be consumed together, either mixed together or consumed at the same sitting.

The DGAC also examined the food additive aspartame. At the level that the U.S. population consumes aspartame, it appears to be safe. However, some uncertainty continues about increased risk of hematopoietic cancer in men, indicating a need for more research.

Individual behaviors along with sound government policies and responsible private sector practices are all needed to reduce foodborne illnesses. To that end, the DGAC updated the established recommendations for handling foods at home.

Cross-cutting Topics of Public Health Importance

The *2010 Dietary Guidelines* included guidance on sodium, saturated fat, and added sugars, and the 2015 DGAC determined that a reexamination of the evidence on these topics was necessary to determine whether revisions to the guidance were warranted. These topics were considered to be of public health importance because each has been associated with negative health outcomes when overconsumed. Additionally, the Committee acknowledged that a potential unintended consequence of a recommendation on added sugars might be that consumers and manufacturers replace added sugars with low-calorie sweeteners. As a result, the Committee also examined evidence on low-calorie sweeteners to inform statements on this topic.

The DGAC encourages the consumption of healthy dietary patterns that are low in saturated fat, added sugars, and sodium. The goals for the general population are: less than 2,300 mg dietary sodium per day (or age-appropriate Dietary Reference Intake amount), less than 10 percent of total calories from saturated fat per day, and a maximum of 10 percent of total calories from added sugars per day.

Sodium, saturated fat, and added sugars are not intended to be reduced in isolation, but as a part of a healthy dietary pattern that is balanced, as appropriate, in calories. Rather than focusing purely on reduction, emphasis should also be placed on replacement and shifts in food intake and eating patterns. Sources of saturated fat should be replaced with unsaturated fat, particularly polyunsaturated fatty acids. Similarly, added sugars should be reduced in the diet and not replaced with low-calorie sweeteners, but rather with healthy options, such as water in place of sugar-sweetened beverages. For sodium, emphasis should be placed on expanding industry efforts to reduce the sodium content of foods and helping consumers understand how to flavor unsalted foods with spices and herbs.

Reducing sodium, saturated fat, and added sugars can be accomplished and is more attainable by eating a healthy dietary pattern. For all three of these components of the diet, policies and programs at local, state, and national levels in both the private and public sector are necessary to support reduction efforts. Similarly, the Committee supports efforts in labeling and other campaigns to increase consumer awareness and understanding of sodium, saturated fats, and added sugars in foods and beverages. The Committee encourages the food industry to continue reformulating and making changes to certain foods to improve their nutrition profile. Examples of such actions include lowering sodium and added sugars content, achieving better saturated fat to polyunsaturated fat ratio, and reducing portion sizes in retail settings (restaurants, food outlets, and public venues, such as professional sports stadiums and arenas). The Committee also encourages the food industry to market these improved products to consumers.

Physical Activity

This chapter provides strong evidence supporting the importance of regular physical activity for health promotion and disease prevention in the U.S. population. Physical activity is important for all people—children, adolescents, adults, older adults, women during pregnancy and the postpartum period, and individuals with disabilities. The findings further provide guidance on the dose of physical activity needed across the lifecycle to realize these significant health benefits.

Future Physical Activity Guidelines Advisory Committees will be asked to carefully review the most recent evidence so that the Federal government can fully update the *2008 Physical Activity Guidelines for Americans*. Given the exceedingly low physical activity participation rates in this country, it will be critically important for the next Committee to identify proven strategies and approaches to increase population-level physical activity across the lifespan.

INTEGRATING THE EVIDENCE

The research base reviewed by the 2015 DGAC provides clear evidence that persistent, prevalent, preventable health problems, notably overweight and obesity, cardiovascular disease, type 2 diabetes, and certain cancers, have adversely affected the health of the U.S. public for decades and raise the urgency for immediate attention and bold action. Evidence points to specific areas of current food and nutrient concerns and it pinpoints the characteristics of healthy dietary and physical activity patterns that can reduce chronic disease risk, promote healthy weight status, and foster good health across the lifespan. In addition, research evidence is converging to show that healthy dietary patterns also are more sustainable and associated with more favorable health as well as environmental outcomes.

Effective models of "what works" to promote lifestyle behavior change exist. While they can be improved, especially in terms of our capacity for scaling-up in community and health care settings, the evidence to date can be used to guide programs and services for individuals and families. They also can be used to assist the public and private sectors and communities in facilitating innovative environmental change to promote the population's health.

It will take concerted, bold actions on the part of individuals, families, communities, industry, and government to achieve and maintain the healthy diet patterns and the levels of physical activity needed to promote the health of the U.S. population. These actions will require a paradigm shift to an environment in which population health is a national priority and where individuals and organizations, private business, and communities work together to achieve a population-wide "culture of health" in which healthy lifestyle choices are easy, accessible, affordable, and normative—both at home and away from home. In such a culture, health care and public health professionals also would embrace a new leadership role in prevention, convey the importance of lifestyle behavior change to their patients/clients, set standards for prevention in their own facilities, and help patients/clients in accessing evidence-based and effective nutrition and comprehensive lifestyle services and programs.

Part B. Chapter 1: Introduction

The *Dietary Guidelines for Americans* were first released in 1980, and since that time they have provided science-based advice on promoting health and reducing risk of major chronic diseases through a healthy* diet and regular physical activity. Early editions of the Dietary Guidelines focused specifically on healthy members of the public, but more recent editions also have included those who are at increased risk of chronic disease. Future editions will continue to evolve to address public health concerns and the nutrition needs of specific populations. For example, the Dietary Guidelines have traditionally targeted the general public older than age 2 years, but as data continue to accumulate regarding the importance of dietary intake during gestation and from birth on, a Federal initiative has been established to develop comprehensive guidance for infants and toddlers from birth to 24 months and women who are pregnant. By 2020, the *Dietary Guidelines for Americans* will include these important populations comprehensively.

By law (Public Law 101-445, Title III, 7 U.S.C. 5301 et seq.) the *Dietary Guidelines for Americans* is published by the Federal government every 5 years. To meet this requirement, since the 1985 edition, the Departments have jointly appointed a Dietary Guidelines Advisory Committee of nationally recognized experts in the field of nutrition and health to review the scientific and medical knowledge current at the time. The 2015 Dietary Guidelines Advisory Committee (DGAC) was established for the single, time-limited task of reviewing the 2010 edition of *Dietary Guidelines for Americans* and developing nutrition and related health recommendations to the Federal government for its subsequent development of the 2015 edition. This report presents these recommendations to the Secretaries of Health and Human Services and of Agriculture for use in updating the Guidelines.

The 2015 DGAC recognizes the importance and key function of the Guidelines in forming the basis of Federal nutrition policy and programs. The Guidelines also provides a critical framework for local, state, and national health promotion and disease prevention initiatives. In addition, it provides evidence-based nutrition and physical activity strategies for use by individuals and those who serve them in public and private settings, including public health and social service agencies, health care and educational institutions, and business. The food industry and retailers as well, can use the Guidelines to develop healthy food and beverage products and offerings for consumers.

* Throughout this report, the term "healthy" is used to represent the concept of "health-promoting" as well as to refer to foods or dietary patterns that are consistent with the Dietary Guidelines. See the Glossary for a definition of "health."

The potential for the Guidelines to inform policy and practice is critical, given the significant nutrition-related health issues facing the U.S. population:

- **Overweight, obesity, and other diet-related chronic diseases** (particularly cardiovascular diseases, type 2 diabetes, and certain cancers), as well as less common but important health outcomes, such as bone health, for which nutrition plays an important role. These conditions are prevalent across the entire U.S. population, but are more pronounced in low-income populations, creating critical health disparities that must be addressed.

- **Less than optimal dietary patterns in the United States**, which contribute directly to poor population health and high chronic disease risk. On average, current dietary patterns are too low in vegetables, fruit, whole grains, and low-fat dairy, and too high in refined grains, saturated fat, added sugars, and sodium.

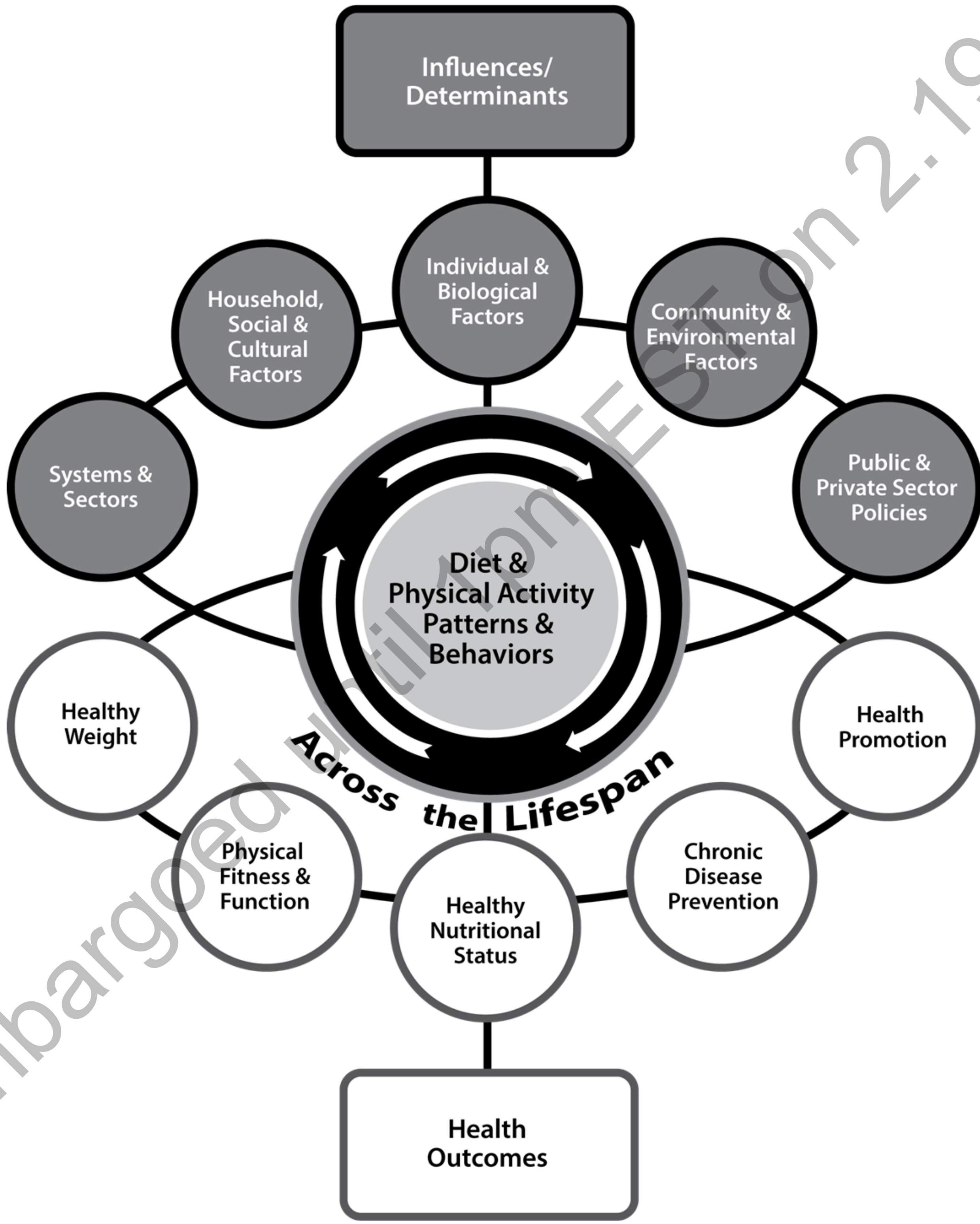
- **Food insecurity**, a condition in which the availability of nutritionally adequate foods, or the ability to acquire acceptable foods in socially acceptable ways, is limited or uncertain. More than 49 million people in the United States, including nearly 9 million children, live in food insecure households.

The economic and social costs of obesity and other diet- and physical activity-related chronic disease conditions are enormous and will continue to escalate if current trends are not reversed. Therefore, improving diet and physical activity in the population and addressing food insecurity and health disparities have great potential to not only reduce the burden of chronic disease morbidity and mortality, but also to reduce health care costs.

The DGAC recognized that a dynamic interplay exists among individuals' nutrition, physical activity, and other health-related lifestyle behaviors and their environmental and social contexts. Acknowledging this, the DGAC created a conceptual model based in part on the socio-ecological model to serve as an organizing framework for its report (Figure B2.1). The figure shows how these personal, social, organizational, and environmental contexts and systems interact powerfully to influence individuals' diet and physical activity behaviors and patterns and how diverse health outcomes result from this dynamic interplay. An accompanying table expands on the figure by listing specific factors that comprise each of the "Determinants" and "Outcomes" circles. The table distinguishes those factors that are addressed in the DGAC report from related factors that are important but beyond the scope of the report (see Table B2.1 at the end of this chapter).

Figure B2.1

Diet and Physical Activity, Health Promotion and Disease Prevention at Individual and Population Levels across the Lifespan



REVIEWING THE EVIDENCE

Drawing from this conceptual model, the 2015 DGAC reviewed an extensive and diverse body of scientific literature to address many research questions. For each of its questions, the Committee used a rigorous, evidence-based process to develop its findings. Some of the resulting evidence was strong to moderate, and some was found to be evolving and more limited. This graded evidence was used to draw scientific conclusion and implication statements and to make recommendations that can be used by HHS and USDA in formulating the *Dietary Guidelines for Americans* policy document.

The DGAC used the findings from its evidence reviews to develop a series of chapters that build on and complement each other:

- **Chapter 1** examines current status and trends in food consumption, nutrient intakes, and eating behaviors and rates and patterns of major nutrition-related health problems. It identifies the nutrients of public health concern and characterizes several dietary patterns that are consistent with those associated with positive health outcomes.
- **Chapter 2** considers relationships between dietary patterns and health outcomes and identifies a number of commonalities across patterns, particularly food groups, associated with positive health outcomes. It examines these relationships for major chronic diseases (cardiovascular diseases, type 2 diabetes, overweight and obesity, and certain cancers), and also evaluates several less common, but important, outcomes (bone health, neurological and psychological illnesses, congenital anomalies). Where possible, evidence on the impact of dietary or comprehensive lifestyle interventions (including diet, physical activity, and behavioral strategies) in reducing chronic disease risk outcomes is summarized and can be used to inform health promotion and disease prevention strategies at individual and population levels.
- **Chapter 3** reviews characteristics associated with individual dietary and lifestyle behaviors, such as meal patterns at home and away from home, acculturation, household food insecurity, and sedentary behaviors. It also assesses methods that are effective in helping individuals improve their diet and physical activity behaviors and in enhancing behavioral interventions.
- **Chapter 4** assesses the roles of food environments and settings in promoting or hindering healthy eating behaviors of specific population groups (such as pre-school and school-age children and adults in the workplace) and evaluates evidence on effective methods and best practices to promote population behavior change in communities as well as public and private settings to influence and improve health.
- **Chapter 5** focuses on secure and sustainable diets by examining how dietary guidance and food intake influence our capacity to meet the nutrition needs of the U.S. population now and

in the future. The chapter also examines issues related to food safety behaviors in the home environment and evaluates new topics of food safety concern, including the safety of coffee/caffeine and aspartame.

- **Chapter 6** considers topics of continuing public health importance that are relevant for topics across Chapters 1 through 5 and, are therefore addressed together in this chapter—sodium, saturated fat, added sugars, and low-calorie sweeteners.
- **Chapter 7** discusses the important role that physical activity plays in promoting health.

FROM THE 2015 DGAC ADVISORY REPORT TO THE *DIETARY GUIDELINES FOR AMERICANS*

A major goal of the 2015 DGAC is to summarize and synthesize the evidence to support USDA and HHS in developing nutrition recommendations that reduce the risk of chronic disease while meeting nutrient requirements and promoting health of the U.S. population ages 2 years and older.

The U.S. Government uses the Dietary Guidelines as the basis of its food assistance programs, nutrition education efforts, and decisions about national health objectives. For example, the National School Lunch Program and the Elder Nutrition Program incorporate the Dietary Guidelines in menu planning; the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC) applies the Dietary Guidelines in its educational materials; and the *Healthy People 2020* objectives for the Nation include objectives based on the Dietary Guidelines.

The evidence described here in the 2015 DGAC Report, which will be used to develop the 2015 *Dietary Guidelines for Americans*, will help policymakers, educators, clinicians, and others speak with one voice on nutrition and health and reduce the confusion caused by mixed messages in the media. The DGAC hopes that the 2015 *Dietary Guidelines for Americans* will encourage the food industry and retailers to grow, manufacture, and sell foods that promote health and contribute to appropriate energy balance.

In reviewing the evidence on effective interventions and best practices at individual and population levels, the 2015 DGAC hopes that the *2015 Dietary Guidelines for Americans* will also lead to the bold actions needed to transform our health care and public health systems, communities, and businesses. A concerted and collaborative focus on prevention is needed and the report provides a foundation of research evidence to help create a national "culture of health" where healthy lifestyles are easier to achieve and normative. Finally, the 2015 DGAC desires that its evidence on healthy dietary patterns, which have been found to be important in reducing disease risk and in promoting food security and sustainability in the near- and long-term, will lead to changes in individual eating behaviors and to systems-wide changes that can help to secure a healthy future for the U.S. population.

A GUIDE TO THE 2015 DGAC REPORT

This Report contains several major sections. Part A provides an Executive Summary to the Report. Part B sets the stage for the Report through this Introduction. A second chapter in this section provides an integration of major findings as well as specific recommendations for how the Report's evidence-based dietary recommendations can be put into action at the individual, community, and population levels.

Part C describes the methodology the DGAC used to conduct its work and review the evidence on diet and health. Part D is the Science Base and contains the chapters described above.

The Report concludes with a number of Appendices, including a compilation of the Committee's research recommendations; several appendices describing sources of evidence the Committee used in its reviews; a glossary; a brief history of the *Dietary Guidelines for Americans*; a summary of the process used to collect public comments; biographical sketches of DGAC members; a list of DGAC Working Group, Subcommittee, and Working/Writing Group members; and Acknowledgments.

Table B2.1: Components of the Conceptual Model

Influences/Determinants

	Factors	Addressed in the DGAC report	Other factors <u>not</u> addressed in the DGAC report
	**Individual & Biological Factors** (Represented in the model by characteristics of individuals and their physical makeup that influence lifestyle behaviors)		
	Biological factors	physical and cognitive function; clinical health and nutritional status profile; weight status	appetite, taste and smell acuity; hunger; physical, mental, and emotional well-being; digestion and metabolism; microbiome composition; genetic profile; prescribed medication use; drug-nutrient interactions
	Nutrition, physical activity, and health-related factors	food label use; dietary or physical activity self-monitoring; personal lifestyle profile characteristics including diet, physical activity, and lifestyle behaviors and practices	early diet experiences; perception of food safety and food security; access to nutrition and preventative health counseling; experiences with personal lifestyle behavior change
	Psychological factors	mental health	self/body image; food, nutrition, and health attitudes, beliefs, and preferences; motivation and intentions; self-efficacy; coping skills; mood; stress
	Demographics	age, gender, race/ethnicity, acculturation, income, geography/region, urban/rural location of residence	education, household composition and culture, religion, profession/occupation

	**Household, Social & Cultural Factors** (Represented in the model by structure, resources, values and norms that influence lifestyle behaviors)		
	Family/household/home	parenting and lifestyle behavioral modeling; food and beverage availability; cooking and storage facilities; family and shared meals; physical activity resources	living situation, composition, person(s) responsible for food purchases/preparation; home food environment
	Social/cultural/religious/ peer networks	engagement and participation in lifestyle and health-related programs and initiatives	beliefs, norms, values, expectations, and information sharing
	Society and culture		values and investments that support healthy communities and reduce health disparities; stewardship of natural resources and healthy environments
	**Community & Environmental Factors** (Represented in the model by physical and structural characteristics and facilities that provide access to and affect the quality of resources that influence lifestyle behaviors)		
	Food and physical activity	types of available retail food outlets, restaurants, food banks, and farmers' markets; safety, quality and sustainability of available food supplies; patterns of food waste	recreational facilities and resources
	Community	neighborhood food access; child care, schools, and worksites	composition, structure and conditions; social capital and networks; trust and power; disparities and inequities in food security, health, healthcare access, after school programs
	Business/Workplace	corporate/worksite wellness policies and programs, nutrition, exercise and health	employee benefits programs

		services, programs and resources	
	Health care and public health	providers and programs that emphasize lifestyle behavior change, health promotion and disease prevention; accessibility of clinical preventive services including nutrition counseling	health insurance benefits and access including preventative lifestyle services; food and nutrition assistance policies and programming; public and private healthcare networks and infrastructure
	Physical/built/natural environment		green spaces, parks, and recreational resources: availability and access; land use and transportation; abandoned buildings/spaces; soil contamination; chemical, fertilizer, antibiotic and pesticide use
	Ecosystems (national to global)	the natural environment, including farmland; plant, animal, marine, land, and water ecosystems; renewable energy resources; land/water/air and soil environments and quality; plant conservation, biodiversity; greenhouse gas emissions, pollution/contamination	plant and natural resources management and conservation; carbon footprint; global climate change
	**Systems & Sectors** (Represented in the model by spheres of influence on food availability and diet and physical activity behavior)		
	Consumer		acquisition, consumption, and demand; use, experience and satisfaction
	Retail and service		products, programs, markets; organization and management
	Food, beverage, and agriculture	usual and high levels of caffeine intake; aspartame	farming; import/export; production, processing, storage, distribution, delivery; supply/markets; food and beverage quality

			and safety; food technology and product formulation; advertising; food marketing
	Economy	income	employment; inflation and recession; social, political and human capital; productivity; prices of food
	Other	technology: mobile health (mHealth)	research and technology; emerging trends; entertainment; advertising and marketing; leisure and recreation; media and social media; globalization of trade
	**Public & Private Sector Policies** (Represented in the model by policies, regulations and laws that influence the availability and quality of products, resources, programs and services that influence diet and physical activity behaviors)		
	Government	federal, state and local food and nutrition assistance programs and/or initiatives that promoting physical activity/movement (e.g. NSLP, SBP, elder nutrition); city and town policies (e.g. taxation, bans, food assistance, price incentives); food and beverage labels	policies, laws and regulations that affect agriculture, food safety and food assistance; educational institutions; employers and worksites; healthcare systems and health insurance
	Business/Workplace	workplace policies on nutrition and physical activity programs, services and resources	employee health benefits (including health insurance) and incentives
	Education and social services across the lifespan	policies, laws and regulations that affect food and beverage availability including competitive foods; nutrition and physical activity programs and services (e.g. in childcare, school, elder care and community settings); food, nutrition, and physical activity services in federal, state	

		and local food assistance settings	

The central portion of the Conceptual Model represents the concept that the combination of a healthy diet and regular physical activity behaviors and patterns is central to promoting overall health and preventing many chronic diseases.

Health Outcomes

	Factors	Addressed in the DGAC report	Other factors <u>not</u> addressed in the DGAC report
	**Healthy Weight** (Represented in the model by measures that characterize a health-promoting weight status)		
	Weight and body composition	childhood and adolescence length/height, weight and Z scores, body weight and weight gain, BMI, waist circumference, abdominal obesity, lean and body fat mass; overweight and obesity	
	**Physical Fitness & Function** (Represented in the model by activities that define a health-promoting level of physical fitness and function)		
	Physical activity and function patterns and behaviors	Aerobic and strengthening activities; occupational, work, and leisure time activity	ability to perform activities of daily living; muscle strength; coordination; falls; physical activity knowledge, awareness and skills
	Sedentary behaviors and sleep patterns	screen time and other sedentary behaviors	sleep patterns (sleep duration, characteristics)

Healthy Nutritional Status	**Healthy Nutritional Status** (Represented in the model by the knowledge, behaviors, environmental factors and measures that characterize healthy nutritional status)		
	Dietary patterns	habitual food and nutrient consumption; overall dietary quality and variety	
	Food, beverage and nutrition intake	foods/food groups, beverages (including alcohol), and macro and micronutrients, nutrients of concern and public health significance	
	Dietary product and nutrient supplement use	dietary product and nutrient supplement use	nutraceutical use
	Food and nutrition knowledge, attitudes and skills	food preparation, cooking and nutrition knowledge, attitudes and skills	
	Food security and safety	selection, storage, handling, and preparation of foods and beverages	
	Risk factors and clinical indicators	iron and protein status, vitamin D and folate levels, Vitamin B12 status, hemoglobin A1c; metabolic syndrome (blood lipids and glucose, blood pressure); bone density	urinary sodium, urinary contaminants; protein/calorie malnutrition; micronutrient status

Chronic Disease Prevention	**Chronic Disease Prevention** (Represented in the model by health outcomes influenced by diet and physical activity behaviors)		
	Health outcomes	cardiovascular diseases (coronary heart disease, heart attack, hypertension and stroke); Type 2 diabetes; diet-related cancers (breast, colorectal, prostate, lung); neurological and psychological conditions (including cognitive function, dementia, Alzheimer's Disease and depression); dental caries; congenital anomalies; fractures and osteoporosis; total mortality	
Health Promotion	**Health Promotion** (Represented in the model by diet and physical activity behaviors that promote good health through the lifespan)		
	Health outcomes	pregnancy course and outcomes; child and adolescent growth and development milestones; peri- and post-menopause status; musculoskeletal and bone health; mental health; gastrointestinal health	fertility; healthy aging

Footnote: The DGAC acknowledges that other lifestyle factors were not addressed in its report but are important in overall health, including tobacco status and use, stress and its management, medical treatment and management, medication use, and addiction.

Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence

The 2015 DGAC set out to examine a broad set of research questions in its effort to develop sound recommendations to guide public policies aimed at promoting individual and population health. As these efforts moved forward, it became clear that a number of important, overarching themes were emerging and that these areas provided a solid base of evidence for the Committee's recommendations. In this chapter, we summarize these themes and put forth our overall recommendations to the Secretaries of Health and Human Services and Agriculture.

DGAC 2015 OVERARCHING THEMES

- **The Problem.** About half of all American adults—117 million individuals—have one or more preventable, chronic diseases that are related to poor quality dietary patterns and physical inactivity, including cardiovascular disease, hypertension, type 2 diabetes and diet-related cancers.[1] More than two-thirds of adults and nearly one-third of children and youth are overweight or obese, further exacerbating poor health profiles and increasing risks for chronic diseases and their co-morbidities.[2,3] High chronic disease rates and elevated population disease risk profiles have persisted for more than two decades and disproportionately affect low-income and underserved communities. These diseases focus the attention of the U.S. health care system on disease treatment rather than prevention; increase already strained health care costs; and reduce overall population health, quality of life, and national productivity. Other less common, but important, diet- and lifestyle-related health problems, including poor bone health and certain neuropsychological disorders and congenital anomalies, pose further serious concerns.

- **The Gap.** The dietary patterns of the American public are suboptimal and are causally related to poor individual and population health and higher chronic disease rates. Few, if any, improvements in consumers' food choices have been seen in recent decades. On average, the U.S. diet is low in vegetables, fruit, and whole grains, and high in sodium, calories, saturated fat, refined grains, and added sugars. Underconsumption of the essential nutrients vitamin D, calcium, potassium, and fiber are public health concerns for the majority of the U.S. population, and iron intake is of concern among adolescents and premenopausal females. Health disparities exist in population access to affordable healthy foods. Eating behaviors of individuals are shaped by complex but modifiable factors, including individual, personal, household, social/cultural, community/environmental, systems/sectorial and policy-level factors (see the 2015 DGAC conceptual model in ***Part B. Chapter 1: Introduction***). However, a dynamic and rapidly evolving food environment epitomized by the abundance of highly processed, convenient, lower-cost, energy-dense, nutrient-poor foods makes it particularly challenging to implement health promoting diet-related behavior changes at individual and population levels.

- **The Dietary Patterns.** Current research provides evidence of moderate to strong links between healthy dietary patterns, lower risks of obesity and chronic diseases, particularly cardiovascular disease, hypertension, type 2 diabetes and certain cancers. Emerging evidence also suggests that relationships may exist between dietary patterns and some neurocognitive disorders and congenital anomalies. ***The overall body of evidence examined by the 2015 DGAC identifies that a healthy dietary pattern is higher in vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes, and nuts; moderate in alcohol (among adults); lower in red and processed meats;[i] and low in sugar-sweetened foods and drinks and refined grains.*** Additional strong evidence shows that it is not necessary to eliminate food groups or conform to a single dietary pattern to achieve healthy dietary patterns. Rather, individuals can combine foods in a variety of flexible ways to achieve healthy dietary patterns, and these strategies should be tailored to meet the individual's health needs, dietary preferences and cultural traditions. Current research also strongly demonstrates that regular physical activity promotes health and reduces chronic disease risk.

- **The Individual.** Sound tools and resources, like the *Dietary Guidelines for Americans* and the *Physical Activity Guidelines for Americans*, can help individuals achieve healthy diet and physical activity patterns. Moderate to strong evidence also demonstrates that dietary interventions implemented by nutrition professionals and individual or small-group comprehensive lifestyle interventions that target diet and physical activity and are led by multidisciplinary professional teams provide optimal results in chronic disease risk reduction, weight loss, and weight loss maintenance. Additional evidence indicates that individuals can be helped in their intentions to implement healthy lifestyles by targeting specific eating and physical activity behaviors (e.g., meal patterns, cooking and preparation techniques, family/household meal experiences, reducing sedentary behaviors in adults and youth, reducing screen time in children). Sound behavioral interventions involve engaging individuals actively in the behavior change process, using traditional face-to-face or small group strategies and new technological approaches (websites and mobile/telephone technology), by providing intensive, long-term professional interventions as appropriate, and by monitoring and offering feedback on sustainable behavioral change and maintenance strategies over time.

- **The Population.** Moderate to strong evidence shows that targeted environmental and policy changes and standards are effective in changing diet and physical activity behaviors and achieving positive health impact in children, adolescents, and adults. Research from early child care settings, schools, and worksites demonstrate that policy changes, particularly when

---

[i] As lean meats were not consistently defined or handled similarly between studies, they were not identified as a common characteristic across the reviews. However, as demonstrated in the food pattern modeling of the Healthy U.S.-style and Healthy Mediterranean-style patterns, lean meats can be a part of a healthy dietary pattern.

combined with multi-faceted programs (e.g., nutrition educational initiatives, parent engagement, food labeling, nutrition standards, nutrition and behavioral intervention services) can increase healthy food choices and overall dietary quality, and improve weight outcomes. Population approaches that engage parents and families, as appropriate, involve collaborations across systems and sectors (e.g., schools, food retail, health care institutions and providers, and health insurers), and mobilize public-private partnerships to provide effective models for producing synergistic effects on diet, physical activity, and health-related outcomes.

- **The Long-term View.** The 2015 DGAC also examined the near- and long-term sustainability of healthy dietary patterns as well as the safety of certain key dietary constituents (i.e., caffeine and aspartame). Quantitative modeling research showed how healthy dietary patterns relate to positive environmental outcomes that improve population food security. Moderate to strong evidence demonstrates that healthy dietary patterns that are higher in plant-based foods, such as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in calories and animal-based foods are associated with more favorable environmental outcomes (lower greenhouse gas emissions and more favorable land, water, and energy use) than are current U.S. dietary patterns. Furthermore, sustainable dietary patterns can be achieved through a variety of approaches consistent with the *Dietary Guidelines for Americans* and, therefore, offer individuals many options and new opportunities to align with personal and population health and environmental values systems. Healthy, sustainable dietary patterns also may provide new themes for consumer education and communication on lifestyle practices that can promote food security now and for future generations and create a "culture of health" at individual and population levels.

In summary, the research base reviewed by the 2015 DGAC provides clear and consistent evidence that persistent, prevalent, preventable health problems, notably overweight and obesity, cardiovascular diseases, diabetes, and certain cancers, have severely and adversely affected the health of the U.S. population across all stages of the lifespan for decades and raise the urgency for immediate attention and bold action. Evidence points to specific areas of food and nutrient concern in the current U.S. diet. Moderate to strong evidence pinpoints the characteristics of healthy dietary and physical activity patterns established to reduce chronic disease risk, prevent and better manage overweight and obesity, and promote health and well-being across the lifespan.

Although behavior change is complex, moderate to strong evidence now points to effective strategies to promote healthy lifestyle behavior changes at individual and population levels. This overall research evidence base can be used to inform policy changes, multi-sectorial collaborations, as well as product/service reformulation as needed. It can be used with confidence to provide guidelines and standards for nutrition and lifestyle intervention services/programs in traditional health care and public health settings. It also provides frameworks for public and private sector initiatives and community programming to make innovative environmental changes that can change population diet and physical activity behaviors to promote population health.

Overall, the evidence base on the links between diet, physical activity, and health has never been as strong or more compelling. The strength of evidence on "what works" to improve individual and population lifestyle behaviors for health also has never been more robust, with solutions and models of "best practices." Furthermore, the increasing convergence of research evidence showing that healthy dietary patterns not only reduce disease risks and improve health outcomes but are associated with food security and sustainability provide a further, convincing rationale for focused attention on prevention and individual and population health promotion. Additional research must be conducted to strengthen this evidence base, and recommendations for such research are made in each of the chapters in ***Part D. Science Base*** (see ***Appendix E-1: Needs for Future Research*** for a compilation of the DGAC's research recommendations).

DGAC 2015 RECOMMENDATIONS FOR ACTION

It will take concerted, bold action on the part of individuals, families, communities, industry, and government to achieve and maintain healthy dietary patterns and the levels of physical activity needed to promote a healthy U.S. population.

This will entail dramatic paradigm shifts in which population health is a national priority and individuals, communities, and the public and private sectors seek together to achieve a population-wide "culture of health" through which healthy lifestyle choices are easy, accessible, affordable and normative—both at home and away from home. In such a culture, preventing diet- and physical activity-related diseases and health problems would be much more highly valued, the resources and services needed to achieve and maintain health would become a realized human right across all population strata, the needs and preferences of the individual would be seriously considered, and individuals and their families/households would be actively engaged in promoting their personal health and managing their preventive health services and activities. Health care and public health professionals would embrace a new leadership role in prevention, convey the importance of lifestyle behavior change to their patients/clients, set model standards for prevention-oriented activities and client/employee services in their own facilities, and manage patient/client referrals to evidence-based nutrition and comprehensive lifestyle services and programs. Communities and relevant sectors of our economy, including food, agriculture, private business, health care (as well as insurance), public health and education, would seek common ground and collaborations in promoting population health. Initiatives would be incentivized to engage communities and health care systems to create integrated and comprehensive approaches to preventing chronic diseases and for weight management. Environmental changes, including policy changes, improved food and beverage standards, reformulation of products and services as needed, and programs that enhance population lifestyle behavior changes and support preventive services also would be incentivized.

Although these propositions are extremely challenging, it is imperative to seek novel and creative, evidence-based solutions. The costs of failing to do so are the continuation of the very high rates of preventable diet- and physical activity-related health problems we confront as a Nation and the worsening of their serious adverse effects on our quality of life, population productivity, and already highly strained healthcare costs. The evidence base has never been stronger to guide solutions. What is needed are strong commitments and leadership, the development of targeted public and private policies and partnerships, and the implementation of evidence-based, cross-sectorial initiatives to achieve them. In the remainder of this chapter, the DGAC summarizes specific recommendations guided by our conceptual model, which is grounded in the socio-ecological theory model of individual and population lifestyle behavior change for health promotion and disease prevention (see ***Part B. Chapter 1: Introduction***).

Actions for Individuals and Families/Households

- Think prevention, know your lifestyle-related health risk profile, make personal goals and commitments, and take action to promote personal and household/family health. Work with health professionals to assess and monitor your health risks and to personalize your preventive lifestyle behavior plan of action.
- Know and understand how to modify your diet and physical activity to reduce personal and family member health risks. Know your current dietary pattern, including your healthy choices that can be maintained as well as areas for potential change. Act on this information. Seek to make gradual and sustainable changes in your dietary behaviors to achieve one of several sound healthy dietary pattern options (e.g., Healthy U.S.-style Pattern, the Healthy Mediterranean-style Pattern, or the Healthy Vegetarian Pattern; see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends)***. For most people, this will mean:
    - Improving food and menu choices, modifying recipes (including mixed dishes and sandwiches), and watching portion sizes.
    - Including more vegetables (without added salt or fat), fruits (without added sugars), whole grains, seafood, nuts, legumes, low/non-fat dairy or dairy alternatives (without added sugars).
    - Reducing consumption of red and processed meat, refined grains, added sugars, sodium, and saturated fat; substituting saturated fats with polyunsaturated alternatives; and replacing solid animal fats with non-tropical vegetable oils and nuts.

- The 2015 DGAC advocates achieving healthy dietary patterns through healthy food and beverage choices rather than with nutrient or dietary supplements except as needed.
- Use available Dietary Guidelines for Americans tools and other sound resources to initiate positive personal lifestyle changes to improve dietary and physical activity behaviors, including goal setting and self-monitoring.
    - As needed, seek regular advice from qualified health care providers to establish a personalized plan for prevention that includes steps to adopt healthy dietary patterns and physical activity. As appropriate, engage with nutrition and health professionals to address personal health risks that can be lowered with sound diet and physical activity, or participate in comprehensive lifestyle interventions conducted by trained interventionists (registered dietitians/nutritionists, exercise and behavioral specialists).
    - Achieve and maintain a healthy weight. Know your level of obesity risk. Know your energy needs and how they change with varying levels of physical activity. Take personal action for obesity prevention or weight loss management, as needed, using sound, evidence-based tools and resources. Seek to achieve a dietary pattern consistent with the *Dietary Guidelines for Americans*, recognizing that many evidence-based options can facilitate weight loss and weight loss maintenance. As appropriate, work with qualified nutrition professionals and health providers to create a personalized plan of action for obesity prevention. When needed, engage in intensive, long-term nutrition counseling or comprehensive lifestyle intervention strategies to achieve maximal, long-term weight loss and weight maintenance results.
    - Ensure at home and in public settings, such as schools and early child care programs, that young children achieve a high-quality dietary pattern and level of physical activity. Encourage their active participation in food experiences and activity choices so that the importance of dietary quality and physical activity are reinforced, and healthy lifestyle behaviors become normative, habitual, and easier to maintain through adolescence and lifelong.
    - Follow on a regular basis, the *Physical Activity Guidelines for Americans*. Engage in at least 2.5 hours a week of moderate-intensity aerobic physical activity, such as brisk walking, or 1.25 hours a week of vigorous-intensity aerobic physical activity. For weight control, at least 1 hour a day of moderate- to vigorous-intensity physical activity may be required. Engage children in at least 1 hour a day of moderate- to vigorous-intensity physical activity each day. Limit children’s screen time to no more than two hours per day. Adults should limit sedentary activity and replace it with aerobic and strengthening exercises. As needed, engage with qualified professionals in comprehensive lifestyle interventions to achieve maximal impact on healthy dietary and physical activity patterns and health outcomes. Get enough sleep!

- Seek and demand the creation and maintenance of food and physical activity environments and resources in your community and in local public, private and retail settings so as to promote a "culture of health." These are strongly needed to facilitate the ease of initiating and meeting the U.S. Dietary Guidelines recommendations at home and away from home.

Actions for Communities and Populations

- Aim to make healthy lifestyles and prevention a national and local priority and reality.
  - Create public and private policy changes at the national level that direct and incentivize collaborations by multiple sectors of influence, including health care, public health, education, food and agriculture, transportation, food retail, the media, non-governmental organizations, and service sectors.
  - Incentivize the development of policies and initiatives at local, state, and Federal levels that are carried out using cross-sectorial collaborations to promote individual healthy lifestyle behavior changes and create community "cultures of health." These may include improvements in built and physical environments to create safe and accessible resources and settings for increased physical activity and more widely available healthy food choices. They may entail changes in policies, standards, and practices in retail, and public and private settings and programs that promote "cultures of health" and facilitate the initiation and maintenance of healthy lifestyle behaviors at individual and community levels.
- Seek a paradigm shift in health care and public health toward a greater focus on prevention and integration with food systems.
  - Incentivize and support nutrition professionals, health care providers, and other qualified professionals in their unique roles of encouraging and counseling patients and clients to adopt healthy dietary and physical activity and in offering evidence-based nutrition services and comprehensive lifestyle interventions. Integrate preventive lifestyle screening, referral, and interventions and services for weight management and chronic disease risk reduction into routine practice guidelines and quality assurance standards.
  - Support health care facilities, such as hospitals and clinics, in seeking to model prevention and achieving "cultures of health" by offering healthy food choices for patients, visitors, and staff; implementing preventive nutrition services and comprehensive lifestyle intervention programs; and making referrals to Federal and local food assistance programs as needed by their staff and clients.
  - Require health insurance providers to use financial and other positive incentives to encourage and motivate health care settings and businesses to support individuals in

adopting healthy behaviors and engaging, as appropriate, in nutrition and exercise counseling and comprehensive lifestyle behavior interventions.

  - Encourage and incentivize health care innovations and community prevention through Affordable Care Act (ACA) policies and programs, including expanding preventive lifestyle services in traditional health services environments and new retail health services environments that link to Federal and local food assistance programs. These should provide resources for individuals to engage and sustain personal lifestyle behavior change. In addition, ACA programs and policies should increase access to qualified professionals and programs and services that promote healthy diet and physical activity behaviors.
  - Incentivize businesses to establish employee health benefits plans that include access to resources and services that encourage personal health promotion and healthy lifestyle behavior changes. Support employers in using positive motivation strategies to realize these changes.

- Establish healthy food environments.
  - Establish local, state, and Federal policies to make healthy foods accessible and affordable and to limit access to high-calorie, nutrient-poor foods and sugar-sweetened beverages in public buildings and facilities. Set nutrition standards for foods and beverages offered in public places. Improve retail food environments and make healthy foods accessible and affordable in underserved neighborhoods and communities.
  - Develop and expand programs that encourage healthy eating and physical activity habits in young children and adolescents within school and early care and other education settings. Establish and implement policies and programs that provide nutritious foods, limit sugar-sweetened beverages and other unhealthy foods, incorporate nutrition curricula and experiences and physical activity opportunities, and increase provider and teacher skills to develop and promote these programs.
  - Implement the comprehensive school meal guidelines (National School Lunch Program) from the USDA that increase intakes of vegetables (without added salt), fruits (without added sugars), and whole grains; limit sodium, added sugars, saturated fat, and trans fat; limit marketing unhealthy foods to children; make drinking water freely available to students throughout the day; ensure competitive foods meet the national nutrition standards (e.g., *Dietary Guidelines for Americans*); and eliminate sugar-sweetened beverages.
  - Improve, standardize and implement Nutrition Facts labels and Front of Package labels to help consumers, including those with low literacy levels, make healthy food choices. The Nutrition Facts label should include added sugars (in grams and teaspoons) and include a

percent daily value, to assist consumers in identifying the amount of added sugars in foods and beverages and making informed dietary decisions. Standardize and create easy-to-understand front-of-package (FOP) label on all food and beverage products to give clear guidance about a food's healthfulness. An example is the FOP label recommended by the Institute of Medicine, which included calories, and 0 to 3 "nutritional" points for added sugars, saturated fat, and sodium. This would be integrated with the Nutrition Facts label, allowing consumers to quickly and easily identify nutrients of concern for overconsumption, in order to make healthy choices.

- Align nutritional and agricultural policies with Dietary Guidelines recommendations and make broad policy changes to transform the food system so as to promote population health, including the use of economic and taxing policies to encourage the production and consumption of healthy foods and to reduce unhealthy foods. For example, earmark tax revenues from sugar-sweetened beverages, snack foods and desserts high in calories, added sugars, or sodium, and other less healthy foods for nutrition education initiatives and obesity prevention programs.
- Align food assistance programs such as SNAP and WIC with the *Dietary Guidelines for Americans*. Provide standards for purchasing that create new demands for healthy foods, such as vegetables and fruits, and discourage the purchase and consumption of foods, such as sugar-sweetened beverages. Support research to explore ways to improve overall diet quality in Federal and local food assistance programs.
- Support changes to the food environment that can help individuals make healthy choices in the foods they consume away from home and those they purchase away from home to consume at home. For example, the Committee encourages the food industry to continue to reformulate and make changes to improve the nutrition profile of certain foods. Examples of such actions include lowering sodium and added sugars content, achieving better saturated fat to polyunsaturated fat ratio, and reducing portion sizes in retail settings (restaurants, food outlets, and public venues, such as professional sports stadiums and arenas). The Committee also encourages the food industry to market these improved products to consumers.
- Implement policies and programs at local, state and national levels in both the public and private sectors to reduce added sugars and sodium in foods, limit availability of sugar-sweetened beverages, and promote healthy snacks. Approaches might include:
    - Making water a preferred beverage choice. Encourage water as a preferred beverage when thirsty. Make water accessible in public settings, child care facilities, schools, worksites and other community places where beverages are offered.
    - Reducing added sugars in foods and sugar-sweetened beverages in school meals.

        - Making "smart snacks" consistent with the Dietary Guidelines in schools, child care settings, parks, recreation centers, sports leagues, after-school programs, worksites, colleges and universities, healthcare, and other community settings.
        - Implementing policies that limit exposure and marketing of foods and beverages high in added sugars and sodium to all age groups, particularly children and adolescents.
        - Implementing economic and pricing approaches to promote the purchase of healthy foods and beverages. For example, taxation on higher sugar-and sodium-containing foods may encourage consumers to reduce consumption and revenues generated could support health promotion efforts. Alternatively, price incentives on vegetables and fruits could be used to promote consumption and public health benefits.
        - Mounting public education campaigns to increase the public's awareness of the health effects of excess added sugars, sodium, saturated fat, and calories.

- Support and expand access to healthy built environments and advocate wide community use.
    - Increase opportunities for regular public engagement in physical activity through improved urban and community designs, enhanced community built environments, business spaces, and transportation networks. Urban and community designs should encourage and promote active transportation, such as walking and biking. Green corridors can increase public safety and enhance active transportation.
    - Incentivize communities to make physical activity accessible, affordable, and safe. Encourage public and private sectors to work together to increase access to gyms, bike trails, pedestrian walkways, ball fields, and other recreation areas in the communities. Promote physical activity through social media, smart phone, and other technologies.
    - Reach out to and engage groups such as new immigrant communities who may abandon their native healthy lifestyle habits and others at highest nutritional and health risk, to ensure that they learn about resources and are motivated to access, engage in, and sustain healthy dietary patterns and physical activities within their cultural preferences.
- Maintain strong support for Federal food and nutrition programs.
    - Recognize their importance in creating demand for healthy food products as well as in shaping and modeling consumer behaviors relating to healthy dietary and physical activity patterns.
    - Align program standards with the *Dietary Guidelines for Americans* so as to achieve the 2015 DGAC recommendations and promote a "culture of health."
- Recognize and place priority on moving toward a more sustainable diet consistent with the healthy dietary pattern options described in this DGAC report. Access to sufficient, nutritious, and safe food is an essential element of food security for the U.S. population. A

sustainable diet helps ensure this access for both the current population and future generations.

- Enhance what is already being done by the private and public sectors to improve environmental policies and practices around production, processing, and distribution *within* individual food categories.
- Align local, state, and national practices and policies across sectors to promote a sustainable and safe food supply to ensure long-term food security. Support robust private and public sector partnerships, practices, and policies across the supply chain and extending from farms to distribution and consumption that can incentivize actions to develop a food system that embraces a core set of values that embody healthy, safe, and sustainable dietary patterns. Monitor, evaluate, and reward sectors that do this. Establish new, well-coordinated policies that include, but are not limited to, agriculture, economics, transportation, energy, water use, and dietary guidance. Encourage all participants in the food system, as they are central to creating and supporting sustainable and safe diets.
- Shift toward a greater emphasis on healthy dietary patterns and an improved environmental profile across food categories to maximize environmental sustainability, including encouraging consumption of a variety of wild caught or farmed seafood.
- Improve the nutrient profiles of certain farmed seafood species, particularly EPA and DHA levels, through improved feeding and processing systems and preserve the favorable nutrient profiles of other seafood. Establish strong policy, research, and stewardship to improve the environmental sustainability of farmed seafood systems.
- Offer consumer-friendly information that facilitates understanding the environmental impact of different foods in food and menu labeling initiatives.
- Recognize the importance of foodborne illness prevention and encourage consumer behavior consistent with the four food safety principles described in the *Dietary Guidelines for Americans*—Clean, Separate, Cook, and Chill, which are the foundation of the Fight BAC!® campaign (www.fightbac.org).

REFERENCES

1. Ward BW, Schiller JS, Goodman RA. Multiple chronic conditions among US adults: a 2012 update. *Prev Chronic Dis.* 2014;11:130389. DOI: http://dx.doi.org/10.5888/pcd11.130389.
2. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice

Guidelines. J Am Coll Cardiol. 2014;63(25 Pt B):2960-84. PMID: 24239922. http://www.ncbi.nlm.nih.gov/pubmed/24239922.

3. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. J Am Coll Cardiol. 2014;63(25 Pt B):2985-3023. PMID: 24239920. http://www.ncbi.nlm.nih.gov/pubmed/24239920.

Part C. Methodology

COMMITTEE APPOINTMENT

Beginning with the 1985 edition, the U.S. Department of Agriculture (USDA) and U.S. Department of Health and Human Services (HHS) have appointed a Dietary Guidelines Advisory Committee (DGAC) of nationally recognized experts in the field of nutrition and health to review the scientific evidence and medical knowledge current at the time. This Committee has been an effective mechanism for obtaining a comprehensive and systematic review of the science which contributes to successful Federal implementation as well as broad public acceptance of the Dietary Guidelines. The 2015 DGAC was established for the single, time-limited task of reviewing the 2010 edition of *Dietary Guidelines for Americans* and developing nutrition and related health recommendations in this Advisory Report to the Secretaries of USDA and HHS. The Committee was disbanded upon delivery of this report.

Nominations were sought from the public through a Federal Register notice published on October 26, 2012. Criteria for nominating prospective members of the DGAC included knowledge about current scientific research in human nutrition and chronic disease, familiarity with the purpose, communication, and application of the Dietary Guidelines, and demonstrated interest in the public's health and well-being through their research and educational endeavors. They also were expected to be respected and published experts in their fields. Expertise was sought in several specialty areas, including, but not limited to, the prevention of chronic diseases (e.g., cancer, cardiovascular disease, type 2 diabetes, overweight and obesity, and osteoporosis); energy balance (including physical activity); epidemiology; food processing science, safety, and technology; general medicine; gerontology; nutrient bioavailability; nutrition biochemistry and physiology; nutrition education and behavior change; pediatrics; maternal/gestational nutrition; public health; and/or nutrition-related systematic review methodology.

The Secretaries of USDA and HHS jointly appointed individuals for membership to the 2015 DGAC. The chosen individuals are highly respected by their peers for their depth and breadth of scientific knowledge of the relationship between dietary intake and health in all relevant areas of the current Dietary Guidelines.

To ensure that recommendations of the Committee took into account the needs of the diverse groups served by USDA and HHS, membership included, to the extent practicable, a diverse group of individuals with representation from various geographic locations, racial and ethnic groups, women, and persons with disabilities. Equal opportunity practices, in line with USDA and HHS policies, were followed in all membership appointments to the Committee. Appointments were made without discrimination on the basis of age, race and ethnicity, gender, sexual orientation, disability, or cultural, religious, or socioeconomic status. Individuals were appointed to serve as members of the Committee to represent balanced viewpoints of the scientific evidence, and not to represent the viewpoints of any specific group. Members of the DGAC were classified as Special Government Employees (SGEs) during their term of appointment, and as such were subject to the ethical standards of conduct for all federal employees.

CHARGE TO THE 2015 DIETARY GUIDELINES ADVISORY COMMITTEE

The Dietary Guidelines for Americans provide science-based advice on how nutrition and physical activity can help promote health across the lifespan and reduce the risk for major chronic diseases in the U.S. population ages 2 years and older.

The Dietary Guidelines form the basis of Federal nutrition policy, standards, programs, and education for the general public and are published jointly by HHS and USDA every 5 years. The charge to the Dietary Guidelines Advisory Committee, whose duties were time-limited and solely advisory in nature, was described in the Committee's charter as follows:

- Examine the *Dietary Guidelines for Americans*, 2010 and determine topics for which new scientific evidence is likely to be available that may inform revisions to the current guidance or suggest new guidance.
- Place its primary focus on the systematic review and analysis of the evidence published since the last DGAC deliberations.
- Place its primary emphasis on the development of food-based recommendations that are of public health importance for Americans ages 2 years and older.
- Prepare and submit to the Secretaries of HHS and USDA a report of technical recommendations with rationales, to inform the development of the *2015 Dietary Guidelines for Americans*. DGAC responsibilities included providing authorship for this report; however, responsibilities did not include translating the recommendations into policy or into communication and outreach documents or programs.
- Disband upon the submittal of the Committee's recommendations, contained in the Report of the Dietary Guidelines Advisory Committee on the *Dietary Guidelines for Americans*, 2015 to the Secretaries.
- Complete all work within the 2-year charter timeframe.

THE COMMITTEE PROCESS

Committee Membership

Fifteen members were appointed to the Committee, one of whom resigned within the first 3 months of appointment due to new professional obligations (see the ***DGAC Membership***). The Committee served without pay and worked under the regulations of the Federal Advisory Committee Act (FACA). The Committee held seven public meetings over the course of 1½ years. Meetings were held in June 2013 and January, March, July, September, November, and December 2014. The members met in person on the campus of the National Institutes of Health in Bethesda, Maryland, for six of the seven meetings. The Committee met by webinar for the November 2014 meeting. All meetings were made publically available live by webcast. In addition, members of the general public were able to attend the Committee's first two meetings in person in Washington DC area. For the remaining meetings, members of the public were able to observe by webcast. All meetings were announced in the *Federal Register*. Meeting summaries, presentations, archived recordings of all of the meetings, and other documents pertaining to Committee deliberations were made available at www.DietaryGuidelines.gov. Meeting materials also were provided at the reference desks of the HHS National Institutes of Health.

Public Comments

Written public comments were received throughout the Committee's deliberations through an electronic database and provided to the Committee. This database allowed for the generation of public comment reports as a result of a query by key topic area(s). A general description of the types of comments received and the process used for collecting public comments is described in ***Appendix E-7. Public Comments***.

DGAC Conceptual Model

Recognizing the dynamic interplay that exists among the determinants and influences on diet and physical activity as well as the myriad resulting health outcomes, the Committee developed a conceptual model to complement its work. The Committee began by reviewing the socio-ecological model in the 2010 *Dietary Guidelines for Americans* and identified the primary goals of the new model: 1) characterize the multiple interrelated determinants of complex nutrition and lifestyle behaviors and health outcomes at individual and population levels, and 2) highlight those areas within this large system that are addressed by the 2015 DGAC review of the evidence. In addition, the Committee sought to develop a model that provided an organizing framework to show readers how the Science Base chapters in this report relate to each other and to the larger food and agriculture, nutrition, physical activity, and health systems in the United States. It first developed an outline that identified a large number of factors and highlighted a select number to be addressed in its evidence reviews of this report. A smaller group of Committee members then developed a draft visual approach for conveying the main messages within a conceptual model. Using the structure of that draft visual, the content of the outline was organized into a supplementary table. The draft outline, resulting visual, and supporting table went through review and input by the members at several stages. The resulting conceptual model and supporting table are found in ***Part B. Chapter 1: Introduction***.

Approaches to Reviewing the Evidence

The Committee used a variety of scientifically rigorous approaches to address its science-based questions, and some questions were addressed using multiple approaches. The Committee used the state-of-the-art methodology, systematic reviews, to address 27 percent of its science-based research questions. These reviews are publically available in the Nutrition Evidence Library (NEL) at www.NEL.gov. The scientific community now regularly uses systematic review methodologies, so, unlike the 2010 DGAC, the 2015 Committee was able to use existing sources of evidence to answer an additional 45 percent of the questions it addressed. These sources included existing systematic reviews, meta-analyses, or reports. The remainder of the questions, 30 percent, were answered using data analyses and food pattern modeling analyses. These three approaches allowed the Committee to ask and answer its questions in a systematic, transparent, and evidence-based manner.

For all topics and questions, regardless of the path used to identify and evaluate the scientific evidence, the Committee developed conclusion statements and implications statements. Conclusion statements are a direct answer to the question asked, reflecting the strength of evidence reviewed (see additional details, below, in "Develop Conclusion Statements and Grade the Evidence"). Implications statements were developed to put the Conclusion in necessary context and varied in length depending on the topic or question. The primary purpose of these statements in this report is to describe what actions the Committee recommends that individuals, programs, or policies might take to promote health and prevent disease in light of the conclusion statement. However, some implications statements also provided important statements of fact or references to other processes or initiatives that the Committee felt were critical in providing a complete picture of how their advice should be applied to reach the desired outcomes.

Based on the existing body of evidence, research gaps, and limitations, the DGAC also formulated research recommendations that could advance knowledge related to its question and inform future Federal food and nutrition guidance as well as other policies and programs. Some research recommendations were developed and reported for specific topic areas covered in each chapter; others were overarching and covered an entire chapter.

Committee Working Structures and Process

The Committee's research questions were developed and prioritized initially by three Working Groups, which then organized themselves into five topic area Subcommittees, and four topic-specific Working or Writing Groups to conduct their work. The Subcommittees were: Food and Nutrient Intakes and Health: Current Status and Trends; Dietary Patterns, Foods and Nutrients, and Health Outcomes; Diet and Physical Activity Behavior Change; Food and Physical Activity Environments; and Food Sustainability and Safety. Working Groups were established on an "as needed" basis when a topic crossed two or more subcommittees. The three working groups were: Sodium, Added Sugars, and Saturated Fats. In addition, a Physical Activity Writing Group was established within the subcommittee on Food and Physical Activity Environments. The Subcommittees, Working Groups, and Writing Groups were made up of three to seven Committee members, with one Committee member appointed as the chair (for subcommittees) or lead (for working or writing groups). The membership of each group is listed in ***Appendix E-9***. Although the chair or lead member was responsible for communicating and coordinating all the work that needed to be accomplished within the group, recommendations coordinated by each group ultimately reflected the consensus of the entire Committee from deliberations in the public meetings. In addition, the Committee's Chair and Vice-chair served in an advisory role on each group.

Subcommittees and working/writing groups met regularly and communicated by conference calls, webinars, e-mail, and face-to-face meetings. Each group was responsible for presenting the basis for its draft conclusions and implications to the full Committee within the public meetings, responding to questions from the Committee, and making changes, if warranted. To gain perspective for interpreting the science, some groups invited experts on a one-time basis to participate in a meeting to provide their expertise on a particular topic being considered by the group. Two subcommittees also used consultants, who were experts in particular issues within the purview of the subcommittee's work. These consultants participated in subcommittee discussions and decisions on an ongoing basis, but were not members of the full Committee. Like Committee members, they completed training and were reviewed and cleared through a formal Federal process. Seven invited outside experts presented to the full Committee at the January and March, 2014, public meetings. These experts addressed questions posed by the Committee in advance and responded to additional questions during the meetings.

In addition to these five subcommittees and four working/writing groups, the DGAC included a Science Review Subcommittee, similar to that formed for the 2010 DGAC. The members included the DGAC Chair and Vice-chair and the two 2015 DGAC members who had also served on the 2010 DGAC. The main focus of this subcommittee was to provide oversight to the whole DGAC process. This Subcommittee played a primary role in organizing the Committee members into their initial work groups, then into subcommittees and working/writing groups. It facilitated the prioritization of topics to be considered by the Committee and provided oversight to ensure that consistent and transparent approaches were used when reviewing the evidence. This oversight also included monitoring the progress of work toward the development of this report in the allotted timeline. As the review of the science progressed, the Science Review Subcommittee meetings were opened to subcommittee Chairs and eventually to other working/writing group Leads when cross-cutting topics were placed on the agenda. In order to adhere to FACA guidelines, full Committee participation was not allowed.

The Committee members were supported by HHS's Designated Federal Officer, who led the administrative effort for this revision process and served as one of four Co-executive Secretaries (two from HHS and two from USDA). Support staff for managing Committee operations consisted of HHS and USDA Dietary Guidelines Management Team members and NEL Team members, including two research librarians. A third Federal staff team, the Data Analyses Team, provided support to the Committee by providing data upon the request of the Committee (see ***DGAC Membership*** for a list of these DGAC support staff).

DGAC Report Structure

Reflecting the DGAC subcommittee and working/writing group structure, the bulk of the report consists of seven science-based chapters that summarize the evidence assessed and evaluated by the Committee. Five chapters correspond to the work of the five subcommittees; one chapter covers the cross-cutting topics of sodium, saturated fat, and added sugars and low-calorie sweeteners; and one chapter addresses physical activity.

Throughout its deliberations, the Committee considered issues related to overall dietary patterns and the need for integrating findings from individual diet and nutrition topic areas. As a result, the Committee included an additional chapter—***Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence***.

SYSTEMATIC REVIEW OF THE SCIENTIFIC EVIDENCE

The USDA's Nutrition Evidence Library (NEL), housed within the Center for Nutrition Policy and Promotion, was responsible for assisting the 2015 DGAC in reviewing the science and supporting development of the 2015 DGAC Report. The NEL used state-of-the-art methodology informed by the Agency for Healthcare Research and Quality (AHRQ),[1] the Cochrane Collaboration,[2] the Academy of Nutrition and Dietetics[3] and the 2011 Institute of Medicine systematic review (SR)[4] standards to review, evaluate, and synthesize published, peer-reviewed food and nutrition research. The NEL's rigorous, protocol-driven methodology is designed to maximize transparency, minimize bias, and ensure SRs are relevant, timely, and high-quality. Using the NEL evidence-based approach enables HHS and USDA to comply with the Data Quality Act, which states that Federal agencies must ensure the quality, objectivity, utility, and integrity of the information used to form Federal guidance.

DGAC members developed the SR questions and worked with NEL staff to implement the SRs. The following represent overarching principles for the NEL process:

- The DGAC made all substantive decisions required during the process.
- NEL staff provided facilitation and support to ensure that the process was consistently implemented in accordance with NEL methodology.
- NEL used document templates, which served as a starting point and were tailored to each specific review.
- When working with the DGAC, the Science Review Subcommittee provided oversight to the DGAC's work throughout the deliberative process, ensuring that the Subcommittees used consistent and transparent approaches when reviewing the evidence using NEL SRs.

The NEL employed a six-step SR process, which leveraged a broad range of expert inputs:

- Step 1: Develop systematic review questions and analytic frameworks
- Step 2: Search, screen, and select studies to review
- Step 3: Extract data and assess the risk of bias of the research
- Step 4: Describe and synthesize the evidence
- Step 5: Develop conclusion statements and grade the evidence
- Step 6: Identify research recommendations

Each step of the process was documented to ensure transparency and reproducibility. Specific information about each review is available at www.NEL.gov, including the research questions, the related literature search protocol, literature selection decisions, an assessment of the methodological quality of each included study, evidence summary materials, evidence tables, a description of key findings, graded conclusion statements, and identification of research limitations and gaps. These steps are described below.

Develop Systematic Review Questions and Analytic Frameworks

The DGAC identified, refined, and prioritized the most relevant topics and then developed clearly focused SR questions that were appropriate in scope, reflected the state of the science, and targeted important policy relevant to public health issue(s). Once topics and systematic review questions were generated, the DGAC developed an analytical framework for each topic in accordance with NEL methodology. These frameworks clearly identified the core elements of the systematic review question/s, key definitions, and potential confounders to inform development of the systematic review protocol.

The core elements of a SR question include Population, Intervention or Exposure, Comparator, and Outcomes (PICO). These elements represent key aspects of the topic that need to be considered in developing a SR framework. An analytic framework is a type of evidence model that defines and links the PICO elements and key confounders. The analytical framework serves as a visual representation of the overall scope of the project, provides definitions for key SR terms, helps to ensure that all contributing elements in the causal chain will be examined and evaluated, and aids in determining inclusion and exclusion criteria and the literature search strategy.

Search, Screen, and Select Studies to Review

Searching, screening, and selecting scientific literature was an iterative process that sought to identify the most complete and relevant body of evidence to answer a SR question. This process was guided by inclusion and exclusion criteria determined a priori by the DGAC. The NEL librarians created and implemented search strategies that included appropriate databases and search terms to identify literature to answer each SR question. The results of the literature search were screened by the NEL librarians and staff in a dual, step-wise manner, beginning with titles, followed by abstracts, and then full-text articles, to determine which articles met the criteria for inclusion in the review. Articles that met the inclusion criteria were hand searched in an effort to find additional pertinent articles not identified through the electronic search. In addition, NEL staff and the DGAC conducted a duplication assessment to determine whether high-quality SRs or meta-analyses (MA) were available to augment or replace a NEL SR.

The DGAC provided direction throughout this process to ensure that the inclusion and exclusion criteria were applied appropriately and the final list of included articles was complete and captured all research available to answer a SR question. Each step of the process also was documented to ensure transparency and reproducibility.

The NEL established and the DGAC approved standard inclusion and exclusion criteria to promote consistency across reviews and ensure that the evidence being considered in NEL SRs was most relevant to the U.S. population. The DGAC used these standard criteria and revised them a priori as needed to ensure that they were appropriate for the specific SR being conducted. In general, criteria were established based on the analytical framework to ensure that each study included the appropriate population, intervention/exposure, comparator(s), and outcomes. They were typically established for the following study characteristics:

- Study design
- Date of publication
- Publication language
- Study setting
- Study duration
- Publication status (i.e., peer reviewed)
- Type, age, and health status of study subjects
- Size of study groups
- Study dropout rate

To capitalize on existing literature reviews, the NEL performed duplication assessments, which identified any existing high-quality SRs and/or MAs that addressed the topic or SR questions posed. Existing SRs and MAs were valuable sources of evidence and were used for two main purposes in the NEL SR process:

- To augment a NEL SR as an additional source of evidence, but not as an included study in the review (in this case, the studies in the existing SR or MA would not be included individually in the NEL review that was conducted); or
- To replace a de novo NEL SR.

NEL also used existing SRs to provide background and context for current reviews, inform SR methodology, and cross-check the literature search for completeness.

If multiple relevant, low risk of bias, and timely SRs or MA were available, the reviews were compared and a decision was made as to whether an existing SR/MA would be used, or whether a de novo SR would be conducted. This decision was made based on the relevancy of the review in relation to the SR question and, when more than one review was identified, the consistency of the findings. If existing SRs/MA addressed different aspects of the outcome, more than one SR/MA may have been be used to replace a de novo SR. More information on the use of existing SRs/MAs to replace a de novo NEL SR is provided below in the section "Existing Sources of Evidence."

Extract Data and Assess the Risk of Bias

Key information from each study included in a systematic review was extracted and a risk of bias assessment was performed by a NEL abstractor. NEL abstractors are National Service Volunteers from across the United States with advanced degrees in nutrition or a related field who were trained to review individual research articles included in NEL systematic reviews (a list of the Volunteers is included in ***Appendix E-10: Dietary Guidelines Advisory Committee Report Acknowledgments***). From the evidence grids, summary tables are created for each SR that highlight the most relevant data from the reviewed papers. These tables are available on www.NEL.gov.

The risk of bias (i.e., internal validity) for each study was assessed using the NEL Bias Assessment Tool (BAT) (see Table C.1 at the end of this chapter). This tool helped in determining whether any systematic error existed to either over- or under-estimate the study results. This tool was developed in collaboration with a panel of international systematic review experts.

NEL staff reviewed the work of abstractors, resolved inconsistencies, and generated a draft of a descriptive summary of the body of evidence. The DGAC reviewed this work and used it to inform their synthesis of the evidence.

Describe and Synthesize the Evidence

Evidence synthesis is the process by which the DGAC compared, contrasted, and combined evidence from multiple studies to develop key findings and a graded conclusion statement that answered the SR question. This qualitative synthesis of the body of evidence involved identifying overarching themes or key concepts from the findings, identifying and explaining similarities and differences between studies, and determining whether certain factors affected the relationships being examined.

To facilitate the DGAC's review and analysis of the evidence, staff prepared a "Key Trends" template for each SR question. This document was customized for each question and included questions related to major trends, key observations, themes for conclusion statements and key findings. It also addressed methodological problems or limitations, magnitude of effect, generalizability of results, and research recommendations. DGAC members used the description of the evidence, along with the full data extraction grid, and full-text manuscripts to complete the "Key Trends" questions. The responses were compiled and used to draft the qualitative evidence synthesis and the conclusion statement.

Develop Conclusion Statements and Grade the Evidence

The conclusion statement is a brief summary statement worded as an answer to the SR question. It must be tightly associated with the evidence, focused on general agreement among the studies around the independent variable(s) and outcome(s), and may acknowledge areas of disagreement or limitations, where they exist. The conclusion statement reflects the evidence reviewed and does not include information that is not addressed in the studies. The conclusion statement also may identify a relevant population, when appropriate. In addition, "key findings" (approximately 3 to 5 bulleted points) were drafted for some questions to provide context and highlight important findings that contributed to conclusion statement development (e.g., brief description of the evidence reviewed, major themes, limitations of the research reviewed or results from intermediate biomarkers).

The DGAC used predefined criteria to evaluate and grade the strength of available evidence supporting each conclusion statement. The grade communicates to decision makers and stakeholders the strength of the evidence supporting a specific conclusion statement. The grade for the body of evidence and conclusion statement was based on five elements outlined in the NEL grading rubric: quality, quantity, consistency, impact and generalizability (see Table C.2 at the end of this chapter for the full NEL grading rubric).

EXISTING SOURCES OF EVIDENCE: REPORTS, SYSTEMATIC REVIEWS, AND META-ANALYSES

For a number of topics, the DGAC chose to consider existing high-quality sources of evidence such as existing reports from leading scientific organizations or Federal agencies, SRs, and/or MA to fully or partially address questions. (These three categories of existing sources of evidence are collectively referred to in this report as "existing reports.") This was done to prevent duplication of effort and promote time and resource management. The methods generally used to identify and review existing reports are described below, and any modifications to this process for answering a question are described in the Methodology section of the individual Science Base chapters (e.g., the DGAC relied on three Federal reports to write the Physical Activity chapter; see the Methods section of ***Part D. Chapter 7: Physical Activity*** for details on the process the Committee used to review the evidence and develop conclusion statements from these existing reports).

First, an analytical framework was developed that clearly described the population, intervention/exposure, comparator, and outcomes (intermediate and clinical) of interest for the question being addressed. When Committee members were aware of high-quality existing reports that addressed their question(s), they decided a priori to use existing report(s), rather than to conduct a de novo NEL SR. A literature search was then conducted to identify other existing reports to augment the existing report(s) identified by the Committee. The literature was searched by a NEL librarian to identify relevant studies. The process used to create and execute the literature search is described in detail above (see "Search, Screen, and Select Studies to Review"). In other cases, the Committee was not aware of any existing reports and intended to conduct a de novo NEL SR. However, as part of the duplication assessment step of the NEL process, one or more existing SRs or MA were identified that addressed the question that led to the Committee deciding to proceed using existing SRs/MA rather than complete an independent review of the primary literature. This process is also described above. Finally, for some questions, the Committee used existing reports as the primary source of evidence to answer a question, but chose to update one or more of those existing reports using the NEL process to identify and review studies that had been published after the completion of the literature search for the existing report(s).

When SRs or MA that addressed the question posed by the Committee were identified, staff conducted a quality assessment using the Assessment of Multiple Systematic Reviews (AMSTAR) tool.[5] This tool includes 11 questions, each of which is given a score of one if the criterion is met or a score of zero if the criterion is not met, is unclear, or is not applicable (see Table C.3 at the end of this chapter). Guidance for answering some of the questions was tailored for the work of the Committee. Articles rated 0-3 were considered to be of low quality, 4-7 of medium quality, and 8-11 of high quality.[6] Unless otherwise noted, only high quality SRs/MA, receiving scores of 8-11, were considered by the DGAC.

In a few cases, existing reports were considered that did not examine the evidence using SR or MA. These reports were discussed by the subcommittees and determined to be of high-quality. The subcommittees also had the option of bringing existing reports to the Science Review Subcommittee to ensure that the report met the quality standards of the Committee, if needed.

Next, if multiple high-quality existing reports were identified, their reference lists were compared to find whether any references and/or cohorts were included in more than one of the existing reports. The Committee then addressed the overlap in their review of the evidence ensuring that, in cases where overlap existed, that the quantity of evidence available was not overestimated. In a few cases, if two or more SRs/MAs appropriately answered a question and there was substantial reference overlap, the Committee chose to only use one of the SRs/MA to answer the question.

Tables or other documents that summarized the methodology, evidence, and conclusions of the existing reports were used by the Committee members to facilitate their review of the evidence. For example, a “Key Trends” document was often used to help identify themes observed in the body of evidence. The “Key Trends” document included questions related to major trends, key observations, themes for key findings, and conclusion statements. Members of the DGAC used the description of the evidence, along with summary tables and the original reports, to answer the questions. Feedback from the DGAC on the “Key Trends” document was compiled and used to draft the qualitative evidence synthesis and the conclusion statement. As described above, the conclusion statement is a brief summary statement worded as an answer to the question. In drawing conclusions, Committee members could choose to:

1. Carry forward findings or conclusions from existing report(s).

2. Synthesize the findings from multiple existing report(s) to develop their own conclusions.
3. Place primary emphasis on the existing report(s) and discuss how new evidence identified through the NEL process relates to the conclusions or findings of the existing report(s).

Next, the Committee graded their conclusion statement using a table of strength of evidence grades adapted specifically use with existing reports (see Table C.4 at the end of this chapter). In cases where the DGAC used an existing report with its own formally graded conclusions, the Committee acknowledged the grade assigned within that existing report, and then assigned a DGAC grade that was the closest equivalent to the grade assigned in the existing report.

DATA ANALYSES

Federal Data Acquisition

Earlier Committees used selected national, Federal data about the dietary, nutritional, and health status of the U.S. population. In the 2015 DGAC, a Data Analysis Team (DAT) was established to streamline the data acquisition process and efficiently support the data requests of the Committee. During the Committee's work, the data used by the DGAC were publically available through www.DietaryGuidelines.gov. Upon publication, the data became available through the report's references and appendices.

Upon request from the DGAC, the DAT either conducted data analyses or compiled data from their agencies' publications for the DGAC to use to answer specific research questions. The DGAC took the strengths and limitations of data analyses into account in drawing conclusions. The grading rubric used for questions answered using NEL systematic reviews do not apply for to questions answered using data analyses; therefore, these conclusions were not graded.

Most of the analyses used the National Health and Nutrition Examination (NHANES) data and its dietary component, What We Eat in America (WWEIA), NHANES.[7] These data were used to answer questions about food and nutrient intakes because they provide national and group level estimates of dietary intakes of the U.S. population, on a given day as well as usual intake distributions. These data contributed substantially to questions answered using data analyses (see ***Appendix E-4: NHANES Data Used in DGAC Data Analyses*** for additional discussion of the NHANES data used by the 2015 DGAC).

NHANES Data

The NHANES data used by the 2015 DGAC included:

- Estimates of the distribution of usual intakes of energy and selected macronutrients and micronutrients from food and beverages by various demographic groups, including the elderly population, race/ethnicities, and pregnant women.
- Estimates of the distribution of usual intakes of selected nutrients from food, beverages, and supplements.
- Estimates of the distribution of usual intake of USDA Food Pattern food groups by demographic population groups.
- Eating behaviors such as meal skipping, contribution of meals and snacks to energy and nutrient intakes.
- Nutrients and food group content per 1,000 calories of food and beverages obtained from major point of purchase.
- Nutritional quality of food prepared at home and away from home.
- Energy, selected nutrients, and food groups obtained from food categories by demographic population groups.
- Selected biochemical indicators of diet and nutrition in the U.S. population.
- Prevalence of health concerns and trends, including body weight status, lipid profiles, high blood pressure, and diabetes.

Other Data Sources

The DGAC also used data from the National Health Interview Survey, the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) statistics, and heart disease and stroke statistics from the 2014 report of the American Heart Association.[8, 9] In addition, the Committee used USDA National Nutrient Database for Standard Reference, Release 27, 2014 to list food sources ranked by amounts of selected nutrients (calcium, fiber, iron, potassium, and Vitamin D) and energy per standard food portions and per 100 grams of foods.[10]

SPECIAL ANALYSES USING THE USDA FOOD PATTERNS

As described above, the Committee used NEL systematic reviews, existing reports, and data analyses to draw the majority of its conclusions on the relationship between diet and health. Because the primary charge of the Committee is to provide food-based recommendations with the potential to inform the next edition of the *Dietary Guidelines for Americans*, it was imperative that the Committee also advise the government on how to articulate the evidence on the relationships between diet and health through food patterns. This was a critical task for the

Committee because the *Dietary Guidelines* are the basis for all Federal nutrition assistance and educational initiatives. For this reason, like the 2005 and 2010 DGAC's, this Committee developed a number of questions to be answered through a food pattern modeling approach, using the USDA Food Patterns.

Briefly, the USDA Food Patterns describe types and amounts of food to consume that will provide a nutritionally adequate diet. They include recommended intakes for five major food groups and for subgroups within several of the food groups. They also recommend an allowance for intake of oils and limits on intake of calories from solid fats and added sugars. The calories and nutrients that would be expected from consuming a specified amount from each component of the patterns (e.g., whole grains, fruits, or oils) are determined by calculating nutrient profiles. A nutrient profile is the average nutrient content for each component of the Patterns. The profile is calculated from the nutrients in nutrient-dense forms of foods in each component, and is weighted based on the relative consumption of each of these foods. Additional details on the USDA Food Patterns can be found in the report for the food pattern modeling analysis, *Adequacy of the USDA Food Patterns* (see ***Appendix E-3: USDA Food Patterns for Special Analyses).***

The USDA Food Patterns were originally developed in the 1980s,[11, 12] and were substantially revised and updated in 2005, concurrent with the development of the 2005 Dietary Guidelines.[13] The Patterns were updated and slightly revised in 2010, concurrent with the development of the 2010 Dietary Guidelines.[14] The 2005 and 2010 updates included use of nutrient goals from the Institute of Medicine *Dietary Reference Intakes* reports that were released from 1997 to 2004.[15-20] The developmental process and the food patterns resulting from the 2005 and 2010 updates have been documented in detail.[13, 14, 21]

A food pattern modeling process was developed for the 2005 DGAC and used by the 2005 and 2010 DGACs to determine the hypothetical effect on nutrients in and adequacy of the Food Patterns when specific changes are made.[13, 14] The structure of the USDA Food Patterns allows for modifications that test the overall influence on diet quality of various dietary recommendation scenarios. Most analyses involved identifying the impact of specific changes in amounts or types of foods that might be included in the pattern. Changes might involve modifying the nutrient profiles for a food group, or changing amounts recommended for a food group or subgroup, based on the assumptions for the food pattern modeling analysis. For example, 2005 DGAC subcommittees requested analyses to obtain information on the potential effect of consumers selecting only lacto-ovo vegetarian choices, eliminating legumes, or choosing varying levels of fat as a percent of calories[22] on nutritional adequacy. The use of food pattern modeling analyses for the 2005 and 2010 DGAC have been documented.[23-26]

The DGAC referred questions that could be addressed through food pattern modeling to the Food and Nutrient Intakes and Health: Current Status and Trends Subcommittee. The DGAC identified that a number of questions could be answered by modeling analyses conducted for the 2005 or 2010 DGACs. The food pattern modeling analyses conducted for the 2015 DGAC are listed in ***Appendix E-3: USDA Food Pattern Modeling Analyses***. For each question answered using food pattern modeling, a specific approach was drafted by USDA staff and provided to the DGAC for comment. After the approach was adjusted and approved by the DGAC, USDA staff completed the analytical work and drafted a full report for the DGAC's consideration.

The modeling process also was used to develop new USDA Food Patterns based on different types of evidence: the "Healthy Vegetarian Pattern," which takes into account food choices of self-identified vegetarians, and the "Healthy Mediterranean-style Pattern," which takes into account food group intakes from studies using a Mediterranean diet index to assess dietary patterns. The latter were compiled and summarized to answer the questions addressed on dietary patterns composition. The food group content of dietary patterns reviewed by the DGAC and found to have health benefits formed the basis for answering these questions. WWEIA food group intakes and USDA Food Pattern recommendations were compared with the food group intake data from the healthy dietary patterns as part of the answer for these questions.

REFERENCES

1. Methods Guide for Comparative Effectiveness Reviews. AHRQ Publication No. 10(14)-EHC063-EF. Rockville, MD: Agency for Healthcare Research and Quality; January 2014. Available from: www.effectivehealthcare.ahrq.gov.
2. Higgins J, Green S, editors. Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0: The Cochrane Collaboration; 2011 [updated March 2011]. Available from: http://handbook.cochrane.org/.
3. American Dietetic Association. Research and Strategic Business Development. Evidence Analysis Manual Chicago, IL: American Dietetic Association; 2012 [updated January 2012]. Available from: http://www.andeal.org/files/Docs/2012_Jan_EA_Manual.pdf.
4. Institute of Medicine. Finding What Works in Health Care: Standards for Systematic Reviews. Washington, DC: The National Academies Press; 2011. Available from: http://books.nap.edu/openbook.php?record_id=13059.
5. Shea BJ, Grimshaw JM, Wells GA, Boers M, Andersson N, Hamel C, et al. Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews. BMC Med Res Methodol. 2007;7:10. PMID: 17302989. http://www.ncbi.nlm.nih.gov/pubmed/17302989.
6. Sharif MO, Janjua-Sharif FN, Ali H, Ahmed F. Systematic reviews explained: AMSTAR-how to tell the good from the bad and the ugly. Oral Health Dent Manag. 2013;12(1):9-16. PMID: 23474576. http://www.ncbi.nlm.nih.gov/pubmed/23474576.
7. Zipf G, Chiappa M, Porter K. National Health and Nutrition Examination Survey: Plan and operations, 1999–2010. Vital Health Stat. 2013; 1(56). Available from: http://www.cdc.gov/nchs/data/series/sr_01/sr01_056.pdf.

8. Blackwell D, Lucas J, Clarke T. Summary health statistics for U.S. adults: National Health Interview Survey, 2012. Vital Health Stat 10(260). National Center for Health Statistics; 2014. Available from: http://www.cdc.gov/nchs/data/series/sr_10/sr10_260.pdf.

9. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, et al. Heart disease and stroke statistics--2014 update: a report from the American Heart Association. Circulation. 2014;129(3):e28-e292. PMID: 24352519. http://www.ncbi.nlm.nih.gov/pubmed/24352519.

10. U.S. Department of Agriculture. Agricultural Research Service. USDA National Nutrient Database for Standard Reference, Release 27. Nutrient Data Laboratory Home Page. 2014. Available from: http://www.ars.usda.gov/Services/docs.htm?docid=8964.

11. Cronin FJ, Shaw AM, Krebs-Smith SM, Marsland PM, Light L. Developing a food guidance system to implement the dietary guidelines. Journal of Nutrition Education. 1987;19(6):281-302. http://www.sciencedirect.com/science/article/pii/S0022318287802376.

12. Welsh S, Davis C, Shaw A. USDA’s Food Guide: Background and Development. Hyattesville, MD: U.S. Department of Agriculture. Human Nutrition Information Service. Misc. Publication 1514, September 1993. Available from: http://www.cnpp.usda.gov/sites/default/files/archived_projects/FGPBackgroundAndDevelopment.pdf.

13. Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food Guidance System. J Nutr Educ Behav. 2006;38(6 Suppl):S78-92. PMID: 17116598. http://www.ncbi.nlm.nih.gov/pubmed/17116598.

14. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Updated US Department of Agriculture Food Patterns meet goals of the 2010 dietary guidelines. J Acad Nutr Diet. 2012;112(10):1648-55. PMID: 22853987.

15. Institute of Medicine. Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Washington, DC: The National Academies Press; 1997. Available from: http://www.nap.edu/catalog.php?record_id=5776.

16. Institute of Medicine. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington, DC: The National Academies Press; 1998. Available from: http://www.nap.edu/openbook.php?record_id=6015.

17. Institute of Medicine. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. Washington, DC: The National Academies Press; 2000. Available from: http://www.nap.edu/openbook.php?record_id=9810.

18. National Research Council. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington, DC: The National Academies Press; 2001. Available from: http://www.nap.edu/openbook.php?record_id=10026.

19. National Research Council. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington, DC: The National Academies Press; 2002. Available from: http://www.nap.edu/openbook.php?record_id=10490.

20. National Research Council. Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate. Washington, DC: The National Academies Press; 2005. Available from: http://www.nap.edu/catalog.php?record_id=10925.
21. Marcoe K, Juan W, Yamini S, Carlson A, Britten P. Development of food group composites and nutrient profiles for the MyPyramid Food Guidance System. J Nutr Educ Behav. 2006;38(6 Suppl):S93-S107. PMID: 17116599. http://www.ncbi.nlm.nih.gov/pubmed/17116599.
22. Dietary Guidelines Advisory Committee. 2005. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, to the Secretary of Health and Human Services and the Secretary of Agriculture: U.S. Department of Agriculture, Agricultural Research Service; 2005. Available from: http://www.health.gov/dietaryguidelines/dga2005/report/default.htm.
23. Britten P, Lyon J, Weaver CM, Kris-Etherton PM, Nicklas TA, Weber JA, et al. MyPyramid food intake pattern modeling for the Dietary Guidelines Advisory Committee. J Nutr Educ Behav. 2006;38(6 Suppl):S143-52. PMID: 17116592. http://www.ncbi.nlm.nih.gov/pubmed/17116592.
24. Nicklas TA, Weaver C, Britten P, Stitzel KF. The 2005 Dietary Guidelines Advisory Committee: developing a key message. J Am Diet Assoc. 2005;105(9):1418-24. PMID: 16129084. http://www.ncbi.nlm.nih.gov/pubmed/16129084.
25. Weaver C, Nicklas T, Britten P. The 2005 Dietary Guidelines Advisory Committee Report: Achieving Nutritional Recommendations Through Food-based Guidance. Nutrition Today. 2005;40(3):102-7. Available from: http://journals.lww.com/nutritiontodayonline/Abstract/2005/05000/The_2005_Dietary_Guidelines_Advisory_Committee.3.aspx
26. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Impact of typical rather than nutrient-dense food choices in the US Department of Agriculture Food Patterns. J Acad Nutr Diet. 2012;112(10):1560-9. PMID: 22906562. http://www.ncbi.nlm.nih.gov/pubmed/22906562.

Table C.1 Nutrition Evidence Library Bias Assessment Tool (BAT)

The NEL Bias Assessment Tool (NEL BAT) is used to assess the risk of bias of each individual study included in a SR. The types of bias that are addressed in the NEL BAT include:

Selection Bias	Systematic differences between baseline characteristics of the groups that are compared; error in choosing the individuals or groups taking part in a study
Performance Bias	Systematic differences between groups in the intervention/exposure received, or in experience with factors other than the interventions/exposures of interest
Detection Bias	Systematic differences between groups in how outcomes are determined; outcomes are more likely to be observed or reported in certain subjects
Attrition Bias	Systematic differences between groups in withdrawals from a study, particularly if those who drop out of the study are systematically different from those who remain in the study
Adapted from: Cochrane Bias Methods Group: http://bmg.cochrane.org/assessing-risk-bias-included-studies	

The NEL BAT is tailored by study design, with different sets of questions applying to randomized controlled trials (14 questions), non-randomized controlled trials (14 questions), and observational studies (12 questions). Abstractors complete the NEL BAT after data extraction for each article. There are four response options:

- **Yes:** Information provided in the article is adequate to answer "yes".
- **No:** Information provided in the article clearly indicates an answer of "no".
- **Cannot Determine:** No information or insufficient information is provided in the article, so an answer of "yes" or "no" is not possible.
- **N/A:** The question is not applicable to the article.

The NEL Bias Assessment Tool (NEL BAT)		
Risk of Bias Questions	**Study Designs**	**Type of Bias**
Were the inclusion/exclusion criteria similar across study groups?	Controlled trials Observational studies	Selection Bias
Was the strategy for recruiting or allocating participants similar across study groups?	Controlled trials Observational studies	Selection Bias
Was the allocation sequence randomly generated?	RCTs	Selection Bias
Was the group allocation concealed (so that assignments could not be predicted)?	RCTs	Selection Bias Performance Bias
Was distribution of health status,	RCTs	Selection Bias

demographics, and other critical confounding factors similar across study groups at baseline? If not, does the analysis control for baseline differences between groups?	Controlled trials Observational studies	
Did the investigators account for important variations in the execution of the study from the proposed protocol or research plan?	RCTs Controlled trials Observational studies	Performance Bias
Was adherence to the study protocols similar across study groups?	RCTs Controlled trials Observational studies	Performance Bias
Did the investigators account for the impact of unintended/unplanned concurrent interventions or exposures that were differentially experienced by study groups and might bias results?	RCTs Controlled trials Observational studies	Performance Bias
Were participants blinded to their intervention or exposure status?	RCTs Controlled trials	Performance Bias
Were investigators blinded to the intervention or exposure status of participants?	RCTs Controlled trials	Performance Bias
Were outcome assessors blinded to the intervention or exposure status of participants?	RCTs Controlled trials Observational studies	Detection Bias
Were valid and reliable measures used consistently across all study groups to assess inclusion/exclusion criteria, interventions/exposures, outcomes, participant health benefits and harms, and confounding?	RCTs Controlled trials Observational studies	Detection Bias
Was the length of follow-up similar across study groups?	RCTs Controlled trials Observational studies	Attrition Bias
In cases of high or differential loss to follow-up, was the impact assessed (e.g., through sensitivity analysis or other adjustment method)?	RCTs Controlled trials Observational studies	Attrition Bias
Were other sources of bias taken into account in the design and/or analysis of the study (e.g., through matching, stratification, interaction terms, multivariate analysis, or other statistical adjustment such as instrumental variables)?	RCTs Controlled trials Observational studies	Attrition, Detection, Performance, and Selection Bias
Were the statistical methods used to assess the primary outcomes adequate?	RCTs Controlled trials Observational studies	Detection Bias

The completed NEL BAT is used to rate the overall risk of bias for the article by tallying the responses to each question. Each "Yes" response receives 0 points, each "Cannot Determine" response receives 1 point, each "No" response receives 2 points, and each "N/A" response receives 0 points. Since 14 questions are answered for randomized controlled trials and non-randomized controlled trials, they will be assigned a risk of bias rating out of a maximum of 28 points; while observational studies will be out of 24 points. The lower the number of points received, the lower the risk of bias.

Table C.2 NEL Grading Rubric

USDA Nutrition Evidence Library Conclusion Statement Evaluation
Criteria for judging the strength of the body of evidence supporting the Conclusion Statement

Elements	***Grade I: Strong***	**Grade II: Moderate**	**Grade III: Limited**	**Grade IV: Grade Not Assignable***
Risk of bias (as determined using the NEL Bias Assessment Tool)	Studies of strong design free from design flaws, bias and execution problems	Studies of strong design with minor methodological concerns OR only studies of weaker study design for question	Studies of weak design for answering the question OR inconclusive findings due to design flaws, bias or execution problems	Serious design flaws, bias, or execution problems across the body of evidence
Quantity • Number of studies • Number of subjects in studies	Several good quality studies; large number of subjects studied; studies have sufficiently large sample size for adequate statistical power	Several studies by independent investigators; doubts about adequacy of sample size to avoid Type I and Type II error	Limited number of studies; low number of subjects studied and/or inadequate sample size within studies	Available studies do not directly answer the question OR no studies available
Consistency of findings across studies	Findings generally consistent in direction and size of effect or degree of association and statistical significance with very minor exceptions	Some inconsistency in results across studies in direction and size of effect, degree of association or statistical significance	Unexplained inconsistency among results from different studies	Independent variables and/or outcomes are too disparate to synthesize OR single small study unconfirmed by other studies
Impact • Directness of studied outcomes • Magnitude of effect	Studied outcome relates directly to the question; size of effect is clinically meaningful	Some study outcomes relate to the question indirectly; some doubt about the clinical significance of the effect	Most studied outcomes relate to the question indirectly; size of effect is small or lacks clinical significance	Studied outcomes relate to the question indirectly; size of effect cannot be determined
Generalizability to the U.S. population of interest	Studied population, intervention and outcomes are free from serious doubts about generalizability	Minor doubts about generalizability	Serious doubts about generalizability due to narrow or different study population, intervention or outcomes studied	Highly unlikely that the studied population, intervention AND/OR outcomes are generalizable to the population of interest

Table C.3 AMSTAR (Assessment of Multiple Systematic Reviews) Tool

		YES	NO	Can't Answer	N/A
1	**Was an 'a priori' design provided?** *The research question and inclusion criteria should be established before the conduct of the review.*				
2	**Was there duplicate study selection and data extraction?** *There should be at least two independent data extractors and a consensus procedure for disagreements should be in place.*				
3	**Was a comprehensive literature search performed?** *At least two electronic sources should be searched. The report must include years and databases used (e.g. Central, EMBASE, and MEDLINE). Key words and/or MESH terms must be stated and where feasible the search strategy should be provided. All searches should be supplemented by consulting current contents, reviews, textbooks, specialized registers, or experts in the particular field of study, and by reviewing the references in the studies found.*				
4	**Was the status of publication (i.e. grey literature) used as an inclusion criterion?** **The authors should state that they searched for reports regardless of their publication type. The authors should state whether or not they excluded any reports (from the systematic review), based on their publication status, language, etc.*				
5	**Was a list of studies (included and excluded) provided?** *A list of included and excluded studies should be provided.*				
6	**Were the characteristics of the included studies provided?** *In an aggregated form such as a table, data from the original studies should be provided on the participants, interventions and outcomes. The ranges of characteristics in all the studies analyzed e.g. age, race, sex, relevant socioeconomic data, disease status, duration, severity, or other diseases should be reported.*				
7	**Was the scientific quality of the included studies assessed and documented?** *'A priori' methods of assessment should be provided (e.g., for effectiveness studies if the author(s) chose to include only randomized, double-blind, placebo controlled studies, or allocation concealment as inclusion criteria); for other types of studies alternative items will be relevant.*				
8	**Was the scientific quality of the included studies used appropriately in formulating conclusions?** *The results of the methodological rigor and scientific quality should be considered in the analysis and the conclusions of the review, and explicitly stated in formulating recommendations.*				
9	**Were the methods used to combine the findings of studies appropriate?** **For the pooled results, a test should be done to ensure the studies were combinable, to assess their homogeneity (i.e. Chisquared test for homogeneity, I2). If heterogeneity exists a random effects model should be used and/or the clinical appropriateness of combining should be taken into consideration (i.e. is it sensible to combine?).*				
10	**Was the likelihood of publication bias assessed?** *An assessment of publication bias should include a combination of graphical aids (e.g., funnel plot, other available tests) and/or statistical tests (e.g., Egger regression test).*				
11	**Was the conflict of interest stated?** *Potential sources of support should be clearly acknowledged in both the systematic review and the included studies.*				

* The guidance for answering this question was adapted for the 2015 Dietary Guidelines Advisory Committee.

Table C.4 Strength of Evidence terminology to support a conclusion statement when a question is answered with existing reports

Strong	The conclusion statement is substantiated by a large, high quality, and/or consistent body of evidence that directly addresses the question. There is a high level of certainty that the conclusion is generalizable to the population of interest, and it is unlikely to change if new evidence emerges.
Moderate	The conclusion statement is substantiated by sufficient evidence, but the level of certainty is restricted by limitations in the evidence, such as the amount of evidence available, inconsistencies in findings, or methodological or generalizability concerns. If new evidence emerges, there could be modifications to the conclusion statement.
Limited	The conclusion statement is substantiated by insufficient evidence, and the level of certainty is seriously restricted by limitations in the evidence, such as the amount of evidence available, inconsistencies in findings, or methodological or generalizabilty concerns. If new evidence emerges, there could likely be modifications to the conclusion statement.
Grade not assignable	A conclusion statement cannot be drawn due to a lack of evidence, or the availability of evidence that has serious methodological concerns.

Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends

INTRODUCTION

Humans require a wide range of essential micronutrients and macronutrients for normal growth and development and to support healthy aging throughout the life cycle. Essential nutrients, including most vitamins, minerals, amino acids and fatty acids, water and fiber, must be obtained through foods and beverages because they cannot for the most part be endogenously synthesized, or are not endogenously synthesized in adequate amounts to need recommended intakes. Understanding the extent to which the U.S. population and various age, sex, and racial/ethnic groups within the population achieve nutrient intake requirements through available food and beverage intake, including foods and beverages[*] that are enriched or fortified, is an important task of the DGAC. Notably, the DGAC considers that the primary source of nutrients should come from foods and beverages. Nutrient-dense forms of foods (those providing substantial amounts of vitamins, minerals and other nutrients and relatively few calories) are recommended to ensure optimal nutrient intake without exceeding calorie intake or reaching excess or potentially toxic levels of certain nutrients.

In the process of evaluating adequacy of nutrient intake of the U.S. population, the DGAC identified two levels of “Nutrients of Concern”. Shortfall nutrients are those that may be underconsumed relative to the Estimated Average Requirement (EAR) or Adequate Intake (AI). Overconsumed nutrients are those that are consumed in amounts above the Tolerable Upper Limit of Intake (UL)[1] or other nationally recognized standard.[2] Nutrients of Public Health Concern were those shortfall or overconsumed nutrients that also had evidence of under- or overconsumption through biochemical nutritional status indicators[3] plus evidence that the nutrient inadequacy or nutrient excess is directly related to a specific health condition. This information is critical in determining where dietary intake improvements may be warranted that will benefit the health of the population. The 2015 DGAC recognizes that the 2010 DGAC specifically addressed whether or not multivitamins provided health benefits. The 2015 DGAC did not specifically address multivitamins, but recognizes that some dietary supplements may be recommended for some populations or life-cycle phases (pregnancy, for example).

In addition, many foods contain constituents that enable them to be produced, preserved, and thus widely available year round. Some of these ingredients, such as sodium, are used to make foods shelf stable and can help ensure food availability and food security for the population as a whole.[4] Other ingredients, such as added sugars, are used as a food preservative and to enhance palatability. Despite the functional nature of both sodium and added sugars in the food supply, excess consumption of these

[*] Note: The DGAC considered foods and beverages in its review of intake data. Throughout this chapter, references to “foods” should be taken to mean “foods and beverages.”

dietary constituents poses potential health risks and was of particular concern to the DGAC. This chapter reviews data on intakes of sodium, added sugars and saturated fat; other chapters consider sodium, added sugars, and saturated fat from additional perspectives (see ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance)*** including health outcomes. The food supply also contains ingredients that are both naturally occurring and also added to foods and beverages, such as caffeine, that have generated considerable attention in recent years. This chapter examines intake levels across age and sex groups of the U.S. population; ***Part D. Chapter 5: Food Sustainability and Safety*** considers several safety aspects of caffeine consumption.

The U.S. food supply is complex. Tens of thousands of foods and food products are available in a variety of forms. Some foods are whole foods that are often eaten alone without additions, such as fruit and milk, while others, such as sandwiches and mixed dishes, are mixtures of multiple components from more than one food group.

The DGAC recognizes the importance of understanding the totality of food and beverage intake at the level of food groups and basic ingredients (e.g., fruit, vegetables, whole grains, refined grains, dairy, protein foods) as well as at the level of foods as they are typically consumed, called food categories (e.g., pizza, pasta dishes, burgers, sandwiches) and how these contribute to nutrient adequacy or nutrient excess. To better understand current food intakes of the U.S. population, the Committee reviewed data on several issues, such as which of these food groups (e.g., refined grains) and food categories (e.g., sandwiches, beverages, snacks and sweets) contribute the most energy (calories), sodium, and saturated fat.

Understanding the totality of food and beverage intake also involved acknowledging that individuals purchase and procure food in a diverse array of locations, including large grocery stores, convenience stores, schools, the workplace, quick-serve restaurants, and sit-down restaurants. The DGAC examined the diet quality of the foods and meals at each major procurement point, as it is important to understand not only where foods are purchased or obtained, but also the extent to which they contribute to the overall nutritional adequacy and nutritional quality of the diet. This information may be relevant to guidance for federal nutrition programs. The DGAC also considered the diet quality of foods prepared and purchased at places such as supermarkets, but consumed at home. For example, many supermarkets have salad bars and hot food bars, but these foods are then consumed at home. However, on examination, it was determined that these types of data were not available. The DGAC also examined eating behaviors, such as meal skipping, and identifying which nutrients and how much energy are consumed at specific eating occasions and locations, because an understanding of these behaviors can help inform public policy and population as well as individual guidance.

The DGAC considered the composition of dietary patterns that were found to be linked to health outcomes in ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***. Understanding the characteristics of diets characterized as “Healthy U.S.” or “Mediterranean-style” dietary patterns and others patterns found to have health benefits, will provide specific, healthful food and beverage-based guidance for the U.S. population. These patterns are defined using dietary quality/adherence indices, [e.g., Healthy Eating Index (HEI)], based upon data-driven approaches (e.g., cluster or factor analysis), or may be self-identified patterns (e.g., vegetarian).

To address the issues described above, the DGAC presents the current status and trends in nutrient, food, food group, and food category intakes, and describes major sources of energy, sodium, added sugar, and saturated fat, and dietary pattern intake among representative samples of the U.S. population from the National Health and Nutrition Examination Survey (NHANES) What we Eat in America (WWEIA) dietary survey.[5] We also describe eating behaviors, such as number of meals per day, diet quality of foods, location of food purchase and consumption and diet quality of foods based on location where the food was purchased or consumed.

Finally, we describe the prevalence of diet-related health outcomes in the U.S. population, including obesity, diabetes, cardiovascular diseases, certain cancers, osteoporosis, congenital anomalies and psychological health (including mental health), and neurological illness (such as Alzheimer's Disease). The examination of diet-related health outcomes was more extensive than in earlier DGAC reports. The high rates of the chronic conditions and the presence of other less common, but important diet-related health problems, provided compelling reasons to study them in greater detail. These data provide a backdrop for other chapters, particularly those which examine the strength of associations between diet and health outcomes (***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***) and methods for improving disease risk outcomes and improving health at individual (***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes*** and ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*** and population levels (***Part D. Chapter 4:Food Environment and Settings***).

One of the overarching motivations for this broad examination of nutrient intake, food group and food category intake, and food purchase location is to better understand the relationship of food intake (both inadequacy and excess) and the food environment to nutrition-related health conditions. This comprehensive evaluation of food and nutrient intakes by the U.S. population (and various subgroups) along with the food and eating environment enables the consideration of factors on a broad scale that may facilitate behavior change and adoption of healthy eating practices in the population at large. Taken together, these dimensions of our analysis inform the remaining chapters in the report, which, taken together, will provide the contextual and scientific foundation for the 2015 Dietary Guidelines for Americans.

LIST OF QUESTIONS

Nutrient Intake and Nutrients of Concern

1. What are current consumption patterns of nutrients from foods and beverages by the U.S. population?
2. Of the nutrients that are underconsumed or overconsumed, including over the Tolerable Upper Limit of Intake (UL), which present a substantial public health concern?
   a. What would be the effect on food choices and overall nutrient adequacy of limiting saturated fatty acids to 6 percent of total calories by substituting mono- and polyunsaturated fatty acids?
3. Is there evidence of overconsumption of any micronutrients from consumption of fortified foods and supplements?
4. What is the level of caffeine intake derived from foods and beverages on the basis of Institute of Medicine (IOM) Dietary Reference Intakes age and sex categories in the U.S. population?
5. How well do updated USDA Food Patterns meet IOM Dietary Reference Intakes and 2010 Dietary Guidelines recommendations? How do the recommended amounts of food groups compare to current distributions of usual intakes for the U.S. population?
   a. How well do the USDA Food Patterns meet the nutritional needs of children 2 to 5 years of age and how do the recommended amounts compare to their current intakes? Given the relatively small empty calorie limit for this age group, how much flexibility is possible in food choices?
6. Can vitamin D Estimated Average Requirements (EARs) and/or Recommended Dietary Allowances (RDAs) be met with careful food choices following recommended amounts from each food group in the USDA Food Patterns? How restricted would food choices be, and how much of the vitamin D would need to come from fortified dairy and other food products?

Food Groups—Current Intakes and Trends

7. What are current consumption patterns of USDA Food Pattern food groups by the U.S. population?
   a. What is the contribution of whole grain foods, fruits and vegetables, and other food groups to (1) total fiber intake and (2) total nutrient intake in the USDA Food Patterns? What is the contribution of fruit and vegetables to current nutrient intake (focus on nutrients of concern, including fiber)?
   b. What would be the impact on the adequacy of the patterns if (1) no dairy foods were consumed, (2) if calcium was obtained from nondairy sources (including fortified foods), and (3) if the proportions of milk and yogurt to cheese were modified? What is the relationship between changes in types of beverages consumed (milk compared with sugar-sweetened beverages) and diet quality?
8. What are the trends in USDA Food Pattern food group consumption by the U.S. population?

Food Categories—Current Intakes and Sources of Energy, Nutrient, and Food Group Intakes

9. What are the current consumption patterns by food categories (i.e., foods as consumed) by the U.S. population?
10. What are the top foods contributing to energy intake by the U.S. population?
11. What are the top foods contributing to sodium, saturated fat, and added sugars intake by the U.S. population?
    a. What is the current contribution of fruit products with added sugars to intake of added sugars?
    b. What is the current contribution of vegetable products with added sodium to intake of sodium?
    c. What is the current contribution of refined grains to intake of added sugars, saturated fat, some forms of polyunsaturated fat, and sodium?
    d. What are the sources of caffeine from foods and beverages on the basis of age and sex subgroups?
12. What is the contribution of beverage types to energy intake by the U.S. population?

Eating Behaviors—Current Status and Trends

13. What are the current status and trends in the number of daily eating occasions and frequency of meal skipping? How do diet quality and energy content vary based on eating occasion?
14. What are the current status and trends in the location of meal and snack consumption and sources of food and beverages consumed at home and away from home? How do diet quality and energy content vary based on the food and beverage source?

Prevalence of Health Conditions and Trends

15. What is the current prevalence of overweight/obesity and distribution of body weight, body mass index (BMI) and abdominal obesity in the U.S. population and in specific age, sex, race/ethnicity and income groups? What are the trends in prevalence?
16. What is the relative prevalence of metabolic and cardiovascular risk factors (i.e., blood pressure, blood lipids, and diabetes) by BMI/waist circumference in the U.S. population and specific population groups?
17. What are the current rates of nutrition-related health outcomes (i.e., incidence of and mortality from cancer [breast, lung, colorectal and prostate] and prevalence of cardiovascular disease (CVD),

high blood pressure, diabetes, bone health, congenital anomalies, and neurological and psychological illness) in the overall U.S. population?

Dietary Patterns Composition

18. What is the composition of dietary patterns with evidence of positive health outcomes (e.g., Mediterranean-style patterns, Dietary Approaches to Stop Hypertension (DASH)-style patterns, patterns that closely align with the Healthy Eating Index, and vegetarian patterns) and of patterns commonly consumed in the United States? What are the similarities (and differences) within and among the dietary patterns with evidence of positive health outcomes and the commonly consumed dietary patterns?
19. To what extent does the U.S. population consume a dietary pattern that is similar to those observed to have positive health benefits (e.g., Mediterranean-style patterns, Dietary Approaches to Stop Hypertension (DASH)-style patterns, patterns that closely align with the Healthy Eating Index, and vegetarian patterns) overall and by age/sex and race/ethnic groups?
20. Using the Food Pattern Modeling process, can healthy eating patterns for vegetarians and for those who want to follow a Mediterranean-style dietary pattern be developed? How do these patterns differ from the USDA Food Patterns previously updated for use by the 2015 DGAC?

METHODOLOGY

To address questions on the current status and trends in food and nutrient intakes, the prevalence of diet-related chronic diseases in the U.S. population, and the composition of healthful dietary patterns, the DGAC relied on analysis of data from several sources and food pattern modeling analyses. Many of the questions relied on analysis of data from What We Eat in America (WWEIA), the dietary component of the National Health and Nutrition Examination Survey (NHANES), using either existing data tables or new analyses conducted by the Data Analysis Team (DAT) upon request of the DGAC (see ***Part C. Methodology***, Data Analyses section, and ***Appendix E-4: NHANES Data Used in DGAC Data Analyses***). Existing data tables were used when available to answer questions about nutrient intake, food group intake, and meal and snack consumption. In some cases, new analyses were conducted by DAT agencies to provide additional information on food or nutrient intake, for example, by specific population groups, such as pregnant women, or information on potential overconsumption of nutrients when supplement intake is considered. New WWEIA/NHANES data analyses also were used to answer questions about food category intakes, the energy content and nutrient density of foods by point of purchase and location of consumption, and the food choices of self-identified vegetarians.

Data from the U.S. Centers for Disease Control and Prevention (CDC) NHANES data tables and from the peer-reviewed literature, also were the source of information on prevalence of health conditions, including body weight status, lipid profiles, high blood pressure, and diabetes. In addition, NHANES data on biochemical indicators of diet and nutrition in the U.S. population were used to help determine nutrients that may be of public health concern. To supplement data from NHANES, additional data sources were drawn upon to answer questions on the prevalence of health conditions, including the National Health Interview Survey, the National Cancer Institute's Surveillance Epidemiology and End Results (SEER) cancer registry statistics, SEARCH for Diabetes in Youth Study (SEARCH), and heart disease and stroke statistics from the 2014 report of the American Heart Association.[6]

Some of the questions posed by the DGAC were best addressed by Food Pattern Modeling (see ***Part C. Methodology***, Special Analyses Using the USDA Food Patterns section). These included questions about the nutrient adequacy of the USDA Food Patterns, modifications of the patterns for specific population groups or to meet specific nutrient targets, and the nutrients provided by various food groups in the Patterns. In some cases, questions could be answered with modeling analyses that had been conducted for the 2005 or 2010 DGACs, and so the results of these analyses were brought forward. The modeling process also was used to develop new USDA Food Patterns based on different types of evidence: Healthy Vegetarian Patterns that take into account food choices of self-identified vegetarians, and Healthy Mediterranean-style Patterns that take into account food group intakes from studies using a Med-diet index to assess dietary patterns. The latter were compiled and summarized to answer the questions addressed on dietary patterns composition. The food group content of dietary patterns reviewed by the DGAC and found to have health benefits formed the basis for answering these questions. WWEIA food group intakes and USDA Food Pattern recommendations were compared with the food group intake data from the healthy dietary patterns as part of the answer for these questions.

The DGAC took the strengths and limitations of data analyses into account in formulating conclusion statements. The grading rubric used for questions answered using NEL systematic reviews do not apply to questions answered using data analyses. Therefore, these conclusions were not graded.

NUTRIENT INTAKE AND NUTRIENTS OF CONCERN

An overarching premise of the DGAC is that that the *Dietary Guidelines for Americans* should provide food-based guidance for obtaining the nutrients needed for optimal reproductive health, growth and development, healthy aging, and well-being across the lifespan (ages 2 years and older). Specific nutrient intake requirements are established for each sex and life-stage group by the Food and Nutrition Board of the Institute of Medicine[7] and as such, this DGAC report did not reevaluate IOM recommendations or make independent specific nutrient recommendations. Rather, the DGAC reviewed nutrient intake and biochemical measures of nutritional status and potential nutrient-related health outcomes to identify "shortfall nutrients" and "overconsumed nutrients", and then determined whether these nutrients should be designated as "nutrients of public health concern."

"Shortfall nutrients" are those that may be underconsumed either across the population or in specific groups relative to IOM-based standards, such as the Estimated Average Requirement (EAR) or the Adequate Intake (AI). The EAR is the best measure of population adequacy of nutrient intake as is it is "the average daily intake level estimated to meet the requirement of half of the healthy individuals in a particular life stage and gender group."[7 p.3] The EAR is used to estimate the prevalence of inadequate intakes within a group. The AI is "a recommended average daily nutrient intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate—used when an RDA cannot be determined."[7 p.3] A high prevalence of inadequate intake either across the U.S. population or in specific groups constitutes a shortfall nutrient.

Overconsumed nutrients are those that may be overconsumed either across the population or in specific groups related to IOM-based standards such as the Tolerable Upper Limit of Intake (UL) or other expert group standards. A high prevalence of excess intake either across the U.S. population or in specific group constitutes an overconsumed nutrient.

"Nutrients of concern" are those nutrients that may pose a substantial public health concern and the DGAC divided them into two categories—those of concern due to overconsumption and those of concern due to underconsumption. To be identified as a nutrient of concern, the DGAC used the totality of evidence, evaluating data on nutrient intake and corroborating it with biochemical markers of nutritional status, where available, and evidence for associations with health outcomes to establish nutrients of concern.

Designation as a nutrient of concern for either under- or overconsumption is intended to communicate some level of risk for which the U.S. population may need to modify eating habits. Dietary guidance can then be formulated to assist individuals in increasing or decreasing nutrients that are under- or overconsumed.

Question1: What are current consumption patterns of nutrients from foods and beverages by the U.S. population?

Source of evidence: Data analysis

Conclusion

Nutrient intake data from a representative sample of the U.S. population ages 2 years and older indicate that: vitamin A, vitamin D, vitamin E, folate, vitamin C, calcium, and magnesium are underconsumed relative to the EAR. Iron is under-consumed by adolescent and premenopausal females, including women who are pregnant. Potassium and fiber are underconsumed relative to the AI. Sodium and saturated fat are overconsumed relative to the UL or other standards for maximal intake.

Implications

A dietary pattern emphasizing a variety of nutrient-dense foods will help shift individual and population consumption toward recommended intake levels for nutrients of public health concern.

The U.S. population should increase consumption of foods rich in vitamin A, vitamin D, vitamin E, folate, vitamin C, calcium, and magnesium. Adolescent and premenopausal females should increase consumption of foods rich in iron. Heme iron from lean meats is highly bioavailable, hence, an excellent source.[8] A diet emphasizing a variety of nutrient-dense foods will help shift consumption toward the recommended intake levels of these shortfall nutrients. The U.S. population should increase consumption of foods rich in potassium and fiber. A diet emphasizing a variety of nutrient-dense foods will help ensure optimal intake of these shortfall nutrients. In particular, fruit, vegetables and whole grains are excellent sources of vitamin A, C, folate, fiber, magnesium and potassium. The U.S. population should make concerted and focused efforts to decrease consumption of sodium and saturated fat.

The USDA Food Patterns provide guidance for consumption of a nutrient-dense, energy-balanced diet. Implementation of eating a healthy diet that is energy balanced while providing sufficient intake of shortfall nutrients without exceeding intake of overconsumed nutrients can be achieved through a variety of successful behavioral approaches as described in ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change***. Environmental and policy approaches are also important in helping the U.S. population achieve a healthy diet (see also ***Part D. Chapter 4: Food Environment and Settings***). Federal nutrition assistance programs are a key aspect of providing critical nutrients for growth, development and long-term health for children, those with limited income and older Americans.

Review of the Evidence

To determine nutritional adequacy, the DGAC used 2007-2010 NHANES/WWEIA data to examine the intake distributions for 11 vitamins (vitamin A, vitamin B_6, vitamin B_{12}, vitamin C, vitamin D, vitamin E, vitamin K, folate, thiamin, niacin, and riboflavin), nine minerals (calcium, copper, iron, magnesium, phosphorous, potassium, selenium, sodium, and zinc), energy, macronutrients (total fat, saturated fat, polyunsaturated fat [including 18:2 and 18:3], protein, carbohydrate), and other compounds or components (fiber, carotenoids [alpha-carotene, beta-carotene, lycopene, lutein + zeaxanthin], caffeine, cholesterol, and choline) (see ***Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups***). The DGAC compared the intake estimates across the population age distribution to the Dietary Reference Intakes. The committee used data from foods and beverages as well as foods and beverages plus dietary supplements when supplement data were available. For nutrients with an EAR, the DGAC considered shortfall nutrients to be those where a substantial proportion of either the total population or specific age and sex subgroups had intake estimates below the EAR. Although multiple approaches can be used to estimate the prevalence of nutrient inadequacy in a population, the DGAC used the EAR cut point method.[7] Figure D1.1 shows the percent of the U.S. population with usual intakes below the EAR. From Figure D1.1, the DGAC determined that vitamin D, vitamin E, magnesium, calcium, vitamin A and vitamin C were shortfall nutrients and that there may be a high prevalence of inadequate dietary intake of these nutrients.

Of the nutrients with an AI (vitamin K, choline, dietary fiber, and potassium), the DGAC determined that a low proportion of the population had fiber and potassium intakes above the AI and so potassium and fiber were therefore considered to be underconsumed (Figure D1.2).

Sodium and saturated fat were examined as potentially overconsumed nutrients in relation to the UL (for sodium), and the maximum level from the 2010 Dietary Guidelines of less than 10 percent of calories from saturated fat (for saturated fat). From 63 percent to 91 percent of females and 81 percent to 97 percent of males consumed more than the UL for sodium (Figure D1.3). From 67 percent to 92 percent of females and from 57 percent to 84 percent of males consumed more than 10 percent of calories from saturated fat (Figure D1.4). Therefore, sodium and saturated fat were both determined to be overconsumed by the U.S. population (see ***Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups*** and ***Appendix E-2.2: Usual intake distributions as a percent of energy for fatty acids and macronutrients, 2007-2010, by age/sex groups***).

The DGAC examined population intakes of specific nutrients by age, sex, race/ethnicity, pregnancy status, and acculturation status.

Age and Sex

In addition to the age groups shown in Figures D1.1 and D1.2, the DGAC was interested in understanding the intake of shortfall nutrients in older adults (71 to 79 years and 80 years and older). Calcium intake from foods and beverages did not meet the EAR for older persons, where 71 percent of males and 81 percent of females ages 71 years and older had intakes below the EAR. For these analyses calcium from dietary supplements was also considered. When total intake of foods + beverage + dietary supplements containing calcium was considered, then the proportion of the older adults below the EAR improved to 55 percent for men and 49 percent for women over the age of 71 years. For vitamin D intakes from food and beverages only, about 93 percent of older males and more than 97 percent of older females had intakes below the EAR. Similar to the findings for calcium, intakes improved when considering total intake from foods and beverages plus dietary supplements. The proportions of older adult below the EAR dropped to 52 percent for both males and females older than 71 years.

Fiber was a shortfall nutrient for older adults, where only 4 percent of men and 13 percent of women had a dietary intake of fiber above the AI. Potassium also was a shortfall nutrient for both older males and females, where less than 3 percent of both groups had intakes above the AI. Use of dietary supplements containing potassium did not change the proportion of the older adults with intakes above the AI.

Protein was not identified as a shortfall nutrient for the overall older adult population but it should be noted that 6 percent of men older than 80 years and 11 percent of women older than 80 years old had protein intakes that were below the protein EAR (g/kg/body weight).

The sample size for the older participants in WWEIA 2007-2010 is small compared to other age groupings in the survey sample and despite the excellent population weights used in the WWEIA dataset, the estimates should be viewed with caution because of the limited sample (see ***Appendix E-2.3 Usual nutrient intakes for individuals age 71 years and older***).

Race/Ethnicity

The DGAC examined the shortfall nutrients by race/ethnicity using the following groups: non-Hispanic white, non-Hispanic Black, Mexican-American, and all Hispanic combined (other race/ethnic subgroups not available). For certain shortfall nutrients, non-Hispanic whites have the highest intakes. These include vitamin A, vitamin E, magnesium, folate, iron, potassium, vitamin D, and calcium. Mexican-Americans have the highest intakes of fiber, while all Hispanics combined have the highest intakes of vitamin C. Non-Hispanic Blacks have the lowest intake for most of the shortfall nutrients (Table D1.1). We note that evaluation of intakes relative to the EAR or AI are the most appropriate for assessment of populations, instead of the mean intakes, but for the race/ethnicity groups, only the mean data are available.

Pregnancy

Many of the shortfall nutrients in the general population also were shortfall nutrients among women who are pregnant. Among this group, 26 percent were below the EAR for vitamin A intake and 30 percent had vitamin C intakes below the EAR. For vitamin D, 90 percent had intakes below the EAR and for vitamin E, 94 percent had intakes below the EAR. Calcium intake was also low, where 24 percent had intakes below the EAR, and for folate, 29 percent had intakes below the EAR. Notably, 96 percent of women who were pregnant had iron intakes below the EAR (Table D1.2 and ***Appendix E-2.4: Usual intake distributions, 2007-2010, for pregnant and non-pregnant women in the U.S. ages 19-50 years***).

Fiber was a shortfall nutrient for women who were pregnant, as only 8 percent had fiber intakes above the AI. For potassium only 3 percent had intakes above the AI (Table D1.2).

It is important to note that the sample size for women who were pregnant in WWEIA 2007-2010 is very small (n=133 respondents), so the estimates should be interpreted with caution and the generalizability of the data to all women in the United States who were pregnant is limited.

Acculturation

The U.S. population is highly diverse in terms of race, ethnicity, and cultural origin. Many people immigrate to the United States from all over the world and each comes with distinct dietary habits and cultural beliefs about food and food patterns.[9] Acculturation is defined as the process by which immigrants adopt the attitudes, values, customs, beliefs, and behaviors of a new culture. Acculturation is the gradual exchange between immigrants' original attitudes and behavior and those of the host culture. [10, 11] The DGAC appreciates that many immigrants have difficulties purchasing and preparing foods familiar to them either because the ingredients are not available or the ingredients may be too expensive. A large and growing body of research suggests that the extent of an individual or family's acculturation status may be a predictor of dietary intake and that together, diet and acculturation status may influence health status or disease risk.[9, 10, 12, 13] For this reason, the DGAC felt it was important to examine dietary intake by acculturation status, particularly for shortfall nutrients and nutrients of concern. Additional information on acculturation and diet appears in Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change.

NHANES collects data on some of the variables that can be used to create an acculturation variable, including whether respondents were born outside the United States in a Spanish-speaking country or born outside the United States in a non-Spanish speaking country, their race/ethnicity, and number of years they have resided in the United States.[14] Upon reviewing the data, however, the DGAC found that the sample size was far too small to create meaningful variables to indicate "low acculturation status" or "high acculturation status." The DGAC views this lack of ability to analyze the WWEIA data by acculturation status as a limitation of the available data. It is a very important area that needs further research, particularly when informing nutrition programs for new residents of the United States.

Food Insecurity Status

Readers are referred to ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*** and ***Part D. Chapter 5: Food Sustainability and Safety*** for more detailed discussions of food insecurity and food security issues. For this section of the report, the DGAC determined that it was important to evaluate nutrient intake, particularly for the shortfall nutrients by income status, which can be a marker of food insecurity. For these data analyses, we used the standard cutpoints of less than 131 percent of the poverty index, 131 to 185 percent of the poverty index and more than 185 percent of the poverty index and examined calcium, potassium, fiber and vitamin D (Table D1.3). In general, respondents (all ages 2 years and older) from households with higher income (more than 185 percent of the poverty index) had higher intakes of calcium, potassium, fiber, and vitamin D. Notably, in some of the very young age groups (2 to 5 years), intakes of potassium, fiber, and vitamin D were comparable across income groups, while calcium was highest in those coming from households at the 131 to 185 percent of the poverty index ratio. It may be that many of the households of lower income with small children are receiving important benefits from federal nutrition assistance programs, which could be helping to generate comparability in the intake of shortfall nutrients across the income groups.

For additional details on this body of evidence, visit:

- Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups
- Appendix E-2.2: Usual intake distributions as a percent of energy for fatty acids and macronutrients, 2007-2010, by age/sex groups
- Appendix E-2.3: Usual intakes for Individuals age 71 and older
- Appendix E-2.4: Usual intake distributions, 2007-2010, for pregnant and non-pregnant women in the U.S. ages 19-50 years
- Mean intake of nutrients, 2003-2004, 2005-2006, 2007-2008, and 2009-2010, by race/ethnicity and by percent of the poverty threshold. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349.
- Usual intake of selected nutrients, 2001-2002, 2003-2006, or 2005-2006, by age/sex groups. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=22659.

Question 2: Of the nutrients that are underconsumed or overconsumed, including over the Tolerable Upper Limit of Intake (UL), which present a substantial public health concern?

Source of evidence: Data analysis

Conclusion

Nutrient intake data, together with nutritional biomarker and health outcomes data indicate that vitamin D, calcium, potassium, and fiber are underconsumed and may pose a public health concern. Iron also is a nutrient of public health concern for adolescent and premenopausal females.

Nutrient intake data, together with nutritional biomarker and health outcomes data indicate that sodium and saturated fat are overconsumed and may pose a public health concern.

Implications

The DGAC recommends that strategies be developed and implemented at both the individual and the population level to improve intake of nutrients of public health concern.

Review of the Evidence

These conclusions were reached using a 3-pronged approach, including analysis of data from What We Eat in America, NHANES dietary survey (2007-2010) (see ***Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups***), the Second National Report on Biochemical Indices of Diet and Nutrition in the U.S. Population, Centers for Disease Control and Prevention, 2012,[3] and data on the prevalence of health conditions, from the CDC. The DGAC used the totality of evidence from these sources.

Nutrients of Concern for Underconsumption

Vitamin D. Vitamin D is unequivocally essential for skeletal health.[15] The 2010 IOM report on Dietary Reference Intakes for calcium and vitamin D[15] established new DRIs for vitamin D based on established and consistent evidence for vitamin D's role in skeletal health. Numerous other functions exist for vitamin D, including its role as a transcription factor for more than 200 genes, roles in apoptosis and cellular proliferation, and a growing body of evidence supporting vitamin D's role in preventing cancer, cardiovascular disease, and other chronic diseases.[16-25]

The IOM's rationale for setting the DRI was limited to vitamin D's role in skeletal health, as the evidence for the other diseases was not sufficiently mature at the time of the committee's evidence review. Therefore, any interpretations for vitamin D intake and its classification as a shortfall nutrient and a nutrient of public health concern are restricted to this role in skeletal health. Given the high prevalence of osteoporosis and low bone density, particularly in the older women (see Question 17, on health conditions, below) and due to vitamin D's critical role in bone health, the Committee determined that vitamin D should be classified as an underconsumed nutrient of public health concern.

Vitamin D can be obtained from the diet by consuming fluid milk and some milk products (e.g., some yogurts), fortified juices, finfish, fortified breakfast cereals and some fortified grain products as well as dietary supplements (Table D1.4 and ***Appendix E-3.3: Meeting Vitamin D Recommended Intakes in USDA Food Patterns***). Vitamin D also is synthesized endogenously through cutaneous exposure to ultraviolet-B sunlight. The primary biomarker to assess vitamin D status is serum/plasma 25(OH)D concentrations. This biomarker represents dietary intake plus endogenous synthesis.

Dietary intake of vitamin D in the United States is low and well below the EAR values (Figure D1.1) for all age and sex groups. In addition, independent evidence of nutrient shortfall comes from data demonstrating low serum/plasma 25-hydroxyvitamin D concentrations from the CDC biomarker data, particularly for young adults (ages 20 to 39 years), middle-aged adults (ages 40 to 59 years), non-Hispanic Blacks and Mexican-Americans (Table D1.5). The correlation of dietary intake with the serum measures of 25-hydroxyvitamin D) is modest. In addition several factors predict serum concentrations of nutrients in addition to dietary intake.[19] The DGAC and other expert panels, including the IOM, acknowledge that while numerous variables, including sun exposure and endogenous synthesis, are strong predictors of serum vitamin D status, dietary intake of vitamin D is a critical contributor to vitamin D status.[26, 27] Further, while there is some degree of unexplained variation in serum/plasma 25-hydroxyvitamin D concentrations, the biomarker is still important for evaluating vitamin D inadequacy. Various statistical approaches have been used to evaluate and confirm population inadequacy using the biomarker data. [28] Of note, the CDC biomarker data reviewed by the DGAC should be interpreted knowing that the NHANES Mobile Examination Clinics do not sample residents of northern climates in winter months due to variable sunshine exposure and the possibility that high levels of sunshine exposure may be overrepresented in NHANES. In other words, higher values in the dataset may be over-represented due to the summer blood draws, when 25-OHD tends to be higher from sun exposure and deficiencies may be under represented. [15p.471-473]

The DGAC's decision to classify vitamin D as a nutrient of concern is similar to the conclusion reached by the U.S. Food and Drug Administration (FDA), which designated vitamin D as a nutrient of "public health significance" in its recent review of evidence in publishing a Proposed Rule on the Nutrition Facts label.[29] In addition, multiple national and international groups, including the American Academy of Pediatrics (AAP),[30] the Endocrine Society[31] and the National Osteoporosis Foundation [32] have recommended that strategies to achieve the RDA or higher levels of vitamin D intake could include consumption of fortified foods, broadening the range of dairy products that are fortified, and consideration, in some cases, of the use of a vitamin D supplement or a multivitamin including vitamin D. Such a use is especially appropriate where sunshine exposure is more limited due to climate or sunblock use.

Calcium. Calcium plays a major role in skeletal health and also is essential for proper functioning of the circulatory system, nerve transmission, muscle contractility, cell signaling pathways, and vascular integrity.[15] Dietary calcium is obtained from fluid milk and milk products, fortified juices, and some plant foods, including soy and soy products and vegetables (see Table D1.6 and ***Appendix E-3.2: Food Group Contributions***). However, the bioavailability of calcium from plant foods is lower than from animal foods, such as dairy.

The DGAC reviewed the dietary intake data from WWEIA. Intakes of calcium were often far below the EAR, especially among adolescent girls and adults (Figure D1.1). Even though a reliable biomarker for calcium does not exist, because of its strong link to health outcomes and the risks associated with osteoporosis (see Question 17 on health conditions, below), the DGAC designated calcium as a nutrient of public health concern for underconsumption. In addition, the DGAC also notes that calcium is an underconsumed nutrient of public health concern among pregnant women. This conclusion concurs with the FDA's review that designated calcium as a nutrient of "public health significance" in its recent review of evidence in publishing a Proposed Rule on the Nutrition Facts label.[29]

Strategies to improve calcium intake include increased dairy or fortified products that are important sources of calcium. Concern about the safety of calcium supplements and a relative lack of data about the health benefits of such supplements limit recommendations to use supplementation as a strategy to meet the RDA for calcium, compared to using fortified foods.

The subgroups of particular concern with regard to intake are preadolescent and adolescent females, pregnant females, and middle aged and older females (see Question 1, above).

Potassium. Potassium is the major intracellular cation and it plays critical roles in muscle function, cardiac function, and regulation of blood pressure. Potassium adequacy is also critical for health, as deficiency adversely affects numerous organ systems including the musculoskeletal, renal, and cardiovascular systems. The primary biomarker to assess potassium intake is urinary potassium, and these data are not available in the CDC biomarker dataset. The DGAC designated potassium as a nutrient of public health concern due to its general under consumption relative to the AI across the U.S. population and its association with hypertension and cardiovascular diseases, two common adverse diet-related health outcomes in the United States (see Question 17on health conditions, below). This conclusion concurs with the FDA's review that designated potassium as a nutrient of "public health significance" in its recent review of evidence in publishing a Proposed Rule on the Nutrition Facts label.[29] Even though underconsumption was evident across the population (see Question 1, above), there is a particular concern for middle-aged and older adults, who are at increased risk for cardiovascular diseases (see Question 17). Fruits, vegetables, and legumes are all important sources of potassium (Table D1.7).

Fiber. Dietary fibers are non-digestible carbohydrates, primarily from plant foods, such as whole grains, legumes, fruits and vegetables (Table D1.8). The most important and well-recognized role for fiber is in colonic health and maintenance of proper laxation, but a growing body of evidence also suggests that fiber may play a role in preventing coronary heart disease, colorectal and other cancers, type 2 diabetes, and obesity.[33] The AI for fiber is based on an intake level associated with the greatest reduction in the risk of coronary heart disease. There are no available biomarkers for fiber intake, so the designation as a nutrient of public health concern is based on the very low dietary intakes across all sectors of the U.S. population and its important contribution to health. Because the average intake levels of dietary fiber are half the recommended levels, achieving the recommendation requires selecting high-fiber cereals and whole grains and -meeting current recommendations for fruits and vegetables.

Iron. Iron is an essential mineral whose primary function is to transport oxygen in the blood. Inadequate iron status in the form of iron deficiency anemia leads to poor growth and development and the potential for cognitive deficits in children. Excellent sources of heme iron include red meats, enriched cereal grains, and fortified breakfast cereals (Table D1.9). Dietary intake estimates, together with the CDC nutritional biomarker data indicate that iron is a nutrient of concern for children, premenopausal females, and during pregnancy. Among women who are pregnant, 96 percent are below the EAR for iron intake. Serum ferritin is the biochemical marker used by NHANES and the CDC to evaluate iron status in the U.S. population. These data show that children and women of childbearing age are at risk of iron deficiency anemia. Risk of iron deficiency anemia also is higher among Mexican-American and non-Hispanic Black women than among non-Hispanic white women.[3] Taken together, the DGAC concluded that iron was an underconsumed nutrient of public health concern for adolescent and premenopausal women and women who are pregnant. This conclusion concurs with the

FDA's designated iron as a nutrient of "public health significance" in its recent review of evidence in publishing a Proposed Rule on the Nutrition Facts label.[29]

Nutrients of concern for overconsumption

Sodium. Sodium is the major cation in extracellular fluid that maintains extracelluar fluid volume and plasma volume. It also functions in membrane potential activation and active transport of molecules across cell membranes. In excess, sodium is associated with several adverse health events, particularly hypertension.[34] The DGAC treated sodium as a cross-cutting topic for dietary intake and health outcomes, and a sodium working group was convened. Details on sodium, including dietary sources and health outcomes-related data are found in ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance***). Current sodium intakes of the U.S. population far exceed the UL for all age and sex groups (Figure D1.3). Due to the critical link of sodium intake to health and that intake exceed recommendations, sodium was designated as a nutrient of public health concern for overconsumption across the entire U.S. population.

Saturated fat. The DGAC used the 2013 American Heart Association/American College of Cardiology (AHA/ACC) report on lifestyle management to reduce CVD risk[2] for its evaluation of saturated fat intake. The DGAC concurred with the AHA/ACC report that saturated fat intake exceeds current recommendations in the United States and that lower levels of consumption would further reduce the population level risk of CVD. The DGAC also convened a working group on saturated fat (see ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance*** for details). In addition, the DGAC conducted food pattern modeling to demonstrate the dietary changes that would be necessary to have diets with various levels of saturated fat as a percent of total energy (see USDA Food Patterns Modeling Report in ***Appendix E-3.5: Reducing Saturated Fats in the USDA Food Patterns***). It is important to note that the median intake of saturated fat in the United States was 11.1 percent of total energy for all age groups in the 2007-2010 WWEIA data. However, a large majority (71 percent) of the total population consumed more than 10 percent of calories from saturated fat, with a range by age group from 57 percent to 92 percent (Figure D1.4). Further, 65 percent to 69 percent of the age groups at highest risk of CVD (males and females older than age 50 years) had intakes more than 10 percent of total calories were from saturated fat, the DGAC concluded that the U.S. population should continue to monitor saturated fat intake. Saturated fat is still a nutrient of concern for overconsumption, particularly for those older than the age of 50 years.

Cholesterol. Previously, the Dietary Guidelines for Americans recommended that cholesterol intake be limited to no more than 300 mg/day. The 2015 DGAC will not bring forward this recommendation because available evidence shows no appreciable relationship between consumption of dietary cholesterol and serum cholesterol, consistent with the conclusions of the AHA/ACC report.[2, 35] Cholesterol is not a nutrient of concern for overconsumption.

For additional details on this body of evidence, visit:

- CDC report, Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population 2012. Available from: http://www.cdc.gov/nutritionreport/pdf/Nutrition_Book_complete508_final.pdf.
- Food Labeling: Revision of the Nutrition and Supplement Facts Labels; Proposed Rule. Available from: http://www.gpo.gov/fdsys/pkg/FR-2014-03-03/pdf/2014-04387.pdf.
- Appendix E-3.2: Food Group Contributions to Nutrients in the USDA Food Patterns and Current Nutrient Intakes
- Appendix E-3.3: Meeting Vitamin D Recommended Intakes in USDA Food Patterns
- Appendix E-3.5: Reducing Saturated Fats in the USDA Food Patterns

Question 3: Is there evidence of overconsumption of any micronutrients from consumption of fortified foods and supplements?

Source of evidence: Data analysis

Conclusion

Dietary patterns among Americans, including typical use of fortified foods, rarely lead to overconsumption of folate, calcium, iron, or vitamin D. However, each of these nutrients, as well as other nutrients, are overconsumed in <u>some</u> supplement users, especially those taking high-dose supplements.

Implications

The public may safely use dietary supplements containing RDA level of nutrients, so long as total intake from diet plus supplements does not exceed the UL. Use of products with high doses of nutrients, such that total intake exceeds the UL, should be discussed with a Registered Dietitian or other qualified health care provider.

Supplement users should seek guidance about factors such as whether the amount of nutrients in supplements exceeds the UL for those nutrients. Monitoring of dietary patterns in supplement users should continue to be done, with attention paid to the highest risk groups, such as children and women who are pregnant.

Review of the Evidence

These conclusions were based on analysis of usual intake data for selected nutrients from foods and supplements from WWEIA, NHANES dietary survey (2007-2010) (see ***Appendix E-2.5: Usual intake distributions for supplement users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/sex groups*** and ***Appendix E-2.6: Usual intake distributions for non-supplement users for folate,***

folic acid, vitamin D, calcium, and iron, 2007-2010, by age/sex groups). Nutrients were selected if the DGAC had identified them as a shortfall nutrient and if supplemental intake data were available in WWEIA (Figure D1.5). When possible the total nutrient exposure was considered (food + supplements). The overconsumed nutrients (saturated fat and sodium) are not contained in most dietary supplements so that overconsumed nutrients were not considered for this question.

Folate. The use of supplemental folic acid exceeds the established UL in a small proportion of children, especially those younger than age 9 years. However, this UL is not based on clinical toxicity data in this population and exceeding the UL is primarily associated with supplement use.[36] The risk associated with usual folate intakes among children in the United States is considered low, but caution should be used in advising supplements for children younger than age 9 years.

Calcium. Dietary calcium intake greater than 2000 mg/day (UL) are seen in up to about 20 percent of females, and 15 percent of adult males older than age 50 years. These high intakes are driven primarily by a historical perspective that very high calcium supplement usage may decrease the risk of osteoporosis. Concern exists about the safety of such high intakes and the possible association with CVD risk and little, if any, current evidence supports intakes of calcium above the UL for the purpose of decreasing osteoporosis.[15] Of note, the World Health Organization recommends high dose calcium supplementation (1.5-2 g/day) to prevent hypertensive disorders of pregnancy.[37] This recommendation is not widely followed among low-risk women in the United States. However, use of calcium supplements does not appear to pose a health risk related to overconsumption of calcium.[37]

Iron. In adults of all ages, a small proportion of iron supplement users have intakes above the UL. Concerns related both to cardiovascular health and oxidant damage exist, but are not well-defined. Iron supplementation is very common during early childhood and pregnancy, but is unlikely to pose a health risk.[8]

Vitamin D. Overconsumption of vitamin D occurs when individuals take high dose supplements, usually over a long period of time.[15] The UL of 4000 IU/day is commonly exceeded by individuals with or without the guidance of a physician.[15] In general, it is unlikely that most supplement users, who limit themselves to 10,000 IU/day or less, will have any evidence of toxicity, but a greater risk may exist among some groups, including small children. Those who take high dose supplements often have their serum/plasma 25-hydroxyvitamin D concentrations monitored and this can be helpful although no clearly toxic level of 25-hydroxyvitamin D in the blood is known. Overall, the population risk of overconsumption of vitamin D leading to toxic effects, including hypercalcemia or other clinical symptoms, is uncommon.[38]

For additional details on this body of evidence, visit:

- Appendix E-2.5: Usual intake distributions for supplement users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/sex groups

- Appendix E-2.6: Usual intake distributions for non-supplement users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/sex groups

Question 4: What is the level of caffeine intake derived from foods and beverages on the basis of Institute of Medicine (IOM) Dietary Reference Intakes age and sex categories in the U.S. population?

Source of evidence: Data analysis

Conclusion

In general, intakes of caffeine do not exceed what is currently considered safe levels in any age group. Some young adults may have moderately high intakes. There is less certainty about the safe level of intake in children and adolescents. However, routine consumption patterns do not suggest that excessive intakes are common in these groups.

Implications

The public may safely consume caffeine-containing beverages, such as coffee and tea. However, children, adolescents, and women who are pregnant or considering pregnancy should not consume very high levels of caffeine from beverages or supplements (e.g., energy shots, fortified foods).

Monitoring of caffeine intake should be continued with special attention to high-risk groups, including children and women who are pregnant. Families should monitor caffeine intake in children, and high-dose caffeine supplementations should not be used.

For additional details on caffeine safety please see ***Part D. Chapter 5: Food Sustainability and Safety.***

Review of the Evidence

These conclusions were reached based on analysis of usual intake data from the WWEIA, NHANES dietary survey (2007-2010). Data on intakes of caffeine show that intakes in adults (Figure D1.6) peak at ages 31 to 70 years, and that younger adults (ages 19 to 30 years), older adults (71 years and older), have lower intakes. Relatively few individuals (less than 10 percent) have intakes above 400 mg/day (see ***Appendix E-2.1: Usual intake distributions, 2007-2010, by age/sex groups***), which is a level set as a moderate intake by some groups, including Health Canada.

In children, caffeine intakes increase with age (Figure D1.7) with median intakes remaining below 100 mg/day in adolescents (14 to 18 years). Recommended intakes from Health Canada of no more than 2.5 mg/kg/day, or about 85 mg/day total in children ages 10 to 12 years [39] are not exceeded by most children and adolescents although recent data indicates that as many as 10 percent of children and adolescents ages 12 to 19 years exceed this intake level.[40] These data demonstrate that caregivers should monitor caffeine intake in children and exercise caution with respect to time-dependent changes in caffeine intake.

For additional details on this body of evidence, visit:

- Appendix E-2.1: Usual intake distributions, 2007-2010 by age/sex groups

Question 5: How well do updated USDA Food Patterns[♦] meet IOM Dietary Reference Intakes and 2010 Dietary Guidelines recommendations? How do the recommended amounts of food groups compare to current distributions of usual intakes for the U.S. population?

Source of evidence: Food Pattern Modeling

Conclusion

USDA Food Patterns across a broad range of ages and energy intake meet most goals for nutrient adequacy. The nutrients of public health concern for which the patterns do not meet recommendations are potassium and vitamin D. Recommended amounts of food groups and their component subgroups fall within the broad range of usual food group intake distributions for the U.S. population.

Implications

The USDA Food Patterns provide guidance for consuming a nutrient-dense, energy-balanced diet. To achieve nutrient adequacy, the U.S. population should be advised to consume dietary patterns consistent with the USDA Food Patterns.

Continued vigilance is needed to ensure that food intake patterns meet but do not exceed DRI targets in all age groups. The Patterns meet recommended intake levels or limits for almost all nutrients, including the following nutrients of concern: calcium, fiber, iron, sodium, and saturated fat. Two nutrients of concern (potassium and vitamin D) are not provided in recommended levels by the Patterns. Therefore, potassium and vitamin D intakes require assessment both of individual intake and population intake patterns of foods or supplements to ensure that needs for physiological functioning are met. Meeting the needs for these nutrients may require careful attention to excellent natural sources, food enriched or fortified with the nutrients, or, in some cases, consideration of supplements.

[♦] The USDA Food Patterns referred to in this question are the same as the "Healthy U.S.-style Food Pattern" described later in this chapter (see Question 20). We use the term USDA Food Patterns in this question because the development of the Healthy U.S.-style Food Pattern and two related USDA Food Patterns had not occurred when the Committee addressed this question.

Following the recommended food intake pattern increases intakes of whole grains, vegetables, fruits, and fat-free/low fat dairy and thus increases the likelihood of meeting recommendations for these food groups while decreasing intake of the food components refined grains, solid fats, and added sugars. Following the recommended pattern also decreases intake of the nutrients sodium and saturated fat.

In some situations, specific foods or dietary supplements may be used to increase underconsumed nutrient intakes not met through the USDA Food Patterns.

Review of the Evidence

These conclusions were reached based on the results of the Food Pattern Modeling Report on Adequacy of the USDA Food Patterns. The USDA Food Patterns are intended to represent the types and amounts of foods that will provide nutrients sufficient to meet IOM nutrient recommendations and Dietary Guidelines for Americans recommendations. The Food Patterns are updated every 5 years during the deliberations of the Dietary Guidelines Advisory Committee, and are presented to the Committee for their assessment of the Food Patterns' adequacy. As part of the update, amounts recommended from each food group may be modified to reach all or most of the specified goals. In addition, the amounts from each food group are compared to usual dietary intake patterns of the U.S. population, and are kept within the normal range of consumption. The current analysis, using the 2010 USDA Food Patterns as a baseline, found that the recommended amounts of each food group met almost all nutrient goals and were within the normal range of consumption. Therefore, no updates to the food group amounts from 2010 were needed.

As shown in Figure D1.8, for many nutrients, amounts of a nutrient in the patterns are well above the RDA or AI. Protein, phosphorus, zinc, copper, selenium, manganese, vitamin C, thiamin, riboflavin, niacin, vitamin K, folate, vitamin B_6, and vitamin B12 are above the goal amounts for all age/sex groups.

In contrast, some nutrients are just above the RDA or AI, or marginally below (90 to 100%) goal amounts for several age/sex groups. These include calcium, iron, and magnesium. The percents of the RDA shown in Figure D1.8 are for the lowest calorie level assigned to these age/sex groups—the level applicable for a sedentary/less active physical activity level.

The nutrients for which adequacy goals are not met in almost all patterns are potassium, vitamin D, vitamin E, and choline. Due to the new higher RDA for vitamin D that was recommended by the 2011 Committee to Review Dietary Reference Intakes for vitamin D and calcium,[15] amounts in the patterns are a much smaller percentage of the RDA than previously, and no pattern meets the EAR for vitamin D. To determine if vitamin D recommendations could be met while following the food group recommendations of the USDA Food Patterns, thorough, careful selection of specific foods within each food group, an additional modeling analysis was conducted and reported below (see Question 6).

The USDA Food Intake patterns provide a healthy pattern of food choices and to accomplish this, these patterns deviate from typical food intakes in a number of ways. To ensure that the patterns do not deviate too far beyond the range of what the U.S. population could feasibly consume, the recommended intake amounts in the patterns from each food group or subgroup plus oils were compared to the median and either the 5th or 95th percentile of usual intakes of the population, from WWEIA/NHANES 2007-2010.[41] Table A6 of the Adequacy of the USDA Food Patterns Modeling Report (see ***Appendix E-3.1***, Table A6) shows the comparison of food group recommended intakes to median and 95th percentile intakes.

For underconsumed food groups, such as fruits and vegetables, recommended amounts in the patterns are generally between the median and 95th percentiles of usual intakes. (see ***Appendix E-3.1: Adequacy of the USDA Food Patterns***, Table A6) This indicates that the Food Patterns recommend amounts within the broad intake range for the population. However, for some specific food groups and some age/sex groups, such as vegetables for males ages 14 to 18 years, food group amounts in the Patterns are somewhat above the 95th percentile of usual intake. One exception to this is whole grain recommendations in the Patterns, which are well above the 95th percentile of usual intakes for all age/sex groups. Conversely, refined grain recommendations in the patterns are very low compared to usual intakes—about the 5th percentile of intake for most age/sex groups. This indicates that a major shift from refined to whole grains is needed in order to meet recommendations.

For Food Pattern components that are overconsumed, the limits in the patterns for maximum solid fat and added sugars (see Questions 7 and 8 for more information on solid fats and added sugars) also are very low compared to usual intake amounts—at approximately the 5th percentile of usual intakes for most age/sex groups, and less than the 5th percentile of usual intakes for boys and girls ages 2 to 13 years. (see ***Appendix E-3.1: Adequacy of the USDA Food Patterns***, Table A6)

An additional modeling analysis was conducted to answer the questions: How well do the USDA Food Patterns meet the nutritional needs of children ages 2 to 5 years and how do the recommended amounts compare to their current intakes? Given the relatively small empty calorie limit for this age group, how much flexibility is possible in food choices? (see ***Appendix E-3.4: USDA Food Patterns—Adequacy for Young Children***)

The nutritional needs and the diets of young children are different in some important ways from the nutritional needs and diets of older children and adults. Therefore, this modeling analysis focused on the adequacy of the Patterns for young children, given these differences. Nutrient profiles for the Dairy and Fruit groups were adjusted to better reflect the food choices within these groups of young children. The adjusted Dairy group nutrient profile for young children is based on 70 percent fluid milk, 25 percent cheese, 3.5 percent yogurt, and 1.5 percent soymilk. In contrast, the profile for the overall population is based on 51 percent fluid milk, 45 percent cheese, 2.5 percent yogurt, and 1.5 percent soymilk. In addition, 1 percent milk rather than fat-free milk was used as the representative food for fluid milk. The adjusted Fruit group nutrient profile for young children is based on 42 percent fruit juice and 58 percent whole fruit. In contrast, overall population intake is about 33 percent juice and 67 percent whole fruit. With these adjustments, the adequacy of the Patterns did not change, but amounts of potassium, vitamins D, A, C, and folate increased slightly, and sodium decreased slightly. The amounts recommended in the USDA Food Patterns fall within the broad range of usual intakes by this age group for most food groups and subgroups (see ***Appendix E-3.1: Adequacy of the USDA Food Patterns,*** Table A6).

In addition, the young children's nutrient profiles were higher in energy, mainly due to the use of 1 percent rather than fat-free milk. Therefore, the amount of calories that could be allowed from solid fats and added sugars was adjusted down to keep the Patterns isocaloric. This resulted in limited flexibility in food choices when following the Patterns, especially for children ages 4 and 5 years for whom 2½ cup equivalents (cup eqs) from the Dairy group is recommended (the Patterns for children ages 2 and 3 years recommend 2 cup eqs). Options tested to increase flexibility in food choices included a small reduction of 1/2 ounce eq in the amount of Protein Foods, or a change from 1 percent milk to fat-free milk at 4 years of age. These changes did not result in lower nutrient adequacy levels.

For additional details on this body of evidence, visit:

- Appendix E-3.1: Adequacy of the USDA Food Patterns
- Appendix E-3.4: USDA Food Patterns—Adequacy for Young Children

Question 6: Can vitamin D Estimated Average Requirements and/or Recommended Dietary Allowances be met with careful food choices following recommended amounts from each food group in the USDA Food Patterns? How restricted would food choices be, and how much of the vitamin D would need to come from fortified dairy and other food products?

Source of evidence: Food Pattern Modeling

Conclusion

Through the use of a diet rich in seafood and fortified foods, EAR, but not RDA, levels of vitamin D can be achieved. Additional fortification or supplementation strategies would be needed to reach RDA levels of vitamin D intake consistently, especially in individuals with low intakes of fish/seafood or fortified dairy foods, other fortified foods (e.g. breakfast cereals) and beverages.

Implications

Diet is an important aspect of achieving vitamin D intake targets. The U.S. population should be encouraged to choose foods and beverages fortified with vitamin D. When needed, supplementation can be considered to achieve RDA intakes of vitamin D.

Review of the Evidence

These conclusions were reached based on the results of the Food Pattern Modeling Report titled "Meeting Vitamin D Recommended Intakes in USDA Food Patterns" (see ***Appendix E-3.3***). It may be difficult for individuals to reach the RDA intake of vitamin D from food, including food as it is currently fortified in the United States. The RDA was established by the Institute of Medicine on the assumption of minimal or no sunshine exposure. This was done even though the majority (up to 80 to 90 percent in some parts of the United States) of vitamin D in the body is derived from conversion by solar radiation of pre-vitamin D in the skin. However, during the winter, in much of the United States, this conversion is minimal and furthermore, recommendations for sunscreen use have limited the degree to which one can safely ensure sunshine exposure as a source of vitamin D.

Vitamin D exposure, and likely status, is assessed generally through serum/plasma 25-hydroxyvitamin D concentrations. However, this test is not recommended for routine screening of the entire population[30-32, 42, 43] due to costs and challenges in obtaining measurements throughout the year and interpreting results in populations, including those who are obese. Because many non-screened individuals will still need to reach the RDA for vitamin D, supplement use may be considered for this purpose.

For additional details on this body of evidence, visit:

- Appendix E-3.3 Meeting Vitamin D Recommended Intakes in USDA Food Patterns

FOOD GROUPS--CURRENT INTAKES AND TRENDS

Introduction

As noted for Questions 5 and 6, to help the U.S. population meet recommended dietary goals and improve their health and well-being, the USDA recommends a food-based, total diet approach for meeting the U.S. Dietary Guidelines.[44, 45]

The USDA Food Patterns have changed over time to be consistent with emerging science that is presented in each issuance of the Guidelines. The current USDA Food Patterns identify amounts of foods to consume from five major food groups (fruits, vegetables, grains, protein foods, and dairy) and their sub-groups (dark green vegetables, orange and red vegetables, starchy vegetables, other vegetables, beans and peas, whole grains, enriched/refined grains, meat/poultry/eggs, nuts, seeds, soy products, seafood) and are based on nutrient-dense foods.[44, 45] In 2010, the DGAC developed a vegetarian adaptation of the Food Patterns to provide guidance for consumers wishing to follow a vegetarian diet. For 2015, the DGAC developed a new Healthy Vegetarian Food Pattern based on food intakes of vegetarians. The 2015 DGAC also provided a Mediterranean-style Food Pattern due to the data supporting the health-related benefits of a Mediterranean-style diet (see Dietary Patterns section, Question 20, and ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***). The food groups chosen for all the Patterns include primarily nutrient-dense foods. The patterns are intended to meet the RDA for nutrients so that nutritional adequacy is met without exceeding recommended energy intake. They also are designed so that they are below the 2010 DGA limits for sodium and saturated fat. Recommended amounts to consume from each food group differ depending on an individual's energy and nutrient needs. Patterns are provided for 12 different calorie levels (Table D1.10) and assignment to one of these calorie levels is based on age, sex, and activity level (Table D1.11). In addition, the Patterns provide for limited amounts of solid fats and added sugars. The complete Food Pattern modeling report (including a listing of the nutrients considered for the Patterns) is found in ***Appendix E3.1***, and details on the methods used to derive the Patterns have been published.[44, 46, 47]

Question 7: What are current consumption patterns of USDA Food Pattern food groups by the U.S. population?

Source of evidence: Data analysis

Conclusion

Positive, healthy eating habits provide an excellent foundation for a lifetime of healthy eating. Many young children start out eating very well, particularly with regard to intakes of fruit and dairy foods. Unfortunately, many of these early life healthy habits seem to disappear as children reach school age and beyond. Across all age and sex groups, the vast majority of the U.S. population does not meet recommended intakes for fruit, vegetables, whole grains, and dairy food groups. Each of these food groups are excellent sources of shortfall nutrients and underconsumed nutrients of public health concern. Across all age and sex groups, the vast majority of the U.S. population exceeds recommended intakes of refined grains, solid fats, and added sugars.

Implications

To realize the numerous health benefits from dietary patterns that are higher in fruit, vegetables, whole grains, lean protein, and non-fat and low-fat dairy (see ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes*** for details on the health benefits for dietary patterns with these characteristics), action is needed across all sectors of food production, distribution, and consumption and at individual behavioral and population levels. Individuals, families, schools, worksites, healthcare and public health settings, restaurants, and other food establishments must work together to ensure that all segments of the population can:

- Increase intake of underconsumed food groups and nutrient-dense foods, while maintaining energy balance, and without increasing saturated fat, sodium, and added sugars

Given the complexity of dietary behavior change, consumers will need access to evidence-based educational resources and intervention programs and services in public health and healthcare settings to facilitate adoption and maintenance of healthy dietary behaviors. (See ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*** for discussion of what works at the level of individual behavior change and ***Part D. Chapter 4: Food Environment and Settings*** for discussion of population change through environmental strategies.)

Within the Dairy and Vegetable groups, the following dietary changes in particular will help increase intake of shortfall nutrients and will decrease intake of overconsumed nutrients by the U.S. population:

- Increasing low-fat/fat-free fluid milk and yogurt and decreasing cheese would result in higher intakes of magnesium, potassium, vitamin A, and vitamin D while simultaneously decreasing the intake of sodium and saturated fat.
- Replacing soft drinks and other sugar-sweetened beverages (including sports drinks) with non-fat fluid milk would substantially reduce added sugars and empty calories and increase the intake of shortfall nutrients, including calcium, vitamin D, and magnesium.
- Consuming all vegetables, including starchy vegetables, with minimal additions of salt and solid fat will help minimize intake of overconsumed nutrients – sodium and saturated fat.

Review of the Evidence

This question was answered using data from the WWEIA, NHANES dietary survey (2007-2010) and the National Cancer Institute's examination of the usual intake distributions and percent of the U.S. population meeting USDA Food Pattern recommendations for their age and sex. [41, 48, 49] It is important to note that the Dietary Guidelines for Americans are established only for those ages 2 years and older. However, the WWEIA, NHANES sample includes persons from birth. The NHANES data are presented in these specific age groups that cannot be further divided.

Fruit. When consumed in the amounts recommended in the USDA Food Patterns, fruit contributes substantial amounts of two nutrients of public health concern: fiber and potassium. (Whole fruit and fruit juice provide about 16 percent of dietary fiber and 17 percent of potassium in the Food Patterns (see ***Appendix E-3.2: Food Group Contributions to Nutrients in USDA Food Patterns and Current Nutrient Intakes***).

The majority of children ages 1 to 3 years and 4 to 8 years meet the recommended intakes for total fruit, which is 1 cup and 1 to 1.5 cups per day, respectively. Among older children (boys and girls ages 9 to 13 years), adolescents, and adults of all ages (both men and women), few people consume the recommended daily amounts, which range from 1.5 to 2 cups for older children and adolescents to 1.5 to 2.5 cups for adults (Figure D1.9). Among the overall U.S. population, approximately 15 percent meet the daily fruit intake recommendation while nearly 80 percent do not meet the recommendation.

More than half of the daily fruit intake for all age and sex groups in the U.S. population (ages 1 year and older) comes from whole fruit (Figure D1.10). Among both boys and girls ages 1 to 3 years, whole fruit comprises slightly more than half of the daily fruit intake and the remainder is consumed though 100% fruit juice. The American Academy of Pediatrics (2001)[50] recommends that young children limit their juice intake to 4 to 6 ounces per day. Six ounces of juice is 0.75 cups; the average juice intakes fall within this recommended limit suggesting that juice is not overconsumed among many young children. Among children ages 4 to 8 and 9 to 13 years, fruit intake includes both 100% juice and whole fruit, but whole fruit comprises the majority of intake. Among middle aged and older adults, most of the fruit intake is from whole fruit, albeit below recommended levels, rather than 100% juice.

Vegetables. Vegetables are excellent sources of many shortfall nutrients and nutrients of public health concern. When vegetables are consumed in the amounts recommended in the USDA Food Patterns, vegetables contribute the following (expressed as averages over all the calorie levels): fiber (38 percent), potassium (36 percent), iron (19 percent), folate (23 percent), and vitamin A as provitamin A carotenoids (34 percent). Note that select vegetables do contribute to calcium intake, including spinach, collard greens, turnip greens, but these vegetables are often consumed in smaller amounts than is needed to be considered important sources of calcium (Table D1.6 and ***Appendix E-3.2: Food Group Contributions to Nutrients in the USDA Food Patterns and Current Nutrient Intakes***).

The U.S. population consumes few vegetables (Figure D1.11). Only 10 percent and 15 percent of boys and girls ages 1 to 3 years, respectively, consume the recommended 1 cup of vegetables per day. For children ages 4 to 8 years, less than 5 percent consume the recommended amount of 1.5 to 2 cups of vegetables per day. Vegetable consumption is lowest among boys ages 9 to 13 years (1 percent consume the recommended 2 to 2.5 cups per day) and girls ages 14 to 18 years (less than 1 percent consume the recommended 2 to 2.5 cups/day). Vegetable intakes increase slightly during the adult years, but intakes are still very low. Among young adult males and females ages 19 to 30 years, less than 10 percent meet the 2 to 3.5 cups/day recommendation. Intakes increase only slightly in subsequent age decades (31 to 50 years). Middle aged adults (51 to 70 years) are somewhat closer to the goal as they have the highest vegetable intakes. Even so, only about 20 percent of men and about 30 percent of women meet the daily recommendation of 2 to 3.5 cups per day. Although these intake levels are still below optimal, the positive gains in vegetable consumption are noteworthy. However, vegetable intakes fall again among older adults (71 years and older), with less than 20 percent of men and women meeting intake recommendations. Overall, nearly 90 percent of the U.S. population does not meet daily vegetable intake recommendations.

The USDA Food Pattern food group for vegetables includes five subgroups: dark green vegetables, red and orange vegetables, beans and peas, starchy vegetables, and other vegetables. The U.S. population does not meet intake recommendations for any of these vegetable subgroups (Figures D1.12 to D1.16). More than 80 percent of the U.S. population does not meet the intake recommendation for dark green vegetables, starchy vegetables, and beans and peas, while more than 90 percent do not meet the recommended intakes for red and orange vegetables. “Other vegetables” (Figure D1.16) is a broad group that includes iceberg lettuce, green beans, cucumbers, celery, onions, summer squash, mushrooms, and avocados. More than 50 percent of males and females ages 51 to 70 years meet or exceed the recommended intake amounts of other vegetables and among all ages, nearly 40 percent meet or exceed the recommended intake. Intake of “other vegetables” is more likely to meet recommendations than the other four subgroups, but consumers should be encouraged to increase intake of all vegetables. To meet total vegetable recommendations, higher intakes of all vegetable subgroups are needed, particularly those subgroups where intake is minimal, such as dark green and orange and red vegetables, which are excellent sources of vitamin C, folate, magnesium, and potassium.

Potatoes (white potatoes) are the most commonly consumed single vegetable, and make up about 80 percent of all starchy vegetable consumption.[51] They account for 25 percent of all vegetable consumption and are a good source of both potassium and fiber. Among children and adolescents ages 2 to 19 years, they account for 28 percent to 35 percent of total vegetable consumption, with a higher percentage of vegetables consumed as potatoes among boys than girls in each age category. Potatoes are consumed in a variety of forms, with about 31 percent being boiled (including mashed and in dishes such as potato salad, soups, and stews), 22 percent as chips, sticks, or puffs, 19 percent as French fries, 17 percent as baked, and 12 percent as home fries or hash browns.

Grains (whole and refined). The 2010 Dietary Guidelines for Americans recommended that half of all grain intake should come from whole grains. The 2015 DGAC brings forward this recommendation and here we give rationale and results to support this decision. The background and summary of previous food pattern modeling with respect to grains is important to present here so as to provide context for the 2015 DGAC recommendations.

Whole grains are those “foods made from the entire grain seed, usually called the kernel, which consists of the bran, germ and endosperm. If the kernel has been cracked, crushed or flaked, it must retain nearly the same relative proportions of bran, germ and endosperm as the original grain in order to be called whole grain.”[52p134] Examples of whole grains are brown rice, popcorn, bulgur, whole wheat, oats, and barley. If whole grains were consumed in the amounts recommended in the Food Patterns, whole grains would provide substantial percentages of several key nutrients, such as about 32 percent of dietary fiber, 42 percent of iron, 35 percent of folate, 29 percent of magnesium, and 16 percent of vitamin A (see ***E-3.2: Food Group Contributions to Nutrients in USDA Food Patterns and Current Nutrient Intakes***).

Across all ages and both sexes, the U.S. population does not meet the goal for whole grain intake, as nearly 100 percent of the population consumes amounts that are below the recommended intake levels (Figure D1.17), which range from 1.5 ounce equivalents (oz eq) for young children up to 3 to 3.5 oz eqs for older children and adolescent and adult females. Adolescent and adult males are advised to consume 3 to 4 oz eqs per day. The inadequate intake of whole grains leads to underconsumption of several shortfall nutrients and nutrients of public health concern. Refined grains, such as white flour and products made with white flour, white rice, and de-germed cornmeal, are part of the intake recommendation because they are commonly enriched with iron and several B vitamins, including thiamin, niacin, and riboflavin (e.g., enriched flour, 21 CFR 137.165).[53p.452] Since 1998, enriched grains also have been fortified with folic acid and are thus an important source of folic acid for women of childbearing potential.[53, 54] The effect of the folic acid fortification on the health status of the U.S. population was extensively reviewed by the 2010 DGAC and so was not re-reviewed by the 2015 DGAC. The 2010 DGAC concluded that strong and consistent evidence demonstrates a large reduction in the incidence of neural tube defects (NTDs) in the United States and Canada following mandatory folic acid fortification. They also found only limited evidence to suggest a decline in stroke mortality in the United States and Canada and an increase in colorectal cancer in those countries following mandatory folic acid fortification. Due to the very limited evidence, cause and effect cannot be attributed for folic acid fortification and either stroke or colorectal cancer incidence. The 2015 DGAC brings forward those results with no notable changes in the interpretation of the data presented in 2010. Despite the B vitamins and iron that can be obtained from enriched and fortified refined grains, products made with refined grains also may be a source of excess calories and added sugars. (See Question 11c, food categories, below, and added sugars discussion in ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance.***) Figure D1.18, documents that the U.S. population consumes far too many refined grains. In the overall population for all ages and for both males and females, about 19 percent meet the recommendation for refined grains, while more than 70 percent exceed the recommendation. Intake of refined grains is particularly high among boys and girls ages 4 to 8 years and girls ages 9 to 13 years.

Due to the overconsumption of refined grains and the underconsumption of whole grains relative to the 2010 recommendation that "half of all grain intake should come from whole grains," the DGAC decided that it was important to examine the impact on nutrient intake if: (1) refined/enriched grains intake were reduced to no more than 25 percent or 15 percent of the total grains intake; and (2) overall grain intake were reduced. The Committee relied on food pattern modeling analyses conducted by the 2005 and 2010 DGACs to answer these questions, and brings forward their recommendations, as reiterated below.

The key finding from the 2010 DGAC modeling report was: "As shown by food pattern modeling, consumption of all grains as whole grains, without including any fortified whole grain products, would lower dietary folate and iron intake levels to less than adequate amounts for individuals in population groups who may be at high risk for inadequate intakes of these nutrients. Individuals are encouraged to consume most of their grains as fiber-rich whole grains, and when doing so, should select some of these fiber-rich whole grains as products that have been fortified with folic acid and possibly other nutrients". [55p146]

In its analysis, the 2005 DGAC reported that non-whole grains contributed important amounts of certain nutrients to the dietary patterns, including folate, iron, calcium, fiber, thiamin, riboflavin and niacin.[56append G-2] The 2005 DGAC concluded that including only 3 oz eqs of whole grains, with no non-whole grains, in the food patterns would lower intake of many of these key nutrients and perhaps place certain individuals at risk of nutrient inadequacy. However, the 2010 DGAC found that consuming all grains as whole grains would provide for nutrient adequacy in the patterns if fortified ready to eat (RTE) whole grain breakfast cereals were substituted for RTE refined grain breakfast.[55app E.7] The 2015 DGAC concluded that consumption of only whole grains with no replacement or substitution would result in nutrient shortfalls.

Dairy. Dairy foods in the USDA Food Patterns include fluid milk, cheese, yogurt, ice cream, milk-based replacement meals and milk products, including fortified soymilk, but do not include almond or other plant-based "milk-type" products. Dairy foods are excellent sources of nutrients of public health concern, including vitamin D, calcium, and potassium. Consumption of dairy foods provides numerous health benefits including lower risk of diabetes, metabolic syndrome, cardiovascular disease and obesity.[57-62] When consumed in the amounts recommended by the Food Patterns, on average across the calorie levels, dairy foods contribute about 67 percent of calcium, 64 percent of vitamin D, and 17 percent of magnesium (see ***Appendix E-3.2: Food Group Contributions to Nutrients in the USDA Food Patterns and Current Nutrient Intakes***). The Patterns recommend consumption of low-fat and fat-free foods in the Dairy group to ensure intake of these key nutrients while minimizing intake of saturated fat, which is a nutrient of concern for overconsumption.[44]

More than 60 percent of young boys and girls ages 1 to 3 years meet or exceed the recommended intake of 2 cup eqs per day, with most of this intake coming in the form of fluid milk (see Figure D1.19 and Appendix E-3.4: USDA Food Patterns—Adequacy for Young Children). Intake falls in older children to about 30 percent of boys and girls meeting or exceeding the recommended 2.5 cup eqs per day for those ages 4 to 8 years and 3 cup eqs per day for children ages 9 to 13 years. About 30 percent of adolescent boys meet or exceed the recommended 3 cup eqs per day, but less than 10 percent of adolescent females meet or exceed this recommendation. An age-related decline in dairy intake appears to begin in adolescence and intakes persist at very low levels among adult females across the age distribution. Less than 5 percent of adult females consume the recommended 3 cup equivalents per day. Overall, more than 80 percent of the entire U.S. population does not meet the daily dairy intake recommendation.

To determine the extent to which individuals could meet recommendations for calcium and other shortfall nutrients intake, given various levels of dairy foods in the Food Patterns, the 2015 DGAC conducted a food pattern modeling analysis (see ***Appendix E-3: Dairy Group and Alternatives***). The DGAC considered nutrient adequacy of the Food Patterns under the following scenarios: 1) no dairy was consumed; 2) calcium was obtained from non-dairy sources (including fortified foods); and 3) the proportions of yogurt and cheese in the patterns were modified. The DGAC further evaluated the relationship between changes in the types of beverages consumed (milk, fruit juices, fruit drinks and sports beverages) and diet quality.

If no dairy is consumed, the modeling analysis shows that levels of calcium, magnesium, iron, vitamin A and riboflavin, drop below 100 percent of goals, and intake levels of potassium, vitamin D and choline also drop substantially. When no dairy is consumed, calcium intake levels drop by 68 to 88 percent in all age and sex groups, while vitamin D intake is lowered by 20 to 30 percent (see ***Appendix E-3.6: Dairy Group and Alternatives***, Table 2). Most of the milk alternatives are fortified with calcium, so similar amounts of calcium can be obtained from fortified rice, soy and almond milks, and fortified juices, but absorption of calcium is less efficient from plant beverages.[63] Magnesium intake also is comparable from plant-based milk alternatives. However, vitamin D and potassium amounts vary across these milk alternatives (see ***Appendix E-3.6: Dairy Group and Alternatives***, Table 3). Calorie levels also are higher for most of the plant-based alternative milk products for a given calcium intake level. In other words, to obtain a comparable amount of calcium as one cup eq for non-fat fluid milk, the portion size required to meet the calcium intake need results in higher energy intake (see ***Appendix E-3.6: Dairy Group and Alternatives***, Table 4).

Currently, the U.S. population consumes the recommended 3 cup equivalents/day as 53 percent fluid milk, 45 percent cheese, and 2 percent as yogurt. Through the food pattern modeling, the DGAC examined the effect on nutrient intake if fluid milk were to be increased and cheese decreased. Increasing the proportion of fat-free milk, while decreasing the proportion of cheese, would increase the intake of magnesium, potassium, vitamin A, vitamin D and would decrease intake of sodium and saturated fat (see ***Appendix E-3.6: Dairy Group and Alternatives***, Table 5). A potential approach to increasing intake of shortfall nutrients and nutrients of public health concern while simultaneously decreasing intake of overconsumed nutrients of public health concern would be to increase intake of fat-free or low-fat fluid milk in lieu of cheese.

If milk is completely eliminated from the diet and replaced by soft drinks, fruit drinks, sports beverages, and other sugar-sweetened beverages, diet quality deteriorates significantly, making it very hard for individuals to meet nutrient recommendations (see ***Appendix E-3.6: Dairy Group and***

Alternatives, Table 6). Indeed, among U.S. adolescents' milk consumption is very low as are intakes of the "shortfall" nutrients.

Protein Foods. Protein Foods comprise a broad group of foods including meat, poultry, fish/seafood, eggs, soy,[∞] nuts, and seeds. Dairy also contains protein, but since it has its own food group, its nutrient contributions are counted in its own group. The inclusion of both animal and non-animal protein foods allows vegetarian options to be accommodated. In addition to providing essential amino acids, some protein foods are important sources of iron, and iron is a shortfall nutrient and nutrient of public health concern among adolescent and adult females. Meat foods in the protein group provide heme iron, which is more bioavailable than non-heme plant-derived iron. Heme iron is especially important for young children and women who are pregnant.

Nearly 80 percent of boys and 75 percent of girls ages 1 to 3 years meet or exceed the protein foods recommendation of 2 ounce equivalents per day (Figure D1.20). Similarly, more than 60 percent of boys and girls ages 4 to 8 years meet or exceed the recommended intake of 3 to 4 oz eqs/day. Intake declines somewhat for boys and girls ages 9 to 13 years, as approximately 40 percent and 45 percent meet or exceed the recommended 3 ounce equivalents/day. Although nearly 60 percent of adolescent males ages 14 to 18 years meet the 5.5 to 6.5 oz eq/day recommendation, less than 25 percent of females ages 14 to 18 meet their 5-5.5 oz eq/day recommendation. Intakes begin to increase again for adult males across the age distribution, and about 62 percent of males ages 31 to 50 and 78 percent of males 51 to 70 years meet the 5.5-6.5 oz eq/day intake recommendation. For adult females ages 19 to 30 years, slightly more than 40 percent meet the 5 to 5.5 oz eq/day recommendation and approximately 50 percent of those ages 31 to 50 and about 50 percent of those 51 to 70 years meet the recommendation. Protein foods intake declines in both men and women older than age 71 years; about 30 percent of women and about 50 percent of men meet the recommendation. Across all age groups and in both males and females, nearly 60 percent of the U.S. population meets the protein foods intake recommendation. Although some groups in the U.S. population do not consume recommended amounts from the protein foods group, intakes of protein (as grams/day) are adequate across the population and protein is not a shortfall nutrient. Notably, protein intake also comes from dairy and grains in addition to the foods included in the protein foods group.

Most of the protein foods intake across all age groups and for both males and females comes from meat, poultry, and eggs (Figure D1.21). Nearly 80 percent of the U.S. population meets the intake recommendation for this protein foods subgroup (although less so for adolescent girls and older women).

[∞] Soy foods in the Protein Foods group include foods and ingredients such as tofu, soy noodles, soy flours, and soy protein isolates. Fortified soymilk is part of the Dairy group. Edamame and whole soybeans are part of the vegetable legume subgroup.

In 2010, the DGAC recommended that seafood intake be increased to eight ounces per week for adults. In reviewing the WWEIA/NHANES data, the DGAC 2015 found that the U.S. population has low seafood intake. Across all age groups and for both males and females, only 10 percent of the population meets the 2010 intake recommendations (Figure D1.22). Intake is highest in adult men and women, but remains very low. In the highest intake group, males ages 51 to 70 years, 21 percent of the population meets the intake recommendation.

In addition to reviewing WWEIA/NHANES data, the 2015 DGAC considered the potential influence on diet quality of substituting seafood for terrestrial animal foods (e.g., beef, poultry, pork, game meats). This question was addressed by the 2010 DGAC through a modeling analysis, and the 2015 DGAC decided to bring forward those modeling results. These results indicate seafood could be increased to 8 ounces/week (for adults) with no negative impact on nutrient adequacy.[55app E3.10] This 8 oz amount contributes energy, protein, selenium, vitamin D, and vitamin B-12. With respect to fatty acids, fish is rich in the long-chain eicosapentanoic acid (EPA) and docosahexonoic acid (DHA) and has a higher proportion of total fatty acids coming from polyunsaturated and monounsaturated fatty acids relative to saturated fatty acids. The 2015 DGAC also has examined the sustainability of fish production and consumption, and these results are discussed in ***Part D. Chapter 5: Food Sustainability and Safety***.

Nuts, seeds, and soy. Nuts, seeds, and soy provide protein, selenium, polyunsaturated fatty acids, fiber, magnesium, and zinc. Nuts, seeds, and soy are less commonly consumed protein foods (Figure D1.23). Even so, overall approximately 40 percent of the U.S. population meets or exceeds the food pattern recommended intake of these protein foods.

Empty calories. Solid fats that occur naturally in foods such as meat, dairy, and some tropical foods (e.g., coconut), and sugars that are added to foods either by the consumer or by food manufacturers are referred to as “empty calories” because both provide calories, but few or no nutrients. For the purposes of the USDA Food Pattern Food Groups, the term solid fats and added sugars is an analytic grouping, but going forward for 2015, the DGAC has elected to use the term “empty calories.”

Calories from solid fats and added sugars are included for the USDA Food Patterns because they are a component of the diet that should be limited because they are not nutrient-dense and the solid fats contribute to saturated fat intake, which is overconsumed in the U.S. population (see Nutrient Intake/Nutrients of Concern section, Questions 1 and 2). Solid fats and added sugars are not food groups on their own, as are protein foods, dairy, grains, fruits, and vegetables, but they are included in the Food Patterns because they are an integral component of many foods consumed by the U.S. population either because they occur naturally (in the case of some solid fats) or they are added to foods, such as added sugars or fat added during processing, cooking, or other aspects of food preparation. Additional details about added sugars and saturated fat are provided in ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance***.

Because added sugars and solid fats are not nutrient dense and solid fats contribute to saturated fat intake, the USDA Food Patterns recommend that intake be limited. The guidance on the approximate amounts of solid fats and added sugars that can be part of a healthful diet is as follows: children ages 2 to 8 years: 120 calories/day; children 9 to 13 years: 120 to 250 calories/day; girls ages 14 to 18 years: 120 to 250 calories/day; boys ages 14 to 18: 160 to 330 calories/day; adult women: 120 to 250 calories/day; and adult men: 160 to 330 calories/day. Intake limits varies by age and sex and are based on residual calories after all food group intakes are met. The intake limits include solid fats and added sugars from all sources in the diet: from sugar in sugar-sweetened beverages, including coffee and tea, and breakfast cereals, to solid fats in burgers, sandwiches, and pizza, to the combination of solid fats and added sugars in snacks and desserts such as cookies, cakes, ice cream, and donuts. Question 11 of the Food Categories section of this Chapter provides information on food sources of solid fats and added sugars**.**

The intake of solid fats and added sugars is very high across all age groups and for both males and females in the United States, with nearly 90 percent exceeding the recommended daily limits (Figure D.1.24). Particularly noteworthy is that nearly 100 percent of boys and girls ages 1 to 3 and 4 to 8 years exceed the recommended limit for solid fats and added sugars (see ***Part B. Chapter 6: Cross Cutting Topics of Public Health Importance***).

For additional details on this body of evidence, visit:

- Usual Dietary Intakes: Food Intakes, U.S. Population, 2007-10: Applied Research Program. National Cancer Institute; [updated May 22, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/.
- Appendix E-3.2 USDA Food Pattern Modeling Report: Food Group Contributions
- Appendix E3.6 USDA Food Pattern Modeling Report: Dairy Group and Alternatives
- Food Patterns Equivalent Intakes from Food: Consumed per Individual, 2009-10. U.S. Department of Agriculture, Agricultural Research Service, Food Surveys Research Group. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=23868.
- Seafood Food Pattern Modeling Report for the 2010 Dietary Guidelines Advisory Committee. USDA and HHS, 2010, Appendix E 3.10 USDA and HHS, 2010, Appendix E 3.10. Available from: http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/AppendixE-3-10-Seafood.pdf.
- Replacing all Non-Whole Grains with Whole Grains Food Pattern Modeling Report for the 2010 Dietary Guidelines Advisory Committee. USDA and HHS, 2010, Appendix E3.7. Available from: http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/AppendixE-3-7-Grains.pdf.

- Alternatives for Enriched Grains in Food Intake Patterns Analysis for the 2005 Dietary Guidelines Advisory Committee. U.S. HHS and USDA, 2005, appendix G-2. Available from: http://www.health.gov/dietaryguidelines/dga2005/report/HTML/G2_Analyses.htm#alternativegrain.

Question 8: What are the trends in USDA Food Pattern food group consumption by the U.S. population?

Source of Evidence: Data analysis

Conclusion

The U.S. population has made few dietary changes over time:

- Fruit intake has remained low but stable.
- Vegetable intake has declined, particularly among children of all ages, adolescents, and young adult males.
- Whole grain intake has slightly increased between 2001-2004 and 2007-2010, particularly among middle aged and older adults.
- Dairy intake has been relatively constant over time, but has decreased for girls ages 4 to 8 years and young adult males, and has increased for adults ages 51 to 70 years.
- Added sugars intake has decreased for both males and females across all age groups between 2001-2004 and 2007-2010, but intakes still exceed the limit in the USDA food patterns.

Implications

Individuals and families must make conscious and focused decisions about choosing nutrient-dense foods. In addition, to continue progress toward consumption of a healthy diet among all age and sex groups, action is needed along the entire food processing, delivery, and service supply chain in order to provide the U.S. population with affordable and accessible foods that are nutrient dense and low in added sugars and sodium.

Poor nutritional intake is linked to numerous diet-related chronic diseases (see ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***) and the prevalence of these conditions is too high in the United States (see Health Conditions section, Questions 15 to 17, below). The health of the nation hinges in part on improving dietary intake at individual and population levels, and changes in line with those suggested here could have a measurable positive impact on the health of the population.

Given the complexity of dietary behavior change, consumers will need access to evidence-based educational resources and intervention programs and services in public health and healthcare settings to facilitate adoption and maintenance of healthy dietary behaviors. (See ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*** for discussion of what works at the level of individual behavior change.) In addition, these efforts should be complemented with research-driven environmental strategies that make access to affordable healthy foods possible in retail, community, worksite, and educational settings. (See ***Part D. Chapter 4: Food Environment and Settings*** for discussion of effective environmental approaches to promote dietary change across the lifespan.)

Review of the Evidence

This question was answered using data from WWEIA, NHANES dietary survey data and the National Cancer Institute's examination of usual intake distributions for 2001-2004[64] and 2007-2010.[41]

Fruit. Fruit intake remained relatively stable across the 2001-2004 and 2007-2010 time periods (Figure D1.25). The only group with significant changes over time was males ages 31 to 50 years, for whom mean fruit intake decreased.

Vegetables. Vegetable intake declined from 2001-2004 to 2007-2010 (Figure D1.26). Across the overall population, the mean daily vegetable intake significantly declined. Significant declines in mean intake occurred among males ages 1 to 3, 4 to 8, 9 to 13, 14 to 18, and 19 to 30 years. For females, significant decreases in mean vegetable intake occurred for those ages 1 to 3, 4 to 8, and 9 to 13 years.

Grains (whole and refined). Whole grain intake significantly increased among the overall population between 2001-2004 and 2007-2010 (Figure D1.27). Among males, significant increases in mean intake occurred for those ages 1 to 3, 4 to 8, 14 to 18, 31 to 50, and 51 to 70 years. Among females, significant increases in mean whole grain intake occurred for those ages 9 to 13, 19 to 30, 31 to 50, 51 to 70, and 71 years and older (Figure D1.27). Similarly, refined grain intake has declined in all age and sex groups between 2001-2004 and 2007-2010 (Figure D1.28).

Dairy. Dairy intake remained stable over the entire population between 2001-2004 and 2007-2010 (Figure D1.29). Significant declines in mean daily intake occurred between the two time periods for males ages 19 to 30 years and females ages 4 to 8 years. Significant increases in mean daily dairy intake occurred for both males and females ages 51 to 70 years.

Protein Foods. Protein food intake remained relatively stable for the U.S. population between 2001-2004 and 2007-2010 (Figure D1.30). Females ages 31 to 50 and 51 to 70 years had significantly higher mean intake in 2007-2010 compared to 2001-2004. These were the only groups with any significant change over time.

Added Sugars. Some improvements have been made in added sugars intake, with noticeable declines in mean intakes for all age groups and among both males and females when comparing 2007-2010 data with 2001-2004 data (Figure D1.31). As seen in Figure D1.31, intakes of added sugars are still very high, however, and are well above recommended limits, but the improvements provide some optimism for improved diets.

For additional details on this body of evidence, visit:

- Usual Dietary Intakes: Food Intakes, US Population, 2007-10: Applied Research Program. National Cancer Institute; [updated May 22, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/.

- Usual Dietary Intakes: Food Intakes, US Population, 2001-04: Applied Research Program. National Cancer Institute; [updated April 2, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2001-04/.

FOOD CATEGORIES—CURRENT INTAKES AND SOURCES OF ENERGY, NUTRIENT, AND FOOD GROUP INTAKES

The food sources of nutrients and the patterns in which they are consumed are informative in identifying strategies to modify dietary intake and eating behaviors and help Americans to choose and consume higher quality diets. We examined four questions related to the foods that are top contributors to intakes of energy, food groups, and selected nutrients in the U.S. diet. This section describes those food sources and the implications for meeting recommended or optimal intakes of various food groups and nutrients.

Question 9: What are current consumption patterns by food categories (i.e., foods as consumed) in the U.S. population?

Source of evidence: Data analysis

Conclusion

The mixed dishes food category, which includes foods commonly used as entrees, such as sandwiches, burgers, pizza, pasta or rice mixed dishes, stir-fries, soups, and meat or poultry mixed dishes, is the major contributor to three USDA Food Pattern food groups—grains, vegetables, and protein foods. Fruit and fluid milk intake are seldom consumed as part of mixed dishes. The mixed dishes food category contributes heavily to intake of energy, saturated fat, and sodium; however, mixed dishes do provide vegetables, fiber, grains, and dairy.

Implications

An important strategy for meeting recommended intake levels of calories, saturated fat, and sodium is to change the composition of mixed dishes that are high in calories, saturated fat, and sodium to better meet these nutrition goals. Food manufacturers and the food service sector (e.g., restaurants, schools) should reformulate mixed dishes to improve their nutritional profiles. Americans should be encouraged to modify recipes to lower the sodium and saturated fat content when cooking, to use appropriate portion sizes, and choose reformulated mixed dish options when available.

Review of the Evidence

These conclusions were reached by examining data from the WWEIA Food Categories for the NHANES 2009-2010 dietary survey.[65] The WWEIA Food Categories provide an application that allows analysts to examine foods and beverages as consumed in the U.S. diet. Each food or beverage item (as consumed) that is included in WWEIA is placed in one of 150 mutually exclusive food categories. The focus of this categorization system is on grouping similar foods and beverages together based on usage and nutrient content.

An adaptation of the food categories was used by the 2015 DGAC for this analysis related to the "sandwiches and burgers" and "salads" categories. We placed all food items reported to be eaten as a sandwich, burger, taco, or salad item into the "sandwiches and burgers" or the "salads" categories regardless of whether the components were reported as separated ingredients or as a single combined item. For example, a food reported as a "cheeseburger" (a single item) would always be classified in the category of "burgers, sandwiches, and tacos," but a food reported as the individual food items of a hamburger bun, a hamburger patty, and cheese, eaten as a combination, would have been classified in the categories of "rolls and buns," "ground meat," and "cheese." The adaptation recoded these individually reported foods that were eaten in combination to "burgers, sandwiches, and tacos." By doing this, the categories used for this analysis more fully represented foods as consumed rather than as ingredients.

The 150 categories from WWEIA were condensed into 9 major and 32 sub-categories for analysis of the percent of total intake for energy, nutrients, and food groups from each major and sub-category (see ***Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food Categories***). Analysis was conducted for the population ages 2 and older as a whole; analysis of the percent of energy intake also was conducted for males and females ages 2 to 5, 6 to 11, 12 to 19, 20 to 40, 41 to 50, 51 to 70, and 71 years and older; for race/ethnic groups including Non-Hispanic Whites, Non-Hispanic Blacks, and Hispanics ages 2 years and older; and for those with incomes less than or equal to 185 percent, or greater than 185 percent of the Poverty Index Ratio by three age groups: 2 to 11, 12 to 19, and 20 years and older.

WWEIA data show that Americans consume a substantial amount of foods in the form of mixed dishes (Figure D1.32). More specifically, 31 percent of vegetables, 45 percent of grains, 30 percent of dairy, and 45 percent of protein foods come from mixed dishes. Mixed dishes (which include foods such as sandwiches, burgers, pizza, pasta or rice mixed dishes, stir-fries, soups, and meat or poultry mixed dishes) make up 28 percent of total energy intake. Of note, only small amounts of fruits (1 percent) and fluid milk (3 percent) are consumed in mixed dishes—most are consumed as single food items, such as an apple or glass of milk (see ***Appendix E-2.8: Percent of total food group intake, 2009-2010, for U.S. population ages 2 years and older, from WWEIA Food Categories***).

When mixed dishes contribute to dairy foods, the majority of intake is in the form of cheese. Data show that about two-thirds of all cheese intake is from mixed dishes such as pizza, burgers, sandwiches, and casseroles. Given that cheese is generally higher in saturated fat and sodium and lower in potassium and vitamin D than is fluid milk (see Question 7b, above, and ***Appendix E-3.6: Dairy Group and Alternatives***), modifying the types of cheese products used in these mixed dishes to lower fat and sodium versions would improve their nutritional profile.

When mixed dishes contribute to the grains group, a larger percentage of refined (48 percent) than whole (19 percent) grains are consumed as part of these dishes. Substitution of whole for refined grains in mixed dishes such as burgers, sandwiches, pizza, and casseroles containing pasta or rice could improve the nutritional profile of grains that are consumed this way.

Although mixed dishes account for a substantial amount of intake of some overconsumed nutrients (43 percent of sodium, 36 percent of saturated fat), they also account for 28 percent of fiber, 29 percent of calcium, 24 percent of potassium, and 16 percent of vitamin D, all of which are underconsumed nutrients. Other food categories that contribute substantially to overall energy, sodium, saturated fat, and added sugars intake are discussed in the following two questions—Question 10: “What are the top foods contributing to energy intake in the U.S. population?” and Question 11: “What are the top foods contributing to sodium, saturated fat, and added sugars intake in the U.S. population?”

For additional details on this body of evidence, visit:

- What We Eat in America. Food Categories for the NHANES 2009-2010 dietary survey. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=23429.
- Appendix E-2.7: Major categories and subcategories used in DGAC Analyses of WWEIA Food Categories
- Appendix E-2.8: Percent of total food group intake, 2009-10 for U.S. population ages 2 years and older

Question 10: What are the top foods contributing to energy intake in the U.S. population?

Source of evidence: Data Analysis

Conclusion

Seventy-five percent of total energy intake in the U.S. population comes from 16 of the 32 food sub-categories, with mixed dishes, snacks and sweets, and beverages together contributing to more than half (56 percent) of energy intake in the U.S. population.

Implications

The foods with the highest contribution to energy intake are burgers, sandwiches, and tacos; desserts and sweet snacks; and sugar-sweetened beverages. Given the link to energy intake, reduced consumption of these foods and beverages or modifying the ways these foods are prepared, as well as consumption of smaller portion sizes, may help prevent excess weight gain or may help with weight reduction.

Public health strategies (e.g., programs, regulations, and policies) and product reformulation are needed to help individuals achieve recommendations.

Review of the Evidence

These conclusions were reached by examining data from the WWEIA Food Categories for the NHANES 2009-2010 dietary survey,[65] as described in relation to question 9 (current consumption patterns by food categories in the U.S. population).

The top foods contributing to energy intake in the U.S. population are concentrated in several food categories, as shown in Figure D1.33. Three food categories account for more than half (56 percent) of all energy consumed: 1) Mixed dishes (which include foods such as sandwiches, burgers, pizza, pasta or rice mixed dishes, stir-fries, soups, and meat or poultry mixed dishes); 2) snacks and sweets, which includes foods such as chips, cakes, pies, cookies, doughnuts, ice cream, and candy.), and 3) beverages other than milk and 100% fruit juice (such as soft drinks, fruit drinks, coffee and tea, and alcoholic beverages)

Examining energy intake from the more specific 32 food subcategories shows that almost half of total energy intake comes from just 7 of these sub-categories (Table D1.12): Burgers and sandwiches (13.8 percent); desserts and sweet snacks (8.5 percent); sugar-sweetened beverages (6.5 percent); rice, pasta, and grain-based mixed dishes (5.5 percent); chips, crackers, and savory snacks (4.6 percent); pizza (4.3 percent); and meat, poultry, and seafood mixed dishes (3.9 percent). Further examination of the 32 subcategories shows that 75 percent of all energy intake comes from the 7 subcategories previously described, plus vegetables (including starchy vegetables), alcoholic beverages, yeast breads and tortillas, whole and 2 percent milk and yogurt, breakfast cereals and bars, poultry, and candy and sugars.

As noted in Question 9, (current consumption patterns by food categories in the U.S. population), some of the food sub-categories that provide substantial amounts of energy also provide underconsumed food groups and nutrients. On the other hand, several of these subcategories, notably desserts and sweet snacks and sugar-sweetened beverages, tend to contribute to energy intake with little contribution to underconsumed food groups (see ***Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories***) and nutrients (see ***Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories***), but major contributions to one or more overconsumed food components (see Question 11: What are the top foods contributing to sodium, saturated fat, and added sugars intake in the U.S. population?)

Analysis of the food sources of energy by age and sex groups showed the expected higher percent of energy from dairy among children, especially young children, but no other major differences. Analysis by racial/ethnic groups and by income groups did not show major differences (see ***Appendix 2.10: Percent of total energy intake, 2009-2010, for age/sex groups of the U.S. population, from WWEIA Food Categories***, ***Appendix E-2.11: Percent of total energy intake, 2009-2010, for racial/ethnic groups of the U.S. population, from WWEIA Food Categories***, and ***Appendix E-2.12: Percent of total energy intake, 2009-2010, for age/income groups of the U.S. population, from WWEIA Food Categories***).

For additional details on this body of evidence, visit:

- What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=23429.
- Appendix E-2.7: Major categories and subcategories used in DGAC Analyses of WWEIA Food Categories
- Appendix E-2.8: Percent of total food group intake, 2009-2010, for U.S. population ages 2 years and older, from WWEIA Food Categories
- Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories
- Appendix E-2.10: Percent of total energy intake, 2009-2010, for age/sex groups of the U.S. population, from WWEIA Food Categories
- Appendix E-2.11: Percent of total energy intake, 2009-2010, for racial/ethnic groups of the U.S. population, from WWEIA Food Categories
- Appendix E-2.12: Percent of total energy intake, 2009-2010, for age/income groups of the U.S. population, from WWEIA Food Categories

Question 11: What are the top foods contributing to sodium, saturated fat, and added sugars intake in the U.S. population?

Source of Evidence: Data analysis

Conclusion

Mixed dishes are the largest contributor to intake of sodium (44 percent) and saturated fat (38 percent). Sodium and saturated fat have both been identified as nutrients of concern for overconsumption. Within mixed dishes, the sub-category of burgers and sandwiches is the largest contributor for both nutrients.

Sodium is ubiquitous in the food supply and many food categories contribute to intake. Beverages supply 47 percent of added sugars intake.

Snacks and sweets also are a major contributor to added sugars (31 percent) and saturated fat intake (18 percent).

Implications

To decrease dietary intake from added sugars, the U.S. population should reduce consumption of sugar-sweetened beverages and of desserts and sweet snacks.

The U.S. population can use a variety of strategies to reduce consumption of sodium, saturated fat, and added sugars, including smaller portion sizes, reduced frequency of consumption, and recipe modification.

Given the ubiquity of sodium in the food supply, concerted efforts to reduce sodium in commercially prepared and processed foods, as well as encouragement of home cooking using recipes with small amounts of sodium are needed to decrease intake toward recommended levels.

Review of the Evidence

These conclusions were reached by examining data from the WWEIA Food Categories for the NHANES 2009-2010 dietary survey,[65] as described in relation to Question 9 (current consumption patterns by food categories in the U.S. population).

The category of mixed dishes contributes substantially more saturated fat (36 percent) and sodium (43 percent) to diets of the U.S. population than does any other category. Within this category, the largest share of both saturated fat (19 percent) and sodium (21 percent) comes from the subcategory of burgers, sandwiches, and tacos. The other subcategories that also contribute notable amounts of saturated fat and sodium are pizza (approximately 6 percent for both); rice, pasta, and other grain-based mixed dishes (5 percent and 7 percent); and meat, poultry, and seafood mixed dishes (5 percent and 7 percent). Soups contribute a notable amount of sodium (4 percent) but less saturated fat (1 percent). (Figures D1.34 and D1.35).

Other food categories contributing substantial amounts of saturated fat include snacks and sweets (18 percent), protein foods (15 percent), and dairy (13 percent). Within snacks and sweets, the subcategory providing the largest share is desserts and sweet snacks (12 percent). Within protein foods, saturated fat comes from meats, in general (3 percent), deli and cured meats and poultry (3 percent), poultry (3 percent), and eggs (3 percent), with seafood and nuts, seeds, and soy each contributing less than 3 percent. Within the dairy category, higher fat (whole and 2 percent) milk and yogurt (7 percent) and cheese (4 percent) contribute the most saturated fat.

Sodium is more ubiquitous in the food supply than are other nutrients, and the food categories contributing the highest amounts of sodium include protein foods (14 percent), grains (11 percent), vegetables (11 percent), and snacks and sweets (8 percent). Sodium is distributed throughout many food categories and subcategories with the exception of fruits and fruit juice, which are notably low in sodium (0.1 percent).

The distribution of added sugars in foods as consumed differs from saturated fat and sodium (Figure D1.36) The vast majority of added sugars intake comes from the major categories of beverages (not including milk and 100% fruit juice) (47 percent) and snacks and sweets (31 percent). Grains, including breakfast cereals and bars, contribute 8 percent, mixed dishes contribute 6 percent, and dairy, including sweetened flavored milks and yogurts contribute only 4 percent of total added sugars intake (see ***Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories***).

Four additional questions were examined using the WWEIA Food Categories data. They are:

11a. What is the current contribution of fruit products with added sugars to intake of added sugars?

11b. What is the current contribution of vegetable products with added sodium to intake of sodium?

11c. What is the current contribution of refined grains to intake of added sugars, saturated fat, some forms of polyunsaturated fat, and sodium?

11d. What are the sources of caffeine from foods and beverages on the basis of age and sex categories?

With regard to Question 11a, the DGAC found that:

- Less than 1 percent of total added sugars come from fruits and 100% fruit juice foods (including fresh, canned, frozen, dried fruit and fruit salads) (see ***Appendix E-2.8: Percent of***

total food group intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories).

With regard to Question 11b, the DGAC found that:

- 11 percent of total sodium comes from all vegetables (with starchy vegetables), including beans and peas, vegetable mixtures, lettuce salads, pasta sauces, and vegetable juice (see ***Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories***).
- When vegetables are categorized by starchy or non-starchy, we found that:
  - 7 percent of total sodium comes from all vegetables, excluding starchy vegetables, and
  - 4 percent comes from starchy vegetables, including French fries and other fried potatoes, mashed potatoes, all other potatoes, corn, and other starchy vegetables.

With regard to Question 11c:

- The DGAC could not directly determine the contribution of refined grains to the nutrients of interest with the currently available data. However, the food categories that make up more than 90 percent of all refined grain intake (i.e., burgers, sandwiches, and tacos; breads and tortillas; rice and pasta mixed dishes; desserts and sweet snacks; pizza; chips, crackers, and savory snacks; quick breads; rice and pasta; and meat, poultry, and seafood mixed dishes) account for:
  - 28 percent of all added sugars intake
  - 47 percent of all saturated fat intake
  - 50 percent of all sodium intake

  (see ***Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories*** and ***Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010 for the U.S. population ages 2 years and older, from WWEIA Food Categories***)

With regard to Question 11d, the DGAC found that (Figure D1.37):

- Among children and adolescents, sugar-sweetened and diet beverages and coffee and tea contribute to overall caffeine intake at approximately equal levels.
- Among adults, the primary sources of caffeine from all foods and beverages are coffee and tea.

For additional details on this body of evidence, visit:

- What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=23429.
- Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food Categories
- Appendix E-2.8: Percent of total food group intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories
- Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories

Question 12: What is the contribution of beverage types to energy intake by the U.S. population?

Source of evidence*:* Data analysis

Conclusion

Beverages contribute 19 percent of total energy intake. Of this 19 percent of energy, major sources are sugar-sweetened beverages (35 percent), milk and milk drinks (26 percent), and 100% fruit juices (10 percent).

Implications

The beverages that contribute the most to energy intake, particularly sugar-sweetened beverages, are those that are not nutrient dense and could be targeted for reduction. Others, like milk, fortified low- and non-fat milk, and milk beverage are good sources of key nutrients. Modifying the types of beverages consumed can reduce calories (e.g., switching from sugar-sweetened beverages to water) or improve nutrient intakes (e.g., switching from sugar-sweetened beverages to low-fat or fat-free milk). This may be an important strategy for individuals who need to reduce their energy intake and/or control their weight. Public health strategies (e.g., programs, regulations, and policies) are needed to reduce consumption of sugar-sweetened beverages.

Strategies are needed to encourage the U.S. population to drink water when they are thirsty. Water provides a healthy, low-cost, zero-calorie beverage option. Free, clean water should be available in public settings, as well as child care facilities, schools, worksites, publically funded athletic stadiums and arenas, transportation hubs (e.g., airports) and other community places and should be promoted in all settings where beverages are offered.

Review of the Evidence

These conclusions were reached by examining data from the WWEIA Food Categories data from the NHANES 2009-2010 dietary survey,[65] as described in relation to question 9 (current consumption patterns by food categories in the U.S. population). For this question, a new grouping of all beverages, including fluid milk and 100% fruit juice, was created. The conclusions and details below are based on this category of all beverages (see ***Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food Categories***).

All beverages account for about one-fifth (19 percent) of total energy intake. Within that amount, about one-third (35 percent) is from sugar-sweetened beverages, mostly soft drinks and sweetened fruit drinks (see ***Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA Food Categories***). About 20 percent of the calories from beverages come from alcoholic beverages (21 percent), and milk and milk drinks made with whole and 2 percent fat (18 percent). About 10 percent of the calories from beverages come from 100% fruit and vegetable juice (10 percent), fat-free and low-fat milk and milk drinks (8 percent), and coffee and tea (8 percent) (Figure D1.38).

For additional details on this body of evidence, visit:

- What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=23429.
- Appendix E-2.7: Major categories and subcategories used in DGAC analyses of WWEIA Food Categories
- Appendix E-2.9: Percent of total energy and nutrient intake, 2009-2010, for the U.S. population ages 2 years and older, from WWEIA food categories

EATING BEHAVIORS—CURRENT STATUS AND TRENDS

Diet quality and energy balance directly affect health and weight status. Eating behaviors, such as when people eat (e.g., patterns of meals and snacks, meal and snack frequency), meal skipping, and the locations where food is obtained and consumed (e.g., retail and restaurants) influence dietary intake and quality. Assessing and understanding eating behaviors of the U.S. population can shed light on ways to improve food choices, weight status, and health outcomes of Americans.

Question 13: What are the current status and trends in the number of daily eating occasions and frequency of meal skipping? How do diet quality and energy content vary based on eating occasion?

Source of evidence: Data analysis

Conclusion

The majority of the U.S. population consumes three meals a day plus at least one snack. Children ages 2 to 5 years are most likely to consume three meals a day and adolescent females, young adult males, non-Hispanic Blacks, Hispanics, and individuals with lower incomes are least likely to consume three meals a day. Trend data from 2005-2006 to 2009-2010 show little change in meal and snack intake patterns.

Breakfast tends to have a higher overall dietary quality because of its higher nutrient density compared to other meals and snacks. Adolescents and young adults are the least likely to eat breakfast. Snacks contribute about one-fourth of daily energy intake for the U.S. population and are lower in nutrients of concern relative to energy intake than are meals. For young children ages 2 to 5 years, 29 percent of daily energy is from snacks.

Implications

Understanding eating behaviors is important for designing and implementing strategies to reduce obesity and other diet-related chronic diseases and for improving overall health. Breakfast eating is associated with more favorable nutrient intakes compared to nutrient intakes from other meals or snacks. Adolescents and young adults are the least likely to eat breakfast, and targeted promotion efforts are needed to reach these groups. For children and adolescents, the school breakfast program is an important venue for promoting breakfast consumption and efforts are needed to increase student participation rates.

Americans are frequent snackers and snacks contribute substantially to daily energy intake and tend to be lower than meals in shortfall nutrients of public health concern relative to energy intake. Because snack foods and beverages are readily available and accessible in multiple settings throughout the day, both population-level environmental changes and individual behavioral interventions and communications are needed to ensure that healthy choices are available in these settings and to minimize their contribution to excess energy intake.

Individuals with lower incomes are less likely to eat three meals a day compared to higher income individuals and low-income households are more likely to be food insecure. The federal nutrition programs play a key role in reducing food insecurity and improving nutritional health.

Review of the Evidence

These conclusions were reached by examining existing WWEIA NHANES data tables,[5] from NHANES 2009-2010 for current intakes, and WWEIA, NHANES 2003-2004, 2005-2006 and 2007-2008 data for trends. Respondents self-identified the specific meal or snack occasion for each food and beverage consumed.

Eating Occasions: Meals. Three meals a day is the current norm for most of the U.S. population ages 2 years and older, with almost two-thirds (63 percent) eating breakfast, lunch, and dinner (Figure D1.39). However, there are differences by age, sex, racial/ethnicity group, and income level. By age group, consuming three meals a day follows a modest U-shaped curve where it is most likely for children ages 2 to 5 years (84 percent). It then declines, and reaches its lowest point during adolescence and young adulthood, and then increases with age through the adult years. Adolescent females (12 to 19 years) and young adult males (20 to 29 years) are the most likely to not eat three meals a day (49 percent). For all other age/sex groups, eating three meals a day is reported by 59 to 73 percent of respondents. Eating only one meal a day is most likely for young adult males (12 percent) and adolescent females (10 percent). However, all but 1 percent of these respondents, consumed at least two or more snacks a day (Table D1.13).

Among the U.S. population ages 2 years and older, 15 percent do not eat breakfast, 20 percent do not eat lunch, and 7 percent do not eat dinner. Breakfast is most likely to be skipped by young adults ages 20 to 29 years (28 percent of males, 22 percent of females) and adolescents (25 percent of females, 26 percent of males). Breakfast skipping declines sharply with advancing age. Lunch is not eaten by 25 percent of adolescent females and from 17 to 28 percent of all adult age groups (Table D1.14).

Non-Hispanic whites are most likely to report consuming three meals a day, across all age/sex/racial/ethnic groups, with 68 percent reporting breakfast, lunch, and dinner consumption. For non-Hispanic Blacks, slightly less than half (48 percent) consumed all three meals, and for all Hispanics, slightly more than half (52 percent). Non-Hispanic Blacks ages 12 to 19 years and 20 years and older, and Hispanics ages 12 to 19 years, were least likely to consume three meals a day (42 percent, 45 percent, and 45 percent, respectively) and most likely to consume only one meal a day (18 percent, 11 percent, and 10 percent).[66]

The percent of individuals consuming three meals a day increases with higher income levels. For those below 131 percent and from 131 to 185 percent of the poverty threshold, 53 percent and 56 percent report three meals a day, while for those above 185 percent of the threshold, 70 percent report three meals a day. For lower income individuals, the lower number of meals consumed per day is much more evident for older children and adults. Among children ages 2 to 5 years in the three income groupings, 81 percent, 82 percent, and 88 percent, respectively, report consuming three meals a day, while for adults ages 20 years and older, the corresponding percentages are 48 percent, 54 percent, and 70 percent, respectively.[67]

Eating Occasions: Snacks. Nearly all of the U.S. population ages 2 years and older consume at least one snack a day (96 percent). The most common snacking pattern for most age, sex, racial/ethnic and income groups is two to three snacks per day. Females and males ages 70 years and older are most likely to report eating one or fewer snacks per day (26 percent), and children ages 2 to 5 years are the least likely (10 percent). Children ages 2 to 5 years are most likely of any age group to report four or more snacks per day, across all racial/ethnic groups.[68]

The number of individuals reporting one or fewer snacks per day is highest (25 percent) for those below 131 percent of the poverty threshold, and lowest (17 percent) for those above 185 percent of the threshold. Consumption of four or more snacks per day is lowest (25 percent) for those below 131 percent of the poverty threshold and highest (35 percent) for those above 185 percent of the threshold. However, for all income groups, 2 to 3 snacks per day is the modal number and similar across income groups (51 percent, 48 percent, 48 percent).[67]

Trends. Trend data from NHANES from 2005-2006 to 2009-2010 show little change in number of daily eating occasions or frequency of meal skipping (Table D1.15).

Diet Quality and Energy content by Eating Occasion. For this analysis, diet quality is defined as a comparison of nutrient or food group content to energy content of a specified set of foods or beverages. In this question, diet quality compares the proportion of total nutrient intake at a given eating occasion to the proportion of energy intake at that eating occasion.

This analysis is summarized in Figure D1.40 and described below. In looking at this Figure, it should be noted that percent of total intake of nutrients of concern are shown in comparison to percent of total energy. If a nutrient is above the energy line, the meal/snack is a relatively higher source of that nutrient. If it is below the energy line, it is a relatively lower source.

Breakfast has a higher overall diet quality compared to lunch, dinner or snacks. Breakfast consists of 15 to 20 percent of the day's total energy intake (Table D1.16) but has a higher percent of nutrients. For all the shortfall nutrients of public health concern (fiber, folate, vitamin D, calcium, iron, and potassium), a higher percent of the day's total intake was consumed compared to the percent of energy consumed (Figure D1.40)

Among the U.S. population ages 2 years and older, about one fourth (24 percent) of daily energy intake is consumed at lunch and about one-third (35 percent) is consumed at dinner (Table D1.16). In terms of dietary quality, lunch is neutral, with similar percents of total nutrients and energy intakes for most nutrients. Dinner, which provides the greatest amount of daily total energy intake, has a higher percent of fiber, and potassium in comparison to percent energy, but calcium and several other nutrients are lower in comparison to percent energy. Sodium and saturated fat are higher as a percent of their total intakes than is energy intake. Further, the percent of total daily intake of sodium and saturated fat consumed at dinner is higher compared to other meals and snacks (Figure D1.40).

About one-fourth (24 percent) of daily energy intake comes from snacks. For young children ages 2 to 5 years, 29 percent of daily energy is from snacks (Table D1.17). Snacks provide the lowest percent of key nutrients (protein, iron, vitamin D, fiber, and potassium) relative to the percent of energy provided. Snacks provide 42 percent of the daily intake of added sugars. A lower percent of total sodium than of energy is provided by snacks. Snacks provide roughly the same percent of total intake of calcium as they do energy. This is also true of saturated fat for females (Table D1.17).

For additional details on this body of evidence, visit:

- Percent of the U.S. population consuming or skipping meals and snacks, 2001-2002, 2005-2006, 2007-2008, and 2009-2010 by age/sex groups, race/ethnicity, and percent of the poverty threshold. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349.
- Percent of total energy and nutrient intake by meal/snack, 2001-2002, 2005-2006, 2007-2008 and 2009-2010 by age/sex groups, race/ethnicity, and percent of the poverty threshold. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349.

Question 14: What are the current status and trends in the location of meal and snack consumption and sources of food and beverages consumed at home and away from home? How do diet quality and energy content vary based on the food and beverage source?

Source of evidence: Data analysis

Conclusion

About two-thirds of the calories consumed by the U.S. population are purchased at a store (69 percent), such as a grocery store or supermarket, and consumed in the home. The percent of calories eaten away from home (32 percent) has remained about the same since 2003-2004.

Food group and nutrient quality as measured by the Healthy Eating Index (HEI) vary by where food is obtained. Despite this, no matter where the food is obtained, diet quality of the U.S. populations does not meet recommendations for fruit, vegetables, dairy, whole grains, and exceeds recommendations for sodium, saturated fats, refined grains, solid fats, and added sugars.

Implications

The overall diet quality of the U.S. population's dietary patterns, regardless of where the food is purchased and eaten, is of major public health concern. Given that fruit, vegetables, dairy, and whole grains are consumed in less than recommended amounts and that sodium, saturated fats, refined grains, solid fats, and added sugars exceed recommended levels, urgent action is needed at individual and population levels to alter food purchasing and consumption habits.

Efforts are needed by the food industry and food retail (food stores and restaurants) sectors to market and promote healthy foods. The general public needs to be encouraged to purchase these healthier options. Making healthy options the default choice in restaurants (e.g., fat-free/low-fat milk instead of sugar-sweetened beverages, and fruit and non-fried vegetables in Children's Meals, whole wheat buns instead of refined grain buns for sandwich meals) would facilitate the consumption of more nutrient dense diets. Food manufacturers and restaurants should reformulate foods to make them lower in overconsumed nutrients (sodium, added sugars and saturated fat) and calories and higher in whole grains, fruits and vegetables.

In addition, Federal regulations for food labeling need to be updated. Food labels are an important tool to enable the public to follow the Dietary Guidelines and to make healthy food choices. They provide consumers with quick, easy to use information about the food they are purchasing. They also lead food companies to reformulate their food products to meet consumer demand. As recently proposed by the FDA, updates are needed in the Nutrition Facts label on packaged foods to emphasize calories, serving sizes, and nutrients of concern (including overconsumed nutrients such as sodium). Consumers also may benefit from a standardized Front of Pack label that gives clear guidance such as proposed by the IOM panel on FOP labeling.[69]

In addition to regulatory, policy, environmental and organizational changes, individual behavioral strategies are also needed to help Americans improve dietary behaviors. Comprehensive lifestyle interventions in a variety of settings and nutrition counseling by professionals in health care settings can modify dietary behaviors and improve health outcomes.

Review of the Evidence

This conclusion was reached by examining a new analysis of WWEIA, NHANES food intake data, from WWEIA NHANES 2009-2010 for current status, and WWEIA NHANES 2003-2004, 2005-2006 and 2007-2008 for trends (see ***Appendix E-2.13: Percent of energy intake from major points of purchase and location of eating, 2003-2004, 2005-2006, 2007-2008, 2009-2010, for the U.S. population ages 2 years and older*** and ***Appendix E-2.14: Food group and nutrient content of foods per 1000 calories obtained from major points of purchase, 2003-2004, 2005-2006, 2007-2008, 2009-2010, for the U.S. population ages 2 years and older***). This analysis was requested by the DGAC to answer the question. In addition, the DGAC reviewed the ERS publication *Nutritional Quality of Food Prepared at Home and Away from Home, 1977-2008*[70] to ascertain longer-term trends.

Respondents self-identified the food source (point of purchase) for each food or beverage they reported. For this analysis, food sources were grouped into the following categories: stores (grocery, supermarket, convenience/corner stores), full-service restaurants (defined as table service restaurants), quick-serve restaurants (includes fast food, counter service, and vending machines), school (includes child care). The location of eating, either at home or away from home, also was examined (Figure D1.41).

Americans increased their away-from-home share of caloric intake from 18 percent in 1977-1978 to 32 percent in 2005-2008, mainly from full service and fast food restaurants.[70] The percent of calories eaten away from home has remained roughly the same since 2003-2004. In 2009-2010, 69 percent of calories consumed by Americans were purchased from a store and 58 percent were eaten at home. This is about the same percent from 2003-2008 (Figure D1.41).

Diet quality was assessed using a density approach expressed as the amount of food group or nutrient per 1000 calories consumed, for each source from which food is obtained. The point of purchase (e.g., food store) is used as a proxy for where the food is consumed (e.g., home) because most food from stores are consumed at home, and most foods from other points of purchase are consumed away from home. Diet quality for a food group or nutrient for each food source obtained/consumed was then compared to the standard for a optimal HEI score per 1000 calories.[71] For saturated fat intake, the amount from each source was compared to the 2010 Dietary Guidelines limit for saturated fat intake.

Fruit. Fruit group density (cups per 1000 calories) is well below the HEI standard regardless of where the food is obtained or consumed. Amounts of fruit obtained and consumed differ by source, with full service and fast-food restaurants providing much less fruit per 1000 calories compared to other sources. This changed little from 2003-2004 to 2009-2010. Amount of fruit per 1000 calories is highest from schools/day care, and increased from 2003-2004 to 2009-2010, especially from 2007-2008 on (Figure D1.42).

Vegetables. Density for vegetables (cups per 1000 calories) falls below recommended intakes regardless of where food is obtained (Figure D1.43). Amounts of total vegetables and the starchy and other vegetable subgroups are shown in Figures D1.43 and D1.44. (Other vegetables are those not in the dark green, red orange, or starchy subgroups, such as green beans, iceberg lettuce, onions, cabbage, cucumbers.) Amounts of total vegetables and other vegetables per 1000 calorie are highest for restaurants, especially full service restaurants, with a slight downward trend from 2007-2008 to 2009-2010 (Figures D1.43 and D1.44). Amounts of total vegetables and starchy vegetables per 1000 calories from schools/daycare show a suggestive decrease in 2009-2010 compared to earlier years. Density for all vegetable subgroups by source for 2003-2004 through 2009-2010 are listed in Table D1.18.

Dairy. Amounts of total dairy products (fluid milk, cheese, and yogurt) are highest from schools/day care sources and are above the HEI standard, with an increase from 2007-2008. Amounts from other sources are far below recommendations (Figure D1.45).

Whole and refined grains. Whole grain density per 1000 calories is far below the HEI standard and is low for all food sources with little change since 2003-2004. On the other hand, refined grains exceed the HEI limit for all food sources, with the highest amount coming from quick serve restaurants (Figure D1.46).

Protein foods. Amounts of total protein foods per 1000 calories are above the HEI standard for full service restaurants and fast food restaurants (Figure D1.47).

Sodium. Amounts of sodium per 1000 calories are well above the HEI limit and do not differ greatly across sources. However, the density from full service and fast food restaurants are somewhat higher than from stores. There has been little change from 2003-2004 to 2009-2010 (Figure D1.48).

Saturated fats. Amounts of saturated fat per 1000 calories is well above the Dietary Guidelines limit and do not differ greatly across sources. However, the density from fast food restaurants is somewhat higher than from stores. There has been little change from 2003-2004 to 2009-2010 (Figure D1.49).

Empty calories. (defined as the total calories from solid fats and added sugars). Empty calories are well above the HEI limit (190 calories per 1000 calories) for all food sources, with the highest amount from fast food restaurants, but no large differences among sources. Empty calories have trended downward since 2003-2004 (Figure D1.50). The HEI does not have a separate HEI standard for added sugars and solid fats. Both added sugars and solid fats have decreased since 2003-2004. (Figures D1.51, D1.52) The highest amounts of added sugars are obtained from stores and the highest amounts of solid fats are obtained from fast food restaurants.

Food group density by age group. For children ages 2 to 5 years, fruit group density per 1000 calories from schools and stores reaches the HEI standard. School foods provide the highest fruit density among all food sources for 6-11 year olds, with an increase since 2007-2008. All other age groups do not reach the HEI standard for fruit from any source, although the store location is consistently the top source for adults. Vegetable density from full service restaurants reaches the HEI standard for ages 51-70 and 71 years and older. All sources of vegetables are below the standard for children, adolescents and adults under age 50. Dairy product density from child care and stores meet the HEI standard for children ages 2-5 and from schools for children ages 6-19. School foods provide the highest dairy product density among all food sources in children's diets. For school age children and adolescents, school foods are the only food source that meets the recommended amount of dairy products. Among adults, dairy product density is low for all sources. For children ages 6-11, there is a difference in the added sugars density by source, with schools having less added sugars per 1000 calories than other sources. This difference is not as clear for younger children or adolescents. For adults the highest amount of added sugars per 1000 calories is from stores. For most age groups, there is a slight downward trend, especially in the density of added sugars from stores (see ***Appendix E-2.15: Amount of key nutrients and food groups by age group per 1000 calories from each point of purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010***).

For additional details on this body of evidence, visit:

- Appendix E-2.13: Percent of energy intake from major points of purchase and location of eating, 2003-2004, 2005-2006, 2007-2008, and 2009-2010, for the U.S. population ages 2 years and older

- Appendix E-2.14: Food group and nutrient content of foods per 1000 calories obtained from major points of purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010, for the U.S. population ages2 years and older
- Appendix E-2.15: Amount of key nutrients and food groups by age group per 1000 calories from each major point of purchase, 2003-2004, 2005-2006, 2007-2008, and 2009-2010
- ERS report, Nutritional Quality of Food Prepared at Home and Away from Home, 1977-2008. Available from: http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib105.aspx.

PREVALENCE OF HEALTH CONDITIONS AND TRENDS

Preventable, diet- and lifestyle-related chronic diseases, including high blood pressure, CVD, type 2 diabetes, and certain cancers, contribute to the high and rising costs of U.S. health care. Adults with overweight or obesity frequently have co-morbid conditions and higher chronic disease risk profiles that contribute substantially to higher health care costs. These health problems are persistent in the population and pose major public health concerns. Increasing rates of overweight and obesity among American youth have resulted in rising rates of CVD risk factors, including borderline high blood pressure and diabetes, in this population. Health disparities in risk profiles and disease rates are evident across racial, ethnic, and income strata. In a new health care and public health vision, prevention of chronic diseases and other lifestyle-related health problems would become a major focus. Examining the status and trends in these health conditions provides a framework for discussing their relationship to dietary intake and lifestyle factors and can help in identifying evidence-based strategies for prevention.

Question15: What is the current prevalence of overweight/obesity and distribution of body weight, BMI, and abdominal obesity in the U.S. population and in specific age, sex, racial/ethnic, and income groups? What are the trends in prevalence?

Source of evidence: Data analysis

Conclusion

The current rates of overweight and obesity are extremely high among children, adolescents, and adults. These high rates have persisted for more than 25 years.

Overall, 65 percent of adult females and 70 percent of adult males are overweight or obese, and rates are highest in adults ages 40 years and older. Rates of overweight and obesity in adults vary by age and race/ethnicity.

- Overweight (excluding obesity) is most prevalent in those ages 40 years and older, and in Hispanic American adults.
- Obesity is most prevalent in those 40 years of age or older and in African American adults. Obesity is least prevalent in adults with highest incomes (400+ percent the poverty threshold).

Abdominal obesity is present in U.S. adults of all ages, increases with age, and varies by sex and race/ethnicity.

- Abdominal obesity rates are highest in individuals ages 60 years and older, and are higher in women than men at all ages.

- In men, abdominal obesity rates are slightly higher among non-Hispanic whites than Mexican Americans or African Americans. In women, abdominal obesity rates are lower in non-Hispanic whites than in Mexican Americans or African Americans.

Nearly one in three youth (31 percent), ages 2 to 19 years, is now overweight (85^{th}-94^{th} percentile) or obese ($\geq 95^{th}$ percentile) and these rates vary by age and ethnicity.

- In youth ages 2 to 19 years, obesity prevalence increases with age, and the age category with the highest prevalence is 12-19 year olds.
- In youth ages 2 to 19 years, the race categories with the highest prevalence of obesity are African Americans and Hispanics.

Implications

The persistent high levels of overweight and obesity require urgent population- and individual-level strategies across multiple settings, including health care, communities, schools, worksites, and families.

Comprehensive lifestyle interventions and evidence-based dietary interventions for weight management in individuals and small groups should be developed and implemented by trained interventionists and professional nutrition service providers in healthcare settings as well as in community locations, including public health facilities and worksites.

Quality of care standards in health care settings should include the provision and impact of preventive nutrition services provided by multidisciplinary teams of trained interventionists, as appropriate, and nutrition professionals. Incentives should be offered to providers and systems to develop preventive services.

The public should be encouraged to monitor their body weight and engage with their health care providers at least annually to assess their body weight and BMI. As appropriate, providers should use evidence-based approaches aimed at achieving and maintaining healthy body weight. Health care providers should encourage achieving and maintaining a healthy weight through healthy eating and physical activity behaviors.

The persistent high rates of obesity across the lifespan show the limited impact of our efforts to date. Accelerating progress in reversing obesity trends will require a more targeted, comprehensive, and coordinated strategy and a renewed commitment and action for sustained, large-scale, integrated multi-sectoral and cross-sectoral collaborations. Government at local, state, and national levels, the health care system, schools, worksites, community organizations, businesses, and the food industry all have critical roles in developing creative and effective solutions.

Behavioral change at the individual level is important. However, policy interventions that make healthy dietary and activity choices easier, more routine, and affordable and that reduce unhealthy options are likely to achieve population-wide benefits.

Age-appropriate nutrition and food preparation education should be a mandatory part of primary and secondary school curricula.

Review of the Evidence

To reach these conclusions, the DGAC examined evidence from NHANES 2009-2012, and additional survey years including 1988-1994 to 2011-2012 for trends data. These data are available in summary NHANES data table format on the CDC website, in published peer-reviewed articles by CDC,[72-74] and in analyses requested by the DGAC and provided by CDC/NCHS (see ***Appendix E-2.16: Body mass index, adults ages 20 years and older, NHANES 2009-2012*** and ***Appendix E-2.17: Body mass index, children and adolescents ages 2-19 years, NHANES 2009-2012***).

The prevalence rates of overweight and obesity among U.S. adults have been extremely high for the past 25 years and appear to be at record high levels in women and to have plateaued at near record high levels in men (Figure D1.53). In 2009-2012, combined rates of overweight and obesity in adult men, ages 20 years and older, were 72.6 percent (38.1 percent for overweight and 34.5 percent for obesity) and 64.8 percent (28.8 percent for overweight and 36 percent for obesity) in women (Table D1.19). Rates of overweight and obesity in adults vary by age and ethnicity and are most pronounced in adults ages 40 years and older and in Hispanic and African American adults (Table D1.19).

Overweight affects 29.5 percent of adults ages 20 to 39 years, 35.9 percent of adults ages 40 to 59 years, and 35.7 percent of adults ages 60 years and older, while obesity affects 31.5 percent of adults ages 20 to 39 years, 38 percent of those ages 40 to 59 years, and 37.5 percent of those ages 60 years and older (Table D1.19).

Overweight affects 31.7 percent of adult African American men and 24.5 percent of adult African American women, while obesity affects 37.9 percent of adult African American men and 57.5 percent of adult African American women. Among adult Hispanic men, overweight affects 41.5 percent and obesity affects 38.5 percent, and among adult Hispanic women, overweight affects 33.5 percent and obesity affects 43 percent (Table D1.19).

Obesity is least prevalent (about 31 percent) in adults ages 20 years and older with highest incomes (400 + percent the poverty threshold) in 2007-2010 (Table D1.20), while affecting 37.2 percent of those with incomes below 100 percent of the poverty threshold, 37.3 percent of those with incomes from 100 percent to 199 percent of the poverty threshold, and 36.8 percent of those with incomes from

200 percent to 399 percent of the poverty threshold (Table D1.20). Across all income strata, combined rates of overweight and obesity and particularly obesity rates have risen over the past 25 years.

Abdominal obesity, as measured by waist circumference (WC), and defined as WC more than 102 cm in men and more than 88 cm in women, is a risk factor for CVD and diabetes .[6] Abdominal obesity is prevalent in U.S. adults of all ages and varies by age and sex. In 2011-2012, overall rates of abdominal obesity were about 54 percent in adults ages 20 years and older, with a prevalence of about 44 percent in adult men and 65 percent in adult women (Table D1.21). Data from the NHANES 2007-2008 survey shows that men ages 20 to 39 years have the lowest rates of abdominal obesity (28.5 percent) compared to men ages 40 to 59 years (49.4 percent) and those ages 60 years and older (60.4 percent) (Table D1.21). Women ages 60 years and older have the highest rates of abdominal obesity (73.8 percent) compared to women ages 40 to 59 and 20 to 39 years (65.5 percent and 51.3 percent, respectively). Data from the 2011-2012 survey show that the highest prevalence of abdominal obesity among men is in non-Hispanic white men (44.5 percent), followed by Mexican American men (43.2 percent) and African American men (41.5 percent), while the highest prevalence among women is in African American women (75.9 percent), followed by Mexican American (71.6 percent) and non-Hispanic white women (63.3 percent) (Table D1.21). For 2007-2010, the prevalence of abdominal obesity is very high in obese adults ages 18 years and older (97 percent), and overweight adults (57 percent), compared to normal/underweight adults (8 percent).[75] Since 1999 rates of abdominal obesity have risen in all age and racial strata of both adult males and females (Table D1.21).

After increasing from the 1980s until about 2004, rates of overweight and obesity in children and adolescents ages 2 to 19 years have since remained at very high levels (Figure D1.54). A significant decrease in obesity among children ages 2 to 5 years old was observed in an analysis comparing the survey data from 2003-2004 (13.9 percent) to 2011-2012 (8.4 percent).[74] However, it is not clear whether this comparison of only two time periods reflects an actual downward trend. Currently, 14.9 percent of boys ages 2 to 19 years are overweight (85^{th} to 94^{th} percentile) and 17.6 percent are obese (95^{th} percentile and greater); rates in girls ages 2 to 19 years are 14.9 percent and 16.1 percent, respectively. Furthermore, rates of obesity in youth increase with age and vary by ethnicity, with obesity found in 22.1 percent of African American and 21.8 percent of Hispanic Americans ages 2 to 19 years (Table D1.22).

For additional details on this body of evidence, visit:

- Appendix E-2.16: Body mass index, adults ages 20 years and older, NHANES 2009-2012
- Appendix E-2.17: Body mass index, children and adolescents ages 2-19 years, NHANES 2009-2012

Question 16: What is the relative prevalence of metabolic and cardiovascular risk factors (i.e., blood pressure, blood lipids, and diabetes) by BMI/body weight/waist circumference (abdominal obesity) in the U.S. population and specific population groups?

Source of evidence: Data analysis

Conclusion

Approximately 50 percent of adults who are normal weight have at least one cardiometabolic risk factor. Approximately 70 percent of adults who are overweight and 75 percent of those who are obese have one or more cardiometabolic risk factors.

Rates of elevated blood pressure, adverse blood lipid profiles (i.e., low high density lipoprotein cholesterol [HDL-C], high low density lipoprotein choldesterol [LDL-C], and high triglycerides), and diabetes are highest in adults with elevated abdominal obesity (waist circumference greater than 102 cm in men, greater than 88 cm in women).

Ninety-three percent of the children with type 2 diabetes are ages 12 to 19 years and 90 percent of these children with type 2 diabetes are overweight or obese. In children with type 2 diabetes, the prevalence of obesity is higher in African Americans, followed by American Indians and Hispanics, compared to non-Hispanic whites or Asian Pacific Islander youth.

Dyslipidemia and rates of borderline high blood pressure vary by weight status in boys and girls; rates are particularly high in obese boys.

Nearly three-fourths of the overweight or obese populations have at least one cardiometabolic risk factor.

Implications

The rates of cardiometabolic risk factors in adult Americans are extremely high and reflect the high rates of population overweight and obesity. Many adults have personal health profiles in which multiple metabolic risk factors co-exist and substantially increase risks for coronary heart disease, hypertension and stroke, diabetes, and other obesity-related co-morbidities. These are the most costly health problems in the Nation today and they can be prevented or better managed with intensive, comprehensive, and evidence-based lifestyle interventions carried out by multidisciplinary teams of trained professionals or through medical nutrition therapy provided by registered dietitians or nutritionists (AHA/ACC/TOS).[2] Program plans and interventions needed to confront the nation's obesity epidemic and its devastating metabolic consequences. A shift in the healthcare paradigm toward prevention is critical. Nutrition and lifestyle services for obesity prevention and weight management should be expanded and integrated. As part of this approach, quality of care guidelines need to be revised to incentivize the provision of personalized lifestyle and nutrition interventions to combat obesity and obesity-related chronic diseases and their metabolic risk factors and co-morbidities. As emphasized in ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*** and ***Part D. Chapter 4: Food Environment and Settings***, the most effective approach to preventing and treating overweight and obesity in our nation across the lifespan requires both individual and population-based, environmental strategies. Initiatives in health care and public health and other government sectors should be complemented with collaborative approaches in retail, educational, and social service and agricultural settings to make the long-term adoption of healthy nutrition and lifestyle behavior not only feasible but normative.

The high rates of overweight and obesity in youth and their concomitant cardiometabolic risk factors require early preventive interventions at individual and population levels. Evidence-based strategies in health and public health settings also should be implemented and complemented by environmental approaches across wide-ranging sectors to reverse these priority health problems.

Review of the Evidence

To reach these conclusions, the DGAC examined evidence from NHANES 2007-2010 and 2009-2012 data and SEARCH for Diabetes in Youth Study (SEARCH). These data were available in published peer-reviewed articles by CDC,[76] or SEARCH[77] authors and in analyses requested by the DGAC and provided by CDC/NCHS (see ***Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adults ages 20 years and older, NHANES 2009-2012***, ***Appendix E-2.19: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages 20 years and older, NHANES 2009-2012***, ***Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older, NHANES 2009-2012***, ***Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012***, ***Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol, children and adolescents ages 6–19 years, NHANES 2009-2012***, ***Appendix E-2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents ages 12-19 years, NHANES 2009-2012***, ***Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and adolescents ages 8-17 years, NHANES 2009-2012***).

In U.S. adults ages 18 years and older, weight status is related to prevalent CVD risk. About two-thirds (66.6 percent) of U.S. adults, including more than half (56.1 percent) of normal weight adults (BMI 18.5-<25 kg/m^2), have one or more CVD risk factors (including type I and type II diabetes, hypertension, or dyslipidemia, or self-reported smoking) (Figure D1.55). About 70 percent (69.6 percent) of adults who are overweight (BMI 25-<30 kg/m^2) have at least one or more CVD risk factors, making them candidates for preventive weight management interventions, according to expert guidelines established by the American College of Cardiology, American Heart Association, and The Obesity Society for preventative weight management (see ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***). Furthermore, more than one-quarter (27.8 percent) have two or more CVD risk factors (Figure D1.55). About three-quarters (74.6 percent) of adults who are obese (BMI $\geq$30 kg/m^2) have one or more CVD risk factors and about 39 percent have two or more CVD risk factors (Figure D1.55). Cardio-metabolic risk factors also are substantially more prevalent in adult men and women who have abdominal obesity (Table D1.23).

In terms of plasma lipids, the prevalence of low HDL-C (<40 mg/dl), high LDL-C (≥160 mg/dl), and high triglycerides (≥ 200 mg/dl) is highest in obese adults (ages 20 years and older) compared to normal weight adults (Table D1.23). Similar patterns are observed in those who are overweight compared to normal weight adults (Table D1.23). These lipid profiles also are highest in men with abdominal obesity (> 102 cm) or women (>88 cm). (Table D1.23). High total cholesterol (≥ 200 mg/dl), low HDL-C (<40 mg/dl), and high triglycerides (≥ 130 mg/dl) also are most prevalent in obese compared to overweight or normal weight children and adolescents (Table D1.24). There does not appear to be a difference in the prevalence of high LDL-C (≥ 130 mg/dl) by weight status in children and adolescents (Table D1.24).

In adults ages18 years and older, rates of elevated blood pressure (defined as having measured systolic pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication) are highest with obesity (39.2 percent) compared to normal weight (20 percent) or overweight (26.4 percent). It is also highest in those with elevated waist circumferences (men > 102cm (37.2 percent vs 23.3 percent; and > 88 cm in women (32.9 percent vs 17.8 percent) (Table D1.23). Similar to adults, the rate of borderline high blood pressure (defined as a systolic or diastolic blood pressure ≥ 90th percentile but < 95th percentile or blood pressure levels ≥ 120/80 mm Hg) in youth ages 8 to 17 years was highest in with obesity (16.2 percent) compared to those who are normal weight (5.4 percent) or overweight (10.9 percent) (Table D1.25). Diabetes in adults ages 20 years and above also increases with body mass index from 5.5 percent in those who are of normal weight, to 9 percent in overweight and 20.3 percent in obese adults and is more prevalent in those with abdominal obesity (men > 102cm (19.6 percent vs 8.3 percent); and > 88 cm in women (13.9 percent vs 2.6 percent) (Table D1.23).

Data from 2001 to 2004 in children (ages 3 to 19 years) participating in the SEARCH for Diabetes in Youth Study (SEARCH) show that 93 percent of youth with type 2 diabetes are ages 12 to 19 years. The prevalence of obesity among youth with type 2 diabetes is 79.4 percent and an additional 10.4 percent are overweight (Table D1.26). The percentage of overweight among youth with type 2 diabetes is not significantly different than rates in U.S. youth who do not have type 2 diabetes.[77] However, the prevalence of obesity among youth with type 2 diabetes (79.4 percent) is much higher than in U.S. youth without type 2 diabetes (16.9 percent) (Table D1.26). The prevalence of obesity in those with type 2 diabetes was higher in African Americans (91.1 percent), followed by American Indians (88 percent), and Hispanics (75 percent) in comparison to non-Hispanic white or Asian Pacific Islander youths (about 68 percent for each) (Table D1.26).

For additional details on this body of evidence, visit:

- Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adult ages 20 years and older, NHANES 2009 -2012
- Appendix E-2.19: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages 20 years and older, NHANES 2009-2012
- Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older, NHANES 2009-2012
- Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012
- Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol, children and adolescents ages 6-19 years, NHANES 2009-2012
- Appendix E-2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents ages 12-19 years, NHANES 2009-2012
- Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and adolescents ages 8-17 years, NHANES 2009-2012

Question 17: What are the current rates of nutrition-related health outcomes (i.e., incidence of and mortality from cancer [breast, lung, colorectal, prostate] and prevalence of CVD, high blood pressure, diabetes, bone health, congenital anomalies, neurological and psychological illness) in the overall U.S. population?

Source of evidence: Data analysis

Conclusion

Adults have high rates of nutrition-related chronic diseases, including high blood pressure, CVD, diabetes, and various forms of cancer. Children and adolescents also have nutrition-related chronic diseases, including borderline high blood pressure and type 2 diabetes. At all ages, rates of chronic disease risk are linked to overweight and obesity. The rates of these chronic diseases vary by race/ethnicity and income status. Prevalence of osteoporosis and of low bone mass increases with age, particularly in post-menopausal women. Among the less common health outcomes:

- Nutrition-related neurological and psychological conditions are a growing concern.
- Congenital anomalies are a relatively rare, but important pregnancy outcome.

Implications

Given the high rates of nutrition-related chronic diseases in the adult population and rising rates in youth, it is imperative to develop prevention policies and programs that target all age groups and address nutrition and lifestyle issues with evidence-based interventions that are appropriate for delivery in multiple settings.

Qualified professionals should deliver multidisciplinary interventions and medical nutrition therapies, as appropriate, that are effective in reducing nutrition-related chronic diseases.

More studies are needed to understand the complex etiology of congenital anomalies and neurological and psychological conditions, and factors that influence bone health as well as healthy outcomes of pregnancy so as to inform potential dietary choices by the U.S. population.

Review of the Evidence

To reach these conclusions, the DGAC examined evidence from NHANES 2007-2010 and 2009-2012 (see ***Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adults ages 20 years and older, NHANES 2009-2012, Appendix E-2.19: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages 20 years and older, NHANES 2009-2012, Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older, NHANES 2009-2012, Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012, Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol, children and adolescents ages 6-19 years, NHANES 2009-2012, Appendix E-2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents ages 12-19 years, NHANES 2009-2012, Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and adolescents ages 8-17 years, NHANES 2009-2012***), the National Health Interview Survey (NHIS) 2012,[78] SEARCH for Diabetes in Youth Study,[79] American Heart Association, 2014 report,[6] and the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute.[80] The DGAC also examined evidence from CDC's population-based birth defects surveillance system,[81] Alzheimer's Association 2014 Facts and Figures,[82] and published data by CDC authors.[83]

Cardiovascular Diseases

Cardiovascular diseases, including coronary heart disease, hypertension, and stroke, affect an estimated 83.6 million (35.3 percent) men and women ages 20 years and older in the United States.[6] CVD increases with age, meaning that about half of those with CVD, 42.2 million adults, are ages 60 years and older.[6] Rates of coronary heart disease also vary by race/ethnicity and income. Coronary heart disease is most prevalent in Hispanics (7.8 percent of those reporting the disease) and Native Americans (including Alaskan natives 12.5 percent) adults.[78] Stroke is most prevalent in Native Americans (4.3 percent of those reporting the disease) and African Americans (3.9 percent).[78] Coronary heart disease rates are inversely related to income. Rates are about 9.8 percent and 7.7 percent in those with lower income (less than 100 percent of the poverty threshold and 100 to 199 percent, respectively) compared to those with higher income (200 percent and greater of the poverty threshold; 1.9 percent). Stroke also is more prevalent in those with incomes less than 100 percent of the poverty threshold (4.8 percent) and 100 to 199 percent of the poverty threshold (3.7 percent) compared to those with higher incomes (1.9 percent).[78]

The prevalence of elevated blood pressure (measured systolic pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication), in adults ages 18 years and older (29 percent) is similar in adult men (29.8 percent) and women (28.3 percent) and varies by age and race/ethnicity (Table D1.27). Rates of elevated blood pressure are highest in adults ages 60 years and older (66.3 percent), and African Americans (41.5 percent), relative to non-Hispanic whites (27.9 percent) or Hispanics (26.1 percent) (Table D1.27). A similar pattern is seen in youth ages 8 to 17 years, with borderline high blood pressure in 8.3 percent overall (Table D1.25). Boys (12 percent) are much more likely to have borderline high blood pressure than are girls (4.6 percent), as are those ages 13 to 17 years (12.4 percent) compared to those ages 8 to 12 years (3.8 percent), and African Americans (12.1 percent) compared to non-Hispanic whites (7.2 percent) and Hispanics (8.5 percent) (Table D1.25).

Diabetes

Total diabetes (type I plus type II) is the sum of self-reported diabetes and undiagnosed diabetes. Diagnosed diabetes was obtained by self-report and excludes women who reported having diabetes only during pregnancy. Undiagnosed diabetes was defined as fasting plasma glucose of at least 126 mg/dL or a hemoglobin A1C value of at least 6.5% and was not reported as a physician diagnosis. The prevalence of diabetes in U.S. adults, is 14 percent for men and 10.8 percent for women 20+ years of age (Table D1.27). Rates increase with age, to 26 percent for adults ages 60 years and older, and are higher in African Americans (18.4 percent) and Hispanics (19.3 percent) compared to non-Hispanic whites (9.8 percent) (Table D1.27). Between 2001 and 2009, rates of type 2 diabetes in children and adolescents ages 10 to 19 years increased 30.5 percent[79] and the disease now affects about 1 in 2,000 youth (0.46 per 1000) (Table D1.28). In 2009, type 2 diabetes appeared to be more common in girls than boys (0.58, vs. 0.35 /1000 youth), in older adolescents (ages15 to 19 years; 0.68) compared to those ages 10 to 14 years (0.23), and in American Indian (1.2), African American (1.06), and Hispanic (0.79) youth compared to non-Hispanic Whites (0.17) (Table D1.28).

Nutrition-related Major Cancers

Breast cancer: Breast cancer represents approximately 14 percent of all new cancer cases and 6.8 percent of all cancer deaths in the United States. In 2011, an estimated 2,899,726 (2.9 million) women in the United States had a history of breast cancer. About 232,670 new cases of breast cancer and 40,000 deaths from this disease are estimated for 2014. Breast cancer is the third leading cause of cancer death in the U.S.[80, 84] New cases of breast cancer are highest in the middle age and older women (about 22, 25.5, and 21.3 percent of new cases occur in women ages 45 to 54, 55 to 64 and 65 to 74 years, respectively) (Table D1.29) and in non-Hispanic white women (128/100,000 women per year), followed by African American (122.8/100,000 women). The death rate from this disease is also highest among women ages 55 to 84 years old (ranges 20.6 percent to 21.7 percent of deaths) and African

Americans (30.6 of death/100,000), followed by non-Hispanic white women (21.7/100,000) (Table D1.29).

Prostate cancer: Prostate cancer represents approximately 14 and 5 percent of all new cancer cases and all cancer death, respectively in U.S. men. In 2011, an estimated 2,707,821 (2.7 million) men had a history of prostate cancer. About 233,000 new cases of prostate cancer and 29,480 deaths from this disease are estimated for 2014. Prostate cancer is the fifth leading cause of cancer death in the United States.[84, 85] New cases of prostate cancer are most prevalent in older men (about 32.7, 36.3 and 16.8 percent of new cases in men ages 55 to 64, 65 to 74, and 75 to 84 years, respectively) (Table D1.29) and African American (223.9 of new cases/100,000 men). The death rate from this disease is highest among men ages 75 to 84 years old (36.8 percent of deaths) and African Americans (48.9/100,000) (Table D1.29).

Colorectal cancer: Colorectal cancer represents approximately 8.2 and 8.6 percent of all new cancer cases and all cancer death, respectively in the United States. In 2011, an estimated 1,162,426 (1.2M) adult men and women had a history of colorectal cancer. About 136,830 new cases of colorectal cancer and 50,310 deaths from this disease are estimated for 2014, respectively. Colorectal cancer is the second leading cause of cancer death in the United States.[84, 86] The incidence (new cases) of this cancer is more common in men than women and is more common in those older than age 55 years (highest frequency observed among those ages 65 to 74 years (23.9 percent) (Table D1.29) and in African Americans (62.3 and 47.5 new cases/100,000 persons in African American men and women, respectively). The death rate from this disease also is highest in people older than age 55 years old (highest frequency observed among those ages 75 to 84 years old (27.3 percent of deaths) and in African American (27.7, and 18.5 deaths/100,000 persons in men and women, respectively) (Table D1.29).

Lung and Bronchus cancer: Lung and bronchus cancer represents approximately 13.5 and 27.2 percent of all new cancer cases and all cancer deaths, respectively in the United States. In 2011, an estimated 402,326 people had a history of lung and bronchus cancer. About 224,210 new cases of lung and bronchus cancer and 159,260 deaths from this disease are estimated in 2014, respectively. This cancer is the first leading cause of cancer death in the United States.[84, 87] The incidence of lung and bronchus cancer is more common in men than women and is more common in those older than age 55 years (highest frequency observed among those ages 65 to 74 years (31.7 percent) in African American men (93 new cases/100,000 persons), and in white women (53.8/100,000 persons) (Table D1.29). The death rate from this disease also is higher in older people (highest frequency observed among those ages 65 to 84 years (about 30 percent of deaths) and in African American men (75.7 deaths/100,000 persons), and non-Hispanic white women (39.8/100,000 persons) (Table D1.29).

Bone Health

Approximately 10 million (10.3 percent) American adults ages 50 years and older were reported to have osteoporosis (defined as T-score ≤ -2.5 at either the femoral neck or the lumbar spine) and 43 million (44 percent) to have low bone mass (defined as T-scores between -1.0 and -2.5 at either skeletal site) in NHANES 2005-2010 (Table D1.30). A higher percent of women are affected by osteoporosis (15 percent) and low bone mass (51 percent) than men (about 4 percent and 35 percent, respectively). Osteoporosis increases with advancing age, occurring in about 35 percent in women ages 80 years and older compared to 26 percent in those ages 70 to 79 years old. The prevalence of low bone mass is similar in women ages 50 to 59 year and 80 years and older (ranges from 49 to 53 percent). Osteoporosis and low bone mass are more prevalent in Mexican American (20 percent, 48 percent) and non-Hispanic white (16 percent, 53 percent) relative to African American (8 percent, 36 percent) women (Table D1.30).

Congenital Anomalies

Each year, about 3 percent (one in every 33 babies) is born with spina bifida (without anencephaly); cleft lip (with and without cleft palate), or cleft palate (without cleft lip).[88] The estimated national prevalence of spina bifida was 3.17 per 10,000 live births in 1999-2007.[81] During this same time period, the prevalence of having a baby with spina bifida was reported to be more common in Native Americans/Alaska Natives (4.02/10,000 live birth), followed by Hispanics (3.8/10,000), non-Hispanic whites (3.09/10,000), African-Americans (2.73/10,000), and Asian/Pacific Islanders (1.2/10,000).[81] The estimated national prevalence of cleft palate and cleft lip is 5.67 and 9.3 per 10,000 live birth, respectively.[81] The prevalence of both of these congenital anomalies was highest in non-Hispanic Native Americans/Alaskan Natives (20/10,000 [cleft lip] and 6.5/10,000 [cleft palate]), and was lowest in African-Americans (6/10,000 [cleft lip] and 4.2/10,000 [cleft palate]).[81]

Congenital heart defects affect about 40,000 births (about 1 percent of births) per year in the United States.[89] The number of babies with congenital heart defects, especially those forms that are less severe (ventricular septal defects and atrial septal defects), is increasing compared to the total number of births, while the prevalence of other types has remained stable.[89]

Neurological and Psychological Conditions

There are numerous types of neurological and psychological conditions, and the DGAC focused only on depression and Alzheimer's disease. The prevalence of depression was estimated at 8 percent for the U.S. population ages 12 years and older in the NHANES 2007-2010 survey.[90] Depression is higher in females (10 percent) than in males (6 percent), and highest in those ages 40 to 59 years (12 percent women, 7 percent men).[90] Depression also is reported to be higher in African Americans (8 percent), followed by Mexican-Americans (6.3 percent) and non-Hispanic whites (4.8 percent) (NHANES 2005 -2006).[91]

In 2014, about 3.2 million women and 1.8 million men in the United States, ages 65 years and older are reported to be living with Alzheimer's disease.[82] This disease is most prevalent in those ages 75 to 84 years (44 percent of those with Alzheimer's) and those ages 85 years and older (38 percent).[82] About 63, 59, and 30 percent of those ages 85 years and older with Alzheimer's disease are reported to be Hispanics (primarily Caribbean-American), African Americans, and non-Hispanic white adults, respectively.[82] It has been projected that the number of people with Alzheimer's disease will increase by about threefold from 4.8 million in 2010 to 13.7 million in 2050.[92]

For additional details on this body of evidence, visit:

- Appendix E-2.18: Total cholesterol and high density lipoprotein cholesterol (HDL), adult ages 20 years and older, NHANES 2009-2012
- Appendix E-2.19: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults ages 20 years and older, NHANES 2009-2012
- Appendix E-2.20: Prevalence of high blood pressure, adults ages 18 years and older, NHANES 2009-2012
- Appendix E-2.21: Total diabetes, adults ages 20 years and older, NHANES 2009-2012
- Appendix E-2.22: Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol, children and adolescents ages 6-19 years, NHANES 2009 -2012
- Appendix E-2.23: Low density lipoprotein cholesterol (LDL-C) and triglycerides, adolescents ages 12-19 years, NHANES 2009-2012
- Appendix E-2.24: Prevalence of high and borderline high blood pressure (BP), children and adolescents ages 8-17 years, NHANES 2009-2012
- SEER Cancer Statistics Review, 1975-2011. Available from: http://seer.cancer.gov/csr/1975_2011/.
- SEER Stat Fact Sheets: Breast Cancer. Available from: http://seer.cancer.gov/statfacts/html/breast.html.
- SEER Stat Fact Sheets: Colon and Rectum Cancer. Available from: http://seer.cancer.gov/statfacts/html/colorect.html.
- SEER Stat Fact Sheets: Lung and Bronchus Cancer. Available from: http://seer.cancer.gov/statfacts/html/lungb.html.
- SEER Stat Fact Sheets: Prostate Cancer. Available from: http://seer.cancer.gov/statfacts/html/prost.html.
- Summary health statistics for U.S. adults: National Health Interview Survey, 2012. Available from: http://www.cdc.gov/nchs/data/series/sr_10/sr10_260.pdf.

- Respondent-reported prevalence of heart disease, cancer, and stroke among adults aged 18 and over, by selected characteristics: United States, average annual, selected years 1997-1998 through 2011-2012. Available from: http://www.cdc.gov/nchs/data/hus/2012/044.pdf.
- 2014 Alzheimer's disease facts and figures: includes a special report on women and Alzheimer's disease. Alzheimers Dement. 2014;10(2):131-68. PMID: 22404854. Available from: http://www.alz.org/downloads/facts_figures_2014.pdf.
- Facts about Birth Defects [updated October 20, 2014]. Available from: http://www.cdc.gov/ncbddd/birthdefects/facts.html.
- Depression in the United States household population, 2005-2006. NCHS Data Brief. 2008(7):1-8. PMID: 19389321. Available from: http://www.cdc.gov/nchs/data/databriefs/db07.pdf.
- Congenital Heart Defects. Data and Statistics. Atlanta, GA [updated July 9, 2014; cited 2014 September 2]. Available from: http://www.cdc.gov/ncbddd/heartdefects/data.html.
- Prevalence of Current Depression Among Persons Aged >= 12 Years, by Age Group and Sex United States, National Health and Nutrition Examination Survey, 2007-2010. Morbidity and Mortality Weekly Report (MMWR). 2014;60(51):1747. Available from: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6051a7.htm?s_cid=mm6051a7_w.

DIETARY PATTERNS COMPOSITION

Dietary patterns with positive health benefits are described as high in vegetables, fruit, whole grains, seafood, legumes, and nuts; moderate in low- and non-fat dairy products; lower in red and processed meat; and low in sugar-sweetened foods and beverages and refined grains. The primary dietary patterns examined and described in Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes included both a priori, investigator-derived scoring systems such as DASH/OMNI, Mediterranean diet scores, and the Healthy Eating Index, as well as data-driven approaches using factor/cluster analysis or reduced rank regression. The findings presented come from controlled intervention trials, cohort studies, and nested case-control studies. The DGAC examined these patterns in an attempt to quantify, for the first time, the approximate amounts of each food group in these patterns. The DGAC also examined the range of and commonalities across food group intakes in healthy dietary patterns and compared these ranges to the range of usual adult consumption in the United States and to the range recommended by the USDA Food Patterns.

Question 18: What is the composition of dietary patterns with evidence of positive health outcomes (e.g., Mediterranean-style patterns, Dietary Approaches to Stop Hypertension-style patterns, patterns that closely align with the Healthy Eating Index, and vegetarian patterns), and of patterns commonly consumed in the United States? What are the similarities (and differences) within and among the dietary patterns with evidence of positive health outcomes and the commonly consumed dietary patterns?

Source of evidence: Data analysis

Conclusions

Dietary patterns with varying food group composition, but certain common elements were observed across intervention and cohort studies to have health benefits. A healthful diet can be achieved by following any of these dietary patterns.

In general, the ranges of intake in dietary patterns with positive health benefits are very close to those recommended by the USDA Food Patterns, but amounts of some specific food groups vary across the various diet pattern types.

- DASH-style diets, Mediterranean-style diets, and the USDA Food Patterns are similar with respect to amounts of fruits and vegetables, and the OMNI diets are slightly higher than the USDA Food Patterns.
- Dairy intake is comparable between DASH-style diets and the USDA Food Patterns, but dairy is lower for Mediterranean-style diets than for the USDA Food Patterns.
- Red and processed meats are higher in the Mediterranean-style diets but lower in the DASH-style diet than is recommended by the USDA Food Patterns.
- Seafood intake is similar in DASH-style and higher in Mediterranean-style diets than in the USDA Food Patterns.

The data from the intervention trials and the cohort studies examined provide empirical data that the USDA Food Patterns provide an evidence-based guide to healthy patterns of food consumption.

Implications

The quality of the diets currently consumed by the U.S. population is suboptimal overall and has major adverse health consequences. Several options exist for dietary patterns that can be followed to improve the population's diet quality. The approaches that can be taken are varied and can be adapted to personal and cultural preferences. The ability to offer the U.S. population alternative dietary pattern options and to tailor them to personal preferences may increase the likelihood of long term success of maintaining a healthy diet pattern, ultimately leading to improved health in the U.S. population.

Review of the Evidence

The DGAC analyzed data on food group composition reported in research articles on dietary patterns and health outcomes. These articles were drawn from those included in the questions on dietary patterns and health examined by the Committee (see ***Part D. Chapter 2: Dietary Patterns, Food and Nutrients, and Health Outcomes).*** The studies reported in that chapter D2 were reviewed to identify those that reported semi-quantitative data on food group intakes among the sample or population group with positive health outcomes (Table D1.31).[93-112] These sample or population groups included the intervention group in intervention studies, the highest category (usually the top quintile) in cohorts and nested case-control studies measuring diet with an a priori index, or a specific cluster or factor analysis group. Approximate quantified food group intakes for these subsets of the population or samples with a beneficial health outcome were identified. These intakes were converted to grams per day if not reported this way in the original manuscripts. Then, all data were converted to grams per 1000 calories to allow for comparisons across studies.

For comparison to usual intake levels of each food group in the United States., data from NHANES 2007-2010 for usual intake by adult age/sex groups[41] in cup or ounce equivalents were converted into grams using average weights based on Food Patterns Equivalents Database (FPED) data.[48, 49] The gram weights were divided by the usual calorie intake for that group, and multiplied times 1000 for an estimate of the food group intake per 1000 calories for each adult age/sex group. The range of these intakes was used as a comparator. For comparison to the food group amounts recommended in the USDA Food Patterns (also called the Healthy U.S.-style Patterns; see Question 20) the recommended amount for adult age/sex groups in the patterns at 1600 to 2400 calories were converted to grams per 1000 calories by the same procedure used for the usual intakes (see Figures D1.56 to D1.60).

Vegetable intake in the OMNI diets was higher than both the USDA Food Patterns and current consumption estimates, but DASH-style, PREDIMED, most of the Mediterranean scores, and data driven approaches were very similar to vegetable amounts recommended by the USDA Food Patterns. Fruit intake was higher in the OMNI diets and PREDIMED relative to the USDA Food Patterns and current consumption, but DASH, the Mediterranean score diets, and many of the data driven scores are all within the range of the USDA Food Pattern recommendations. Dairy intakes in OMNI, DASH, and some of the Mediterranean and data driven scores were all within the ranges recommended by the USDA Food Patterns, while PREDIMED and some other scores had lower intakes of dairy. Consumption of red and processed meats was higher in PREDIMED and in some studies using Mediterranean diet scores relative to the USDA Food Patterns, whereas several cohorts using data-driven approaches to assessing diet patterns reported ranges of red and processed meat intake that aligned very well with the USDA Food Pattern recommendations. Intakes of red and processed meat were lower in the OMNI and DASH dietary interventions than in either the USDA Food Patterns or the range of usual intake in the United States. Seafood intakes for the OMNI diets and some of the data-driven dietary pattern studies aligned very well with the USDA Food Patterns. Seafood intake ranges for all the other studies were much higher than both the USDA Food Patterns and the ranges of usual intake in the United States.

For additional details on this body of evidence, visit:

- Usual Dietary Intakes: Food Intakes, U.S. Population, 2007-10: Applied Research Program. National Cancer Institute; [updated May 22, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/.
- Appendix E-3.1: Adequacy of the USDA Food Patterns

Question 19: To what extent does the U.S. population consume a dietary pattern that is similar to those observed to have positive health benefits [e.g., Mediterranean-style pattern, Dietary Approaches to Stop Hypertension (DASH)-style patterns, patterns that closely align with the Healthy Eating Index, and vegetarian patterns] overall and by age/sex and race/ethnic groups?

Source of evidence: Data analysis

Conclusion

Data from WWEIA show that the average HEI score in the U.S. population is 57 points out of a total of 100 points. The best scores (average scores) were observed for the following components: total protein foods (average score of 100 percent of possible points), seafood and plant protein (84 percent of possible points), and dairy (69 percent of possible points), while the poorest scores were observed for whole grains (25 percent of possible points), sodium (37 percent of possible points), fatty acid ratio (41 percent of possible points), greens and beans (46 percent of possible points), and empty calories (60 percent of possible points).

Young children ages 2 to 3 years and middle aged and older adults (ages 51 years and older) have the best HEI scores (total scores of 63 percent and 66 percent, respectively), while preadolescents and adolescents have the poorest HEI scores (total scores of 49 percent and 48 percent, respectively).

Implications

To improve diet quality, the U.S. population should replace most refined grains with whole grains, decrease sodium, decrease saturated fat, consume fewer calories from added sugars, and replace these calories with more varied vegetable choices, seafood, plant proteins, and low-fat dairy.

Young children and middle-aged and older adults have the highest HEI scores. These positive healthy eating habits should continue to be encouraged. Because preadolescents and adolescents have the lowest HEI scores, significant intervention is needed at the level of the individual, family, school, day care, and community settings to help this age group adopt and maintain healthful dietary patterns.

Review of the Evidence

The DGAC examined mean HEI scores and component scores for the entire U.S. population ages 2 years and older (see ***Appendix E-2.25: Average Healthy Eating Index-2010 scores for Americans ages 2 years and older***). These data were examined for the entire population, for males and females and by age subgroups. In general, the best scores for the HEI components were for protein and seafood and plant proteins, while the poorest score was for whole grains. For nearly all of the component scores as well as the total HEI score, females tended to have better scores than males, indicating slightly healthier dietary patterns in females compared to males. Analyses by age showed that the youngest and oldest segments of the population had the best component and total HEI scores (Figure D1.61). For these groups, the component scores were very good to excellent for total fruit and whole fruit. Young children also had excellent scores for dairy, and middle-aged and older adults had excellent scores for total protein and seafood and plant protein. All age groups have poor scores for whole grains.

Data were not available to examine how closely the U.S. population's dietary patterns align with a Mediterranean-style or DASH-style dietary pattern.

For additional details on this body of evidence, visit:

- Healthy Eating Index, Center for Nutrition Policy and Promotion. Available from: http://www.cnpp.usda.gov/HealthyEatingIndex.
- Appendix E-2.25: Average Healthy Eating Index-2010 scores for Americans ages 2 years and older (National Health and Nutrition Examination Survey 2009-2010)

Question 20: Using the Food Pattern Modeling process, can healthy eating patterns for vegetarians and for those who want to follow a Mediterranean-style dietary pattern be developed? How do these patterns differ from the USDA Food Patterns previously updated for potential inclusion in the 2015 DGAs?

Source of evidence: Food Pattern Modeling

Conclusion

Food Pattern Modeling demonstrates that healthy eating patterns can be achieved for a variety of eating styles, including the "Healthy U.S.-style Pattern," the "Healthy Mediterranean-style Pattern," and the "Healthy Vegetarian-style Pattern". Although some differences exist across the three eating patterns, comparable amounts of nutrients can be obtained using nutrient dense foods while maintaining energy balance.

Implications

The U.S. population has a variety of options to help achieve healthful eating patterns that adhere to the Dietary Guidelines. These include the Healthy U.S.-style Pattern, Mediterranean-style Pattern, or Vegetarian Patterns. (Detailed information on these patterns can be found in Table D1.32 and ***Appendix E-3.7: Developing Vegetarian and Mediterranean-style Food Patterns***.) These diets meet nutritional goals without excess calories and use a variety of foods. Importantly, these diets reflect the range of foods that can be used to achieve a healthful eating pattern, and they support the inclusion of diverse foods that are consistent with personal, cultural and religious preferences. These diets can be used in a variety of settings, including homes, schools, worksites, health care facilities, and places of worship.

Review of the Evidence

These conclusions were reached based on the results of the Food Pattern Modeling analysis for vegetarian and Mediterranean-style food patterns. Data from WWEIA from self-reported vegetarians were used to inform the vegetarian eating pattern (Figure D1.62) and data from the Dietary Patterns composition project reviewed above were used to select foods for the Mediterranean-style pattern.[113]

From three dietary patterns ("Healthy U.S.-style," "Healthy Mediterranean-style Patter," and "Healthy Vegetarian Pattern"), selected food group intakes across calorie levels were compared (Table D1.32). Notably, fruit and seafood were higher in the Mediterranean-style diet, while dairy was lower, based on the data presented above (Figures D1.56 to D1.60). For the Vegetarian Pattern, meat and seafood are absent, but eggs and dairy are included because self-reported vegetarians in WWEIA reported consumption of these foods. Legumes, nuts/seeds, and processed soy are all higher in the Vegetarian Pattern compared to the Healthy U.S.-style and the Healthy Mediterranean-style Patterns.

When comparing nutrient intake across these three dietary patterns, as a percent of the RDA using a woman age 19 to 30 years as an example, modest difference emerged (Table D1.33). The Vegetarian pattern is lower in sodium and all three patterns are low in vitamin D.

For additional details on this body of evidence, visit:

- Usual Dietary Intakes: Food Intakes, US Population, 2007-10: Applied Research Program. National Cancer Institute; [updated May 22, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/.
- Appendix E-3.7: Developing Vegetarian and Mediterranean-style Food Patterns

CHAPTER SUMMARY

The DGAC conducted data analyses to address a series of questions related to the current status and trends in the Nation's dietary intake. The questions focused on: intake of specific nutrients and food groups; food categories (i.e., foods as consumed) that contribute to intake; eating behaviors; and the composition of various dietary patterns shown to have health benefits, including Mediterranean-style diets, the Healthy US-style and DASH-style diets. These topics were addressed using data from the WWEIA dietary survey, which is the dietary intake component of the ongoing NHANES. Food pattern modeling using the USDA Food Pattern food groups also was used to address some of the questions of interest. In addition, the DGAC examined the prevalence and trends of health conditions that may have a nutritional origin, or where the course of disease may be influenced by diet.

The DGAC found that several nutrients are underconsumed and the Committee characterized them as shortfall nutrients: vitamin A, vitamin D, vitamin E, vitamin C, folate, calcium, magnesium, fiber, and potassium. For adolescent and premenopausal females, iron also is a shortfall nutrient. Important to note, on the basis of nutrient biomarkers or health outcomes, calcium, vitamin D, fiber, and potassium also are classified as nutrients of public health concern because their underconsumption has been linked in the scientific literature to adverse health outcomes. Iron is included as a shortfall nutrient of public health concern for adolescent females and adult females who are premenopausal due to the increased risk of iron-deficiency in these groups. The DGAC also found that two nutrients—sodium and saturated fat—are overconsumed by the U.S. population and that the overconsumption poses health risks.

The majority of the U.S. population has low intakes of key food groups that are important sources of the shortfall nutrients including vegetables, fruits, whole grains, and dairy. Furthermore, population intake is too high for refined grains and added sugars. The data suggest cautious optimism about dietary intake of the youngest members of the U.S. population because many young children ages 2 to 5 years consume recommended amounts of fruit and dairy. However, a better understanding is needed on how to maintain and encourage the good habits that are started early in life. Analysis of data on food categories, such as burgers, sandwiches, mixed dishes, desserts, and beverages, because they represent such a large proportion of the calories consumed, are prime targets for reformulation to increase population intake of vegetables, whole grains, and other underconsumed food groups and to lower population intake of the nutrients sodium and saturated fat, and the food component refined grains. Dramatically reducing the intake of sugar-sweetened beverages and limiting sweets and desserts would help lower intakes the food component added sugars.

The U.S. population purchases its food in a variety of locations, including supermarkets, convenience stores, schools, and the workplace, and consumes prepared food outside the home. The DGAC found that while diet quality varies somewhat by the setting where food is obtained, overall, independent of where the food is prepared or obtained, the diet quality of the U.S. population does not meet recommendations for fruit, vegetables, dairy, or whole grains, and exceeds recommendations, leading to overconsumption, for the nutrients sodium and saturated fat, and the food components refined grains, solid fats, and added sugars.

Obesity and chronic diseases with a nutritional origin are very common. The Nation must accelerate progress toward reducing the incidence and prevalence of overweight and obesity and chronic disease risk across the U.S. population throughout the lifespan and reduce the disparities in obesity and chronic disease rates that exist in the United States for certain ethnic and racial groups and for those with lower incomes.

The DGAC identified key aspects of several different dietary patterns that are associated with lower risk of many nutrition-related outcomes such as cardiovascular disease, diabetes, some cancers, psychological health and bone health. These patterns and their associated health benefits are described in greater detail in the next chapter.

The DGAC had enough descriptive information from existing research and data to model three dietary patterns and to examine their nutritional adequacy. These patterns are the Healthy U.S.-style Pattern, the Healthy Mediterranean-style Pattern, and the Healthy Vegetarian Pattern. These patterns include the components of a dietary pattern associated with health benefits.

The findings from this chapter and the remainder of the 2015 DGAC report can be used by individuals, families, communities, schools, local, state and federal agencies and the food industry to address the high prevalence of obesity and other nutrition-related health conditions in the United States and help all sectors of the population consume a diet that is healthful, accessible, and affordable.

NEEDS FOR FUTURE RESEARCH

1. Expand WWEIA participation to include more respondents from race/ethnic minorities and non-U.S. born residents.

   Rationale: Very little is known about the dietary habits of many of the cultural subgroups in the United States. This knowledge is essential to moving forward any nutrition programs for first and second generation immigrants. More data on the impact of acculturation also are needed on food and health behaviors. The number of participants in WWEIA using the derived acculturation variable was too small for any analysis. Finally, "Hispanic" is a very broad term and a better understanding is needed of the nutritional profiles (including shortfalls and excesses) across various Spanish-speaking people in the United States, who come from different cultural backgrounds with distinct eating patterns.

2. Include higher proportion of older Americans as respondents in WWEIA.

Rationale: More data are needed on dietary intake of older adults; the sample sizes in WWEIA were too small for any meaningful analyses for those older than the age of 71 years. In addition to nutrient intake, additional information is needed on whether older adults are able to shop and cook, whether polypharmacy plays a role in nutritional adequacy, and whether co-morbidities, such as poor dentition, musculo-skeletal difficulties, arthralgias and other age-related symptoms, affect their ability to establish and maintain proper nutritional status.

3. Increase the number of pregnant women as respondents in WWEIA.

Rationale: The number of pregnant women in WWEIA is currently too small to properly evaluate the status and trends in food and nutrient intake in pregnant women. Since good nutrition in pregnancy is critical to proper growth development of the infant it is critical to properly evaluate food and nutrient intake, which will inform recommendations and public policies for pregnant women.

4. Conduct research on nutrition transitions from childhood to shed light on how and why dietary intake changes so rapidly from early childhood through pre-adolescence and adolescence, and to identify the driving forces behind dietary intake change in these age groups and what programs are most effective at maintaining positive nutrition habits established in very young children.

Rationale: Young children have better dietary intake than older children and adolescents. It is important to maintain the positive gains made in early childhood and identify factors responsible for the declines in intakes of fruit, dairy, and other food groups and increases in added sugars and refined grains as children become enter the elementary school age years, as poor eating patterns in elementary school seem to persist into adolescence and beyond.

5. Evaluate the effects of common variations in dietary patterns in small children on nutrient intakes.

Rationale: Children from 2 to 4 years of age have a highly variable diet and often do not fit readily into the USDA Food Pattern food groups diet pattern analyses. Further information is needed to understand the broad range of diets and supplement use in small children and how this relates to nutrient intake and growth. Research is needed to better characterize their diets so that appropriate guidance can be offered.

6. Increase the quantity and quality of food composition databases available for research.

Rationale: Accurate assessment of nutrient intake and trends over time in the U.S. population is dependent upon the quality of food composition data. Tens of thousands of foods are available for purchase and consumption in the United States, but accurate nutrient content data are available only for less than 10,000 foods and are almost non-existent for many ready-to-eat and restaurant-type foods. Analytic values from foods are needed on specific nutrients and components, such as vitamin D, fiber, added sugars, and sodium. Improved food composition data also is critical for needed research to better define, identify, and quantify total grain, whole grain consumption, and refined grain consumption in dietary studies.

7. Investigate the validity, reliability, and reproducibility of new biomarkers of nutrient intake and biomarkers of nutritional status.

   Rationale: Limited biomarkers are available and some that are available are difficult to interpret due to other contributing factors to the biomarker measure (e.g., vitamin D is obtained in the diet and is also endogenously synthesized).

8. Evaluate effects of fortification strategies and supplement use on consumer behavior related to the intake of foods and supplements containing key nutrients, including calcium, vitamin D, potassium, iron, and fiber

   Rationale: The intake of key nutrients of concern is considerably affected by the rapidly evolving marketplace of food fortification and supplementation. Understanding consumer behavior related to fortification and supplementation would be important in predicting the effects of interventions and marketplace changes in content of these nutrients. Special interest exists regarding fortification strategies of foods, including whole grains and yogurts, in allowing individuals to reach the RDA for vitamin D without using supplements. Data are needed on how supplements may help meet nutrients shortfalls and/or how use of supplements may place individuals at risk of overconsumption. Research on effective consumer guidance is needed.

9. Understand the rationale for and consequences of the use of supplements above the UL for vitamins and minerals. Identify biochemical markers that would indicate the effects of high-dose supplement use.

   Rationale: Consumer use of high-dose supplements has increased. Understanding the influences guiding this use would be helpful in considering how to educate consumers about safe upper intake limits.

10. Develop a standardized research definition for meals and snacks.

    Rationale: Multiple different criteria are used in studies to define a snack or meal occasion, such as time of day, the types or amounts of food consumed, or subjective assessment by the study respondent. Researchers should work toward a consensus on the use of standard definitions.

11. Understand better the concept of dietary patterns and design approaches to quantify the diet in large population-based studies.

    Rationale: More methodological work on dietary patterns is needed. For example, food frequency questionnaires, which are used in most diet assessment studies, do not capture data on meal timing, meal frequency, or the types of foods consumed together. Studies using diet recalls and records are better at capturing specific foods and their quantities consumed (portion sizes) and the types of foods eaten together, but often these detailed assessment methods are not feasible for large population-based studies. Quantification of food group intake is needed. In addition, dietary patterns research encompasses a broader scope of issues than can e addressed by diet scores and data drive approaches.

12. Consistently report the nutrients, foods, and food groups that are used to evaluate dietary patterns in published studies.

    Rationale: The current scientific literature evaluating dietary patterns and health is inconsistent in its provision of dietary patterns composition information. This makes it difficult to compare, across studies, the components of healthful patterns that are associated with health benefits.

13. Conduct population surveillance on the prevalence and trends of nutrition-related chronic diseases including type 2 diabetes, cardiovascular disease, some cancers osteoporosis and neurocognitive disorders.

    Rationale: Current data on diabetes in adults cannot be stratified by disease type (type I or type II), making it very difficult to monitor incidence and prevalence of type 2 diabetes. Continued population surveillance is needed to effectively link nutritional factors with risk of these diseases.

REFERENCES

1. National Research Council. Dietary Reference Intakes: The Essential Guide to Nutrient Requirements Washington, DC: The National Academies Press; 2006. Available from: http://books.nap.edu/openbook.php?record_id=11537.
2. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129(25 Suppl 2):S76-99. PMID: 24222015. http://www.ncbi.nlm.nih.gov/pubmed/24222015.
3. Centers for Disease Control and Prevention. Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population. Atlanta, GA: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 2012. Available from: http://www.cdc.gov/nutritionreport/pdf/Nutrition_Book_complete508_final.pdf.
4. Weaver CM, Dwyer J, Fulgoni VL, 3rd, King JC, Leveille GA, MacDonald RS, et al. Processed foods: contributions to nutrition. Am J Clin Nutr. 2014;99(6):1525-42. PMID: 24760975. http://www.ncbi.nlm.nih.gov/pubmed/24760975.
5. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. What We Eat in America, NHANES dietary survey [updated 10/29/2013]. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=13793.
6. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, et al. Heart disease and stroke statistics--2014 update: a report from the American Heart Association. Circulation. 2014;129(3):e28-e292. PMID: 24352519. http://www.ncbi.nlm.nih.gov/pubmed/24352519.
7. Institute of Medicine. Dietary Reference Intakes: Applications in Dietary Assessment. Washington, DC: The National Academies Press; 2000. Available from: http://www.nap.edu/openbook.php?record_id=9956&page=R1.
8. National Research Council. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc.

Washington, DC: The National Academies Press; 2001. Available from: http://www.nap.edu/openbook.php?record_id=10026.

9. Satia-Abouta J, Patterson RE, Neuhouser ML, Elder J. Dietary acculturation: applications to nutrition research and dietetics. J Am Diet Assoc. 2002;102(8):1105-18. PMID: 12171455. http://www.ncbi.nlm.nih.gov/pubmed/12171455.
10. Abraido-Lanza AF, Chao MT, Florez KR. Do healthy behaviors decline with greater acculturation? Implications for the Latino mortality paradox. Soc Sci Med. 2005;61(6):1243-55. PMID: 15970234. http://www.ncbi.nlm.nih.gov/pubmed/15970234.
11. Redfield R, Linton R, Herskovits M. Memorandum for the study of acculturation. American Anthropologist. 1936;38(1):149-52.
12. Perez-Escamilla R, Putnik P. The role of acculturation in nutrition, lifestyle, and incidence of type 2 diabetes among Latinos. J Nutr. 2007;137(4):860-70. PMID: 17374645. http://www.ncbi.nlm.nih.gov/pubmed/17374645.
13. Neuhouser ML, Thompson B, Coronado GD, Solomon CC. Higher fat intake and lower fruit and vegetables intakes are associated with greater acculturation among Mexicans living in Washington State. J Am Diet Assoc. 2004;104(1):51-7. PMID: 14702584. http://www.ncbi.nlm.nih.gov/pubmed/14702584.
14. Cuellar I, Arnold B, Maldonado R. Acculturation Rating Scale for Mexican Americans-II: A Revision of the Original ARSMA Scale. Hispanic Journal of Behavioral Sciences. 1995;17(3):275-304.
15. Institute of Medicine. Ross AC, Taylor CL, Yaktine AL, Valle HBD, editors. Dietary Reference Intakes for Calcium and Vitamin D. Washington, DC: The National Academies Press; 2011. Available from: http://www.nap.edu/catalog.php?record_id=13050.
16. Schenk JM, Till CA, Tangen CM, Goodman PJ, Song X, Torkko KC, et al. Serum 25-hydroxyvitamin d concentrations and risk of prostate cancer: results from the Prostate Cancer Prevention Trial. Cancer Epidemiol Biomarkers Prev. 2014;23(8):1484-93. PMID: 25085836. http://www.ncbi.nlm.nih.gov/pubmed/25085836.
17. Kristal AR, Till C, Song X, Tangen CM, Goodman PJ, Neuhauser ML, et al. Plasma vitamin D and prostate cancer risk: results from the Selenium and Vitamin E Cancer Prevention Trial. Cancer Epidemiol Biomarkers Prev. 2014;23(8):1494-504. PMID: 24732629. http://www.ncbi.nlm.nih.gov/pubmed/24732629.
18. Cheng TY, Lacroix AZ, Beresford SA, Goodman GE, Thornquist MD, Zheng Y, et al. Vitamin D intake and lung cancer risk in the Women's Health Initiative. Am J Clin Nutr. 2013;98(4):1002-11. PMID: 23966428. http://www.ncbi.nlm.nih.gov/pubmed/23966428.
19. Neuhouser ML, Manson JE, Millen A, Pettinger M, Margolis K, Jacobs ET, et al. The influence of health and lifestyle characteristics on the relation of serum 25-hydroxyvitamin D with risk of colorectal and breast cancer in postmenopausal women. Am J Epidemiol. 2012;175(7):673-84. PMID: 22362582. http://www.ncbi.nlm.nih.gov/pubmed/22362582.
20. Villasenor A, Ballard-Barbash R, Ambs A, Bernstein L, Baumgartner K, Baumgartner R, et al. Associations of serum 25-hydroxyvitamin D with overall and breast cancer-specific mortality in a multiethnic cohort of breast cancer survivors. Cancer Causes Control. 2013;24(4):759-67. PMID: 23361338. http://www.ncbi.nlm.nih.gov/pubmed/23361338.
21. Bjelakovic G, Gluud LL, Nikolova D, Whitfield K, Wetterslev J, Simonetti RG, et al. Vitamin D supplementation for prevention of mortality in adults. Cochrane Database Syst Rev. 2014;1:CD007470. PMID: 24414552. http://www.ncbi.nlm.nih.gov/pubmed/24414552.
22. Abrams SA, Coss-Bu JA, Tiosano D. Vitamin D: effects on childhood health and disease. Nat Rev Endocrinol. 2013;9(3):162-70. PMID: 23381033. http://www.ncbi.nlm.nih.gov/pubmed/23381033.
23. Girgis CM, Clifton-Bligh RJ, Hamrick MW, Holick MF, Gunton JE. The roles of vitamin D in skeletal muscle: form, function, and metabolism. Endocr Rev. 2013;34(1):33-83. PMID: 23169676. http://www.ncbi.nlm.nih.gov/pubmed/23169676.

24. Kalyani RR, Stein B, Valiyil R, Manno R, Maynard JW, Crews DC. Vitamin D treatment for the prevention of falls in older adults: systematic review and meta-analysis. J Am Geriatr Soc. 2010;58(7):1299-310. PMID: 20579169. http://www.ncbi.nlm.nih.gov/pubmed/20579169.
25. Murad MH, Elamin KB, Abu Elnour NO, Elamin MB, Alkatib AA, Fatourechi MM, et al. Clinical review: The effect of vitamin D on falls: a systematic review and meta-analysis. J Clin Endocrinol Metab. 2011;96(10):2997-3006. PMID: 21795448. http://www.ncbi.nlm.nih.gov/pubmed/21795448.
26. Cheng TY, Millen AE, Wactawski-Wende J, Beresford SA, LaCroix AZ, Zheng Y, et al. Vitamin D intake determines vitamin d status of postmenopausal women, particularly those with limited sun exposure. J Nutr. 2014;144(5):681-9. PMID: 24598886. http://www.ncbi.nlm.nih.gov/pubmed/24598886.
27. Abrams SA. Setting Dietary Reference Intakes with the use of bioavailability data: calcium. Am J Clin Nutr. 2010;91(5):1474S-7S. PMID: 20200260. http://www.ncbi.nlm.nih.gov/pubmed/20200260.
28. Taylor CL, Carriquiry AL, Bailey RL, Sempos CT, Yetley EA. Appropriateness of the probability approach with a nutrient status biomarker to assess population inadequacy: a study using vitamin D. Am J Clin Nutr. 2013;97(1):72-8. PMID: 23097269. http://www.ncbi.nlm.nih.gov/pubmed/23097269.
29. U.S. Department of Health and Human Services. Food and Drug Administration. Food Labeling: Revision of the Nutrition and Supplement Facts Labels; Proposed Rule. Federal Register [Internet]. March 3, 2014; 79(41). Available from: http://www.gpo.gov/fdsys/pkg/FR-2014-03-03/pdf/2014-04387.pdf.
30. Golden NH, Abrams SA, Committee on Nutrition. Optimizing bone health in children and adolescents. Pediatrics. 2014;134(4):e1229-43. PMID: 25266429. http://www.ncbi.nlm.nih.gov/pubmed/25266429.
31. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2011;96(7):1911-30. PMID: 21646368. http://www.ncbi.nlm.nih.gov/pubmed/21646368.
32. National Osteoporosis Foundation. Calcium and Vitamin D: What You Need to Know. Available from: http://nof.org/articles/10.
33. National Research Council. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (Macronutrients). Washington, DC: The National Academies Press; 2002. Available from: http://www.nap.edu/openbook.php?record_id=10490.
34. Institute of Medicine. Strom BL, Yaktine AL, Oria M, editors. Sodium Intake in Populations: Assessment of Evidence. Washington, DC: The National Academies Press; 2013. Available from: http://www.nap.edu/openbook.php?record_id=18311.
35. Shin JY, Xun P, Nakamura Y, He K. Egg consumption in relation to risk of cardiovascular disease and diabetes: a systematic review and meta-analysis. Am J Clin Nutr. 2013;98(1):146-59. PMID: 23676423. http://www.ncbi.nlm.nih.gov/pubmed/23676423.
36. Institute of Medicine. Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington, DC: The National Academies Press; 1998. 592 p.
37. WHO recommendations for prevention and treatment of pre-eclampsia and eclampsia: World Health Organization; 2011. Available from: http://whqlibdoc.who.int/publications/2011/9789241548335_eng.pdf.
38. Hathcock JN, Shao A, Vieth R, Heaney R. Risk assessment for vitamin D. Am J Clin Nutr. 2007;85(1):6-18. PMID: 17209171. http://www.ncbi.nlm.nih.gov/pubmed/17209171.
39. Government of Canada. Health Canada Health Products. Food Branch. Food Directorate. Caffeine in Foods [guide]. 2012 [updated 2012-02-16]. Available from: http://www.hc-sc.gc.ca/fn-an/securit/addit/caf/food-caf-aliments-eng.php.
40. Ahluwalia N, Herrick K, Moshfegh A, Rybak M. Caffeine intake in children in the United States and 10-y trends: 2001-2010. Am J Clin Nutr. 2014;100(4):1124-32. PMID: 25240076. http://www.ncbi.nlm.nih.gov/pubmed/25240076.

41. Usual Dietary Intakes: Food Intakes, US Population, 2007-10: Applied Research Program. National Cancer Institute; [updated May 22, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2007-10/.
42. ACOG Committee on Obstetric Practice. ACOG Committee Opinion No. 495: Vitamin D: Screening and supplementation during pregnancy. Obstet Gynecol. 2011;118(1):197-8. PMID: 21691184. http://www.acog.org/-/media/Committee-Opinions/Committee-on-Obstetric-Practice/co495.pdf?dmc=1&ts=20141120T1506445693.
43. LeFevre ML. Screening for Vitamin D Deficiency in Adults: U.S. Preventive Services Task Force Recommendation Statement. Ann Intern Med. 2014. PMID: 25419853. http://annals.org/article.aspx?articleid=1938935.
44. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Updated US Department of Agriculture Food Patterns meet goals of the 2010 dietary guidelines. J Acad Nutr Diet. 2012;112(10):1648-55. PMID: 22853987.
45. Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Impact of typical rather than nutrient-dense food choices in the US Department of Agriculture Food Patterns. J Acad Nutr Diet. 2012;112(10):1560-9. PMID: 22906562. http://www.ncbi.nlm.nih.gov/pubmed/22906562.
46. Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food Guidance System. J Nutr Educ Behav. 2006;38(6 Suppl):S78-92. PMID: 17116598. http://www.ncbi.nlm.nih.gov/pubmed/17116598.
47. Marcoe K, Juan W, Yamini S, Carlson A, Britten P. Development of food group composites and nutrient profiles for the MyPyramid Food Guidance System. J Nutr Educ Behav. 2006;38(6 Suppl):S93-S107. PMID: 17116599. http://www.ncbi.nlm.nih.gov/pubmed/17116599.
48. Bowman SA, Clemens JC, Friday JE, Thoerig RC, Shimizu M, Barrows BR, et al. Food Patterns Equivalents Database 2007-08: Methodology and User Guide. Beltsville, MD: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, U.S. Department of Agriculture,; 2013. Available from: http://www.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/fped/FPED_0708.pdf.
49. Bowman SA, Clemens JC, Thoerig RC, Friday JE, Shimizu M, Moshfegh AJ. Food Patterns Equivalents Database 2009-10: Methodology and User Guide. Beltsville, MD: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, U.S. Department of Agriculture,; 2013. Available from: http://www.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/fped/FPED_0910.pdf.
50. Committee on Nutrition. American Academy of Pediatrics: The use and misuse of fruit juice in pediatrics. Pediatrics. 2001;107(5):1210-3. PMID: 11331711. http://www.ncbi.nlm.nih.gov/pubmed/11331711.
51. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. What We Eat in America, NHANES 2009-10. Table 1a. Fruit: Mean Daily Food Patterns Cup Equivalents Consumed per Individual, by Gender and Age, in the United States, 2009-2010. Available from: http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/fped/Table_1_FPED_GEN_0910.pdf.
52. Dietary Guidelines Advisory Committee. 2010. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, to the Secretary of Agriculture and the Secretary of Health and Human Services Washington, DC: U.S. Department of Agriculture, Agricultural Research Service; 2010.
53. Code of Federal Regulation Title 21, parts 136 & 137: Office of the Federal Register. National Archives and Records Administration.; April 1, 2014. Available from: http://www.gpo.gov/fdsys/pkg/CFR-2014-title21-vol2/pdf/CFR-2014-title21-vol2.pdf.
54. Food and Drug Administration. Food Standards: Amendment of Standards of Identity For Enriched Grain Products to Require Addition of Folic Acid. Federal Register. March 5, 1996;61(44):8781-97. http://www.gpo.gov/fdsys/pkg/FR-1996-03-05/pdf/96-5014.pdf.

55. U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2010. Washington, DC: U.S. Government Printing Office; December 2010 Available from: http://www.health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf.
56. Dietary Guidelines Advisory Committee. 2005. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, to the Secretary of Health and Human Services and the Secretary of Agriculture: U.S. Department of Agriculture, Agricultural Research Service; 2005. Available from: http://www.health.gov/dietaryguidelines/dga2005/report/default.htm.
57. Aune D, Norat T, Romundstad P, Vatten LJ. Dairy products and the risk of type 2 diabetes: a systematic review and dose-response meta-analysis of cohort studies. Am J Clin Nutr. 2013;98(4):1066-83. PMID: 23945722. http://www.ncbi.nlm.nih.gov/pubmed/23945722.
58. Crichton GE, Bryan J, Buckley J, Murphy KJ. Dairy consumption and metabolic syndrome: a systematic review of findings and methodological issues. Obes Rev. 2011;12(5):e190-201. PMID: 21348924. http://www.ncbi.nlm.nih.gov/pubmed/21348924.
59. Labonte ME, Couture P, Richard C, Desroches S, Lamarche B. Impact of dairy products on biomarkers of inflammation: a systematic review of randomized controlled nutritional intervention studies in overweight and obese adults. Am J Clin Nutr. 2013;97(4):706-17. PMID: 23446894. http://www.ncbi.nlm.nih.gov/pubmed/23446894.
60. Soedamah-Muthu SS, Ding EL, Al-Delaimy WK, Hu FB, Engberink MF, Willett WC, et al. Milk and dairy consumption and incidence of cardiovascular diseases and all-cause mortality: dose-response meta-analysis of prospective cohort studies. Am J Clin Nutr. 2011;93(1):158-71. PMID: 21068345. http://www.ncbi.nlm.nih.gov/pubmed/21068345.
61. Tong X, Dong JY, Wu ZW, Li W, Qin LQ. Dairy consumption and risk of type 2 diabetes mellitus: a meta-analysis of cohort studies. Eur J Clin Nutr. 2011;65(9):1027-31. PMID: 21559046. http://www.ncbi.nlm.nih.gov/pubmed/21559046.
62. Wang H, Troy LM, Rogers GT, Fox CS, McKeown NM, Meigs JB, et al. Longitudinal association between dairy consumption and changes of body weight and waist circumference: the Framingham Heart Study. Int J Obes (Lond). 2014;38(2):299-305. PMID: 23736371. http://www.ncbi.nlm.nih.gov/pubmed/23736371.
63. Heaney RP, Dowell MS, Rafferty K, Bierman J. Bioavailability of the calcium in fortified soy imitation milk, with some observations on method. Am J Clin Nutr. 2000;71(5):1166-9. PMID: 10799379. http://www.ncbi.nlm.nih.gov/pubmed/10799379.
64. Usual Dietary Intakes: Food Intakes, US Population, 2001-04: Applied Research Program. National Cancer Institute; [updated April 2, 2014]. Available from: http://appliedresearch.cancer.gov/diet/usualintakes/pop/2001-04/.
65. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. What We Eat in America. Food Categories for the NHANES 2009-10 dietary survey. Available from: http://seprl.ars.usda.gov/Services/docs.htm?docid=23429.
66. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. Meals and Snacks: Distribution of Meal Patterns and Snack Occasions, by Race/Ethnicity and Age. Table 34. What We Eat in America, NHANES 2009-2010. 2012. Available from: http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/0910/Table_34_DMP_RAC_09.pdf.
67. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. Meals and Snacks: Distribution of Meal Patterns and Snack Occasions, by Family Income. Table 36. What We Eat in America, NHANES 2009-2010. Available from: http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/0910/Table_36_DMP_POV_09.pdf.
68. U.S. Department of Agriculture. Agricultural Research Service. Food Surveys Research Group. Snacks: Distribution of Snack Occasions, by Gender and Age. Table 29. What We Eat in America, NHANES 2009-2010. Available from: http://seprl.ars.usda.gov/SP2UserFiles/Place/80400530/pdf/0910/Table_29_DSO_GEN_09.pdf.
69. Institute of Medicine. Wartella EA, Lichtenstein AH, Yaktine A, Nathan R, editors. Front-of-Package Nutrition Rating Systems and Symbols: Promoting Healthier Choices. Washington, DC: The National

Academies Press; 2011. Available from: http://www.nap.edu/catalog/13221/front-of-package-nutrition-rating-systems-and-symbols-promoting-healthier.

70. Lin B-H, Guthrie J. Nutritional Quality of Food Prepared at Home and Away From Home, 1977-2008, EIB-105: U.S. Department of Agriculture, Economic Research Service; December 2012. Available from: http://www.ers.usda.gov/publications/eib-economic-information-bulletin/eib105.aspx.
71. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, et al. Update of the Healthy Eating Index: HEI-2010. J Acad Nutr Diet. 2013;113(4):569-80. PMID: 23415502. http://www.ncbi.nlm.nih.gov/pubmed/23415502.
72. Ford ES, Li C, Zhao G, Tsai J. Trends in obesity and abdominal obesity among adults in the United States from 1999-2008. Int J Obes (Lond). 2011;35(5):736-43. PMID: 20820173. http://www.ncbi.nlm.nih.gov/pubmed/20820173.
73. Ford ES, Maynard LM, Li C. Trends in mean waist circumference and abdominal obesity among US adults, 1999-2012. JAMA. 2014;312(11):1151-3. PMID: 25226482. http://www.ncbi.nlm.nih.gov/pubmed/25226482.
74. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. JAMA. 2014;311(8):806-14. PMID: 24570244. http://www.ncbi.nlm.nih.gov/pubmed/24570244.
75. Ostchega Y, Hughes JP, Terry A, Fakhouri TH, Miller I. Abdominal obesity, body mass index, and hypertension in US adults: NHANES 2007-2010. Am J Hypertens. 2012;25(12):1271-8. PMID: 22895451. http://www.ncbi.nlm.nih.gov/pubmed/22895451.
76. Saydah S, Bullard KM, Cheng Y, Ali MK, Gregg EW, Geiss L, et al. Trends in cardiovascular disease risk factors by obesity level in adults in the United States, NHANES 1999-2010. Obesity (Silver Spring). 2014. PMID: 24733690. http://www.ncbi.nlm.nih.gov/pubmed/24733690.
77. Liu LL, Lawrence JM, Davis C, Liese AD, Pettitt DJ, Pihoker C, et al. Prevalence of overweight and obesity in youth with diabetes in USA: the SEARCH for Diabetes in Youth study. Pediatr Diabetes. 2010;11(1):4-11. PMID: 19473302. http://www.ncbi.nlm.nih.gov/pubmed/19473302.
78. Blackwell D, Lucas J, Clarke T. Summary health statistics for U.S. adults: National Health Interview Survey, 2012. Vital Health Stat 10(260). National Center for Health Statistics; 2014. Available from: http://www.cdc.gov/nchs/data/series/sr_10/sr10_260.pdf.
79. Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. JAMA. 2014;311(17):1778-86. PMID: 24794371. http://www.ncbi.nlm.nih.gov/pubmed/24794371.
80. SEER Stat Fact Sheets: Breast Cancer. Bethesda, MD: National Cancer Institute; [cited 2014 June 15]. Available from: http://seer.cancer.gov/statfacts/html/breast.html.
81. Canfield MA, Mai CT, Wang Y, O'Halloran A, Marengo LK, Olney RS, et al. The association between race/ethnicity and major birth defects in the United States, 1999-2007. Am J Public Health. 2014;104(9):e14-23. PMID: 25033129. http://www.ncbi.nlm.nih.gov/pubmed/25033129.
82. Alzheimer's Association. 2014 Alzheimer's disease facts and figures: includes a special report on women and Alzheimer’s disease. Alzheimers Dement. 2014;10(2):131-68. PMID: 22404854. http://www.alz.org/downloads/facts_figures_2014.pdf.
83. Wright NC, Looker AC, Saag KG, Curtis JR, Delzell ES, Randall S, et al. The Recent Prevalence of Osteoporosis and Low Bone Mass in the United States Based on Bone Mineral Density at the Femoral Neck or Lumbar Spine. J Bone Miner Res. 2014. PMID: 24771492. http://www.ncbi.nlm.nih.gov/pubmed/24771492.
84. Howlader N, Noone A, Krapcho M, Garshell J, Miller D, Altekruse S, et al. SEER Cancer Statistics Review, 1975-2011. Bethesda, MD National Cancer Institute; November 2013 [updated May 12, 2014]. Available from: http://seer.cancer.gov/csr/1975_2011/.
85. SEER Stat Fact Sheets: Prostate Cancer. Bethesda, MD: National Cancer Institute; [cited 2014 June 15]. Available from: http://seer.cancer.gov/statfacts/html/prost.html.
86. SEER Stat Fact Sheets: Colon and Rectum Cancer. Bethesda, MD: National Cancer Institute; [cited 2014 June 15]. Available from: http://seer.cancer.gov/statfacts/html/colorect.html.

87. SEER Stat Fact Sheets: Lung and Bronchus Cancer. Bethesda, MD: National Cancer Institute; [cited 2014 June 15]. Available from: http://seer.cancer.gov/statfacts/html/lungb.html.

88. Centers for Disease Control and Prevention. Facts about Birth Defects [updated October 20, 2014]. Available from: http://www.cdc.gov/ncbddd/birthdefects/facts.html.

89. Centers for Disease Control and Prevention. Division of Birth Defects and Developmental Disabilities. Congenital Heart Defects. Data and Statistics. Atlanta, GA [updated July 9, 2014; cited 2014 September 2]. Available from: http://www.cdc.gov/ncbddd/heartdefects/data.html.

90. Centers for Disease Control and Prevention. QuickStats: Prevalence of Current Depression Among Persons Aged >= 12 Years, by Age Group and Sex United States, National Health and Nutrition Examination Survey, 2007-2010. Morbidity and Mortality Weekly Report (MMWR). 2014;60(51):1747. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6051a7.htm?s_cid=mm6051a7_w.

91. Pratt LA, Brody DJ. Depression in the United States household population, 2005-2006. NCHS Data Brief. 2008(7):1-8. PMID: 19389321. http://www.cdc.gov/nchs/data/databriefs/db07.pdf.

92. Hebert LE, Weuve J, Scherr PA, Evans DA. Alzheimer disease in the United States (2010-2050) estimated using the 2010 census. Neurology. 2013;80(19):1778-83. PMID: 23390181. http://www.ncbi.nlm.nih.gov/pubmed/23390181.

93. Buckland G, Agudo A, Travier N, Huerta JM, Cirera L, Tormo MJ, et al. Adherence to the Mediterranean diet reduces mortality in the Spanish cohort of the European Prospective Investigation into Cancer and Nutrition (EPIC-Spain). Br J Nutr. 2011;106(10):1581-91. PMID: 21736834. http://www.ncbi.nlm.nih.gov/pubmed/21736834.

94. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med. 2013;368(14):1279-90. PMID: 23432189. http://www.ncbi.nlm.nih.gov/pubmed/23432189.

95. Fung TT, Rexrode KM, Mantzoros CS, Manson JE, Willett WC, Hu FB. Mediterranean diet and incidence of and mortality from coronary heart disease and stroke in women. Circulation. 2009;119(8):1093-100. PMID: 19221219. http://www.ncbi.nlm.nih.gov/pubmed/19221219.

96. George SM, Ballard-Barbash R, Manson JE, Reedy J, Shikany JM, Subar AF, et al. Comparing indices of diet quality with chronic disease mortality risk in postmenopausal women in the Women's Health Initiative Observational Study: evidence to inform national dietary guidance. Am J Epidemiol. 2014;180(6):616-25. PMID: 25035143. http://www.ncbi.nlm.nih.gov/pubmed/25035143.

97. Karanja NM, Obarzanek E, Lin PH, McCullough ML, Phillips KM, Swain JF, et al. Descriptive characteristics of the dietary patterns used in the Dietary Approaches to Stop Hypertension Trial. DASH Collaborative Research Group. J Am Diet Assoc. 1999;99(8 Suppl):S19-27. PMID: 10450290. http://www.ncbi.nlm.nih.gov/pubmed/10450290.

98. Martinez-Gonzalez MA, Garcia-Lopez M, Bes-Rastrollo M, Toledo E, Martinez-Lapiscina EH, Delgado-Rodriguez M, et al. Mediterranean diet and the incidence of cardiovascular disease: a Spanish cohort. Nutr Metab Cardiovasc Dis. 2011;21(4):237-44. PMID: 20096543. http://www.ncbi.nlm.nih.gov/pubmed/20096543.

99. Nunez-Cordoba JM, Valencia-Serrano F, Toledo E, Alonso A, Martinez-Gonzalez MA. The Mediterranean diet and incidence of hypertension: the Seguimiento Universidad de Navarra (SUN) Study. Am J Epidemiol. 2009;169(3):339-46. PMID: 19037007. http://www.ncbi.nlm.nih.gov/pubmed/19037007.

100. Romaguera D, Norat T, Mouw T, May AM, Bamia C, Slimani N, et al. Adherence to the Mediterranean diet is associated with lower abdominal adiposity in European men and women. J Nutr. 2009;139(9):1728-37. PMID: 19571036. http://www.ncbi.nlm.nih.gov/pubmed/19571036.

101. Swain JF, McCarron PB, Hamilton EF, Sacks FM, Appel LJ. Characteristics of the diet patterns tested in the optimal macronutrient intake trial to prevent heart disease (OmniHeart): options for a heart-healthy diet. J Am Diet Assoc. 2008;108(2):257-65. PMID: 18237574. http://www.ncbi.nlm.nih.gov/pubmed/18237574.

102. Brunner EJ, Mosdol A, Witte DR, Martikainen P, Stafford M, Shipley MJ, et al. Dietary patterns and 15-y risks of major coronary events, diabetes, and mortality. Am J Clin Nutr. 2008;87(5):1414-21. PMID: 18469266. http://www.ncbi.nlm.nih.gov/pubmed/18469266.
103. Fung TT, Schulze M, Manson JE, Willett WC, Hu FB. Dietary patterns, meat intake, and the risk of type 2 diabetes in women. Arch Intern Med. 2004;164(20):2235-40. PMID: 15534160. http://www.ncbi.nlm.nih.gov/pubmed/15534160.
104. Fung TT, Willett WC, Stampfer MJ, Manson JE, Hu FB. Dietary patterns and the risk of coronary heart disease in women. Arch Intern Med. 2001;161(15):1857-62. PMID: 11493127. http://www.ncbi.nlm.nih.gov/pubmed/11493127.
105. Hu FB, Rimm EB, Stampfer MJ, Ascherio A, Spiegelman D, Willett WC. Prospective study of major dietary patterns and risk of coronary heart disease in men. Am J Clin Nutr. 2000;72(4):912-21. PMID: 11010931. http://www.ncbi.nlm.nih.gov/pubmed/11010931.
106. McCullough ML, Feskanich D, Rimm EB, Giovannucci EL, Ascherio A, Variyam JN, et al. Adherence to the Dietary Guidelines for Americans and risk of major chronic disease in men. Am J Clin Nutr. 2000;72(5):1223-31. PMID: 11063453. http://www.ncbi.nlm.nih.gov/pubmed/11063453.
107. McKeown NM, Meigs JB, Liu S, Wilson PW, Jacques PF. Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham Offspring Study. Am J Clin Nutr. 2002;76(2):390-8. PMID: 12145012. http://www.ncbi.nlm.nih.gov/pubmed/12145012.
108. Villegas R, Yang G, Gao YT, Cai H, Li H, Zheng W, et al. Dietary patterns are associated with lower incidence of type 2 diabetes in middle-aged women: the Shanghai Women's Health Study. Int J Epidemiol. 2010;39(3):889-99. PMID: 20231261. http://www.ncbi.nlm.nih.gov/pubmed/20231261.
109. von Ruesten A, Illner AK, Buijsse B, Heidemann C, Boeing H. Adherence to recommendations of the German food pyramid and risk of chronic diseases: results from the EPIC-Potsdam study. Eur J Clin Nutr. 2010;64(11):1251-9. PMID: 20717136. http://www.ncbi.nlm.nih.gov/pubmed/20717136.
110. Butler LM, Wu AH, Wang R, Koh WP, Yuan JM, Yu MC. A vegetable-fruit-soy dietary pattern protects against breast cancer among postmenopausal Singapore Chinese women. Am J Clin Nutr. 2010;91(4):1013-9. PMID: 20181808. http://www.ncbi.nlm.nih.gov/pubmed/20181808.
111. Samieri C, Sun Q, Townsend MK, Chiuve SE, Okereke OI, Willett WC, et al. The association between dietary patterns at midlife and health in aging: an observational study. Ann Intern Med. 2013;159(9):584-91. PMID: 24189593. http://www.ncbi.nlm.nih.gov/pubmed/24189593.
112. Scarmeas N, Stern Y, Tang MX, Mayeux R, Luchsinger JA. Mediterranean diet and risk for Alzheimer's disease. Ann Neurol. 2006;59(6):912-21. PMID: 16622828. http://www.ncbi.nlm.nih.gov/pubmed/16622828.
113. Juan WY, Yamini S, Britten B. Food intake patterns of self-identified vegetarians among the U.S. population, 2007-2010. 38th Nutrient Data Bank Conference. May 2014. http://www.nutrientdataconf.org/PastConf/NDBC38/NNDC38_PosterAbstracts.pdf.
114. McCullough ML, Feskanich D, Stampfer MJ, Giovannucci EL, Rimm EB, Hu FB, et al. Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. Am J Clin Nutr. 2002;76(6):1261-71. PMID: 12450892. http://www.ncbi.nlm.nih.gov/pubmed/12450892.

Part D. Chapter 1: *Food and Nutrient Intakes, and Health: Current Status and Trends*—Tables

Table Number	Table Title
Nutrients of Concern	
Table D1.1	Mean intake of shortfall and overconsumed nutrients by age and race/ethnicity, for all ages 2+ WWEIA NHANES 2009-10.
Table D1.2	Usual Intakes from Food and Beverages 2007-2010 compared to Dietary Reference Intakes -- females 19-50 years old by pregnancy status. Mean intake and % below EAR, AI, or above UL from food and beverages**,** WWEIA NHANES 2007-10.
Table D1.3.	Mean intake of nutrients of public health concern by income as a % of the poverty threshold, for all ages 2+ WWEIA NHANES 2009-10
Table D1.4	Prevalence (%) of serum 25-hydroxyvitamin D (25(OH)D) concentration levels for the U.S. population aged 1 year and older, NHANES 2003 -2006.
Table D1.5	Vitamin D: Food sources ranked by amounts of vitamin D and energy per standard food portions and per 100 grams of foods
Table D1.6.	Calcium: Food sources ranked by amounts of calcium and energy per standard food portions and per 100 grams of foods
Table D1.7.	Potassium: Food sources ranked by amounts of potassium and energy per standard food portions and per 100 grams of foods
Table D1.8.	Dietary fiber: Food sources ranked by amounts of dietary fiber and energy per standard food portions and per 100 grams of foods
Table D1.9.	Iron: Food sources ranked by amounts of iron and energy per standard food portions and per 100 grams of foods
Food Groups	
Table D1.10.	USDA Food Intake Patterns—recommended daily intake amounts, weekly amounts for Vegetable and Protein Foods subgroups.
Table D1.11.	Energy levels used for assignment of individuals to USDA Food Intake Patterns
Food Categories	
Table D1.12.	Percent of total energy intake from the 32 as-consumed food subcategories, NHANES 2009-10.
Eating Behaviors	
Table D1.13.	Percent of individuals consuming 1, 2, or 3 meals per day, and number of snacks consumed, by age/sex groups, NHANES 2009-10.
Table D1.14.	Percent of individuals skipping specific meals, by age/sex groups, NHANES 2009-10.
Table D1.15.	Meal and snack intake over time--percent reporting consumption of each meal, by age/sex group, NHANES 2005-06 to 2009-10.
Table D1.16.	Percent of energy from each meal and snack occasion over time, by age/sex group, NHANES 2005-06 to 2009-10.
Table D1.17.	Percent of nutrient intake from snacks by age/sex group, NHANES 2009-10.
Table D1.18.	Vegetable density (cups per 1000 calorie) for all vegetable subgroups, by point of purchase, NHANES 2003-04 to 2009-10.
Health Conditions	
Table D1.19	Body mass index (BMI), by sex, age, and race/ethnicity, adults 20 years and older, NHANES 2009 -2012
Table D1.20	Percent of overweight and obesity by income in relation to poverty level, adults 20 years and above
Table D1.21	Trends in prevalence of abdominal obesity among adults, by age, sex and race/ethnicity, NHANES*

Table D1.1 Mean intake of shortfall* and overconsumed nutrients by age and race/ethnicity, for all ages 2+ WWEIA NHANES 2009-10.**

Race/ethnicity and age	n	Vit A* (RAE) µg	Vit D* µg	Vit E* µg	Vit C* mg	Folate* (DFE) µg	Calcium* mg	Magnesium* mg	Iron* mg	Potassium* mg	Dietary fiber* g	Saturated fat** g	Sodium** mg
Ages 2 to 5													
Non-Hispanic White	305	606	6.9	4.8	77.3	405	1081	214	11.2	2070	11.7	21.0	2295
Non-Hispanic Black	150	537	5.8	5.5	86.5	447	879	196	12.6	1956	11.2	19.8	2492
Mexican-American	237	644	7.3	4.3	84.8	450	1057	210	11.8	2141	12.1	19.4	2157
All Hispanic	332	606	7.2	4.4	92.2	439	1031	209	11.5	2144	11.7	18.7	2189
Ages 6 to 11													
Non-Hispanic White	371	618	6.3	5.9	64.9	519	1083	231	13.4	2151	13.6	23.2	2920
Non-Hispanic Black	229	582	5.3	6.2	96.1	526	981	227	14.4	2216	14	23.7	3032
Mexican-American	337	545	6	5.5	78.9	501	970	230	13.9	2175	15.3	22.6	2824
All Hispanic	474	550	5.9	5.5	78.4	518	985	231	13.9	2180	14.7	23.1	2913
Ages 12 to 19													
Non-Hispanic White	425	611	5.9	7.2	67.5	578	1142	262	15.2	2364	14.3	27.7	3584
Non-Hispanic Black	275	502	4.1	7.2	106.7	498	974	234	14.1	2204	13	27.2	3348
Mexican-American	340	518	5	6.7	103.7	538	1074	267	15.4	2431	16.1	25.4	3454
All Hispanic	482	540	5.3	6.9	97.9	565	1081	265	15.7	2411	15.9	25.3	3434
Ages 20 and older													
Non-Hispanic White	2786	682	5.4	8.4	86	559	1070	315	15.6	2868	17.3	26.9	3627
Non-Hispanic Black	1025	555	4.1	6.8	92.4	464	828	261	14.0	2364	13.6	25.2	3358
Mexican-American	1062	537	4.9	6.8	97.8	525	975	320	15.1	2758	20.0	23.7	3368
All Hispanic	1647	525	4.8	6.7	100.9	530	969	307	14.8	2711	18.4	23.6	3417
Ages 2 and older													
Non-Hispanic White	3887	667	5.6	8.0	82.2	551	1079	299	15.2	2728	16.4	26.5	3511
Non-Hispanic Black	1679	549	4.3	6.7	94.3	473	865	251	14.0	2304	13.4	25.0	3273
Mexican-American	1976	545	5.3	6.4	95.2	518	997	291	14.7	2583	18.1	23.4	3206
All Hispanic	2935	537	5.2	6.4	97.1	526	992	284	14.5	2556	17.0	23.3	3252

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors, more nutrients and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.2 Usual Intakes from Food and Beverages compared to Dietary Reference Intakes -- females 19-50 years old by pregnancy status. Mean intake and % below EAR, AI, or above UL from food and beverages, WWEIA NHANES 2007-10.

Nutrient	Pregnancy status**	n	Mean	EAR	% Below EAR	UL	% Above UL
Energy (calorie/day)	Non-pregnant	2957	1848				
	Pregnant	133	2131				
Protein (g/day)	Non-pregnant	2957	69.4				
	Pregnant	133	78.6				
Dietary Fiber (g/day)	Non-pregnant	2957	14.4	25	5		
	Pregnant	133	17.3	28	8*		
Vitamin A (µg RAE/day)	Non-pregnant	2957	549	500	48	3000	<3
	Pregnant	133	728	550	26*	3000	<3
Folate (µg DFE/day)	Non-pregnant	2957	470	320	15	1000	<3
	Pregnant	133	622	520	29*	1000	<3
Vitamin C (mg/day)	Non-pregnant	2957	76.6	60	45	2000	<3
	Pregnant	133	121.0	70	30	2000	<3
Vitamin D (µg/day)	Non-pregnant	2957	3.9	10	>97	100	<3
	Pregnant	133	5.6	10	90*	100	<3
Vitamin E -ATE (mg/day)	Non-pregnant	2957	6.9	12	95		
	Pregnant	133	7.4	12	94*		
Calcium (mg/day)	Non-pregnant	2957	885	800	43	2500	<3
	Pregnant	133	1123	800	24	2500	<3
Iron (mg/day)	Non-pregnant	2957	13.2	8.1	16	45	<3
	Pregnant	133	16.9	22	96*	45	<3
				AI		**UL**	
Potassium (mg/day)	Non-pregnant	2957	2277	4700	<3		
	Pregnant	133	2660	4700	<3		
Sodium (mg/day)	Non-pregnant	2957	3111	1500	>97	2300	84
(overconsumed nutrient)	Pregnant	133	3523	1500	>97	2300	>97

*The values flagged with an asterisk (*) may be less reliable; interpret with caution **Non-pregnant includes non-lactating.

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. For more detailed tables and standard errors, see usual intake tables for pregnant women in ***Appendix E-2.4.***

Table D1.3. Mean intake of nutrients of public health concern by income as a percentage of the poverty threshold, for all ages 2+ WWEIA NHANES 2009-10

Income as % of poverty level and age	n	Dietary fiber g	Vitamin D µg	Calcium mg	Potassium mg
Less than 131% poverty:					
Ages 2-5	431	10.9	6.9	992	2036
Ages 6-11	496	13.9	6.3	1073	2254
Ages 12-19	503	14.1	5.4	1060	2319
Ages 20+	1755	15.5	4.7	942	2564
Ages 2+	3185	14.8	5.2	977	2451
131-185% poverty:					
Ages 2-5	93	12.3	6.8	1090	2160
Ages 6-11	145	12.9	5.8	955	2062
Ages 12-19	162	13.4	3.8	939	2096
Ages 20+	743	15.6	4.7	971	2638
Ages 2+	1143	14.9	4.8	973	2499
Over 185% poverty:					
Ages 2-5	266	12.3	6.8	1057	2070
Ages 6-11	422	14.2	5.9	1052	2134
Ages 12-19	482	14.6	5.8	1126	2417
Ages 20+	2730	17.7	5.3	1053	2866
Ages 2+	3900	16.9	5.5	1061	2735

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors, more nutrients and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.4 Prevalence (%) of serum 25-hydroxyvitamin D (25(OH)D) concentration levels for the U.S. population aged 1 year and older, NHANES 2003 -2006

	Serum 25(OH)D < 30 nmol/L* %(95% conf interval)	Serum 25(OH)D < 40 nmol/L* %(95% conf interval)	Serum 25(OH)D 30 -< 50 nmol/L* %(95% conf interval)	Serum 25(OH)D > 125 nmol/L* %(95% conf interval)
Total, 1 year and older	8.1 (6.7 – 9.8)	17.2 (14.7 – 20.0)	23.6 (21.6 – 25.8)	0.9 (0.6 – 1.2)
Sex				
Male	6.3 (5.0 – 7.9)	14.6 (12.3 – 17.4)	23.1 (20.8 – 25.6)	0.4 (0.3 – 0.7)
Female	9.9 (8.1 – 11.9)	19.6 (16.9 – 22.7)	24.1 (22.1 – 26.3)	1.3 (0.9 – 1.9)
Age category (years)				
1 to 5	0.7 (0.4 – 1.3)	2.7 (1.8 – 4.0)	8.9 (7.1 – 11.0)	§
6 to 11	1.8 (1.3 – 2.6)	5.7 (4.2 – 7.7)	14.1 (11.5 – 17.2)	§
12 to 19	8.5 (6.5 – 11.2)	17.1 (13.8 – 21.0)	24.2 (21.3 – 27.3)	1.4 (0.9 – 2.1)
20 -39	9.5 (7.6 – 11.8)	19.7 (16.4 – 23.4)	26.2 (23.6 – 29.0)	1.5 (0.9 – 2.4)
40 -59	9.3 (7.4 – 11.7)	20.0 (16.6 – 23.9)	25.0 (22.2 – 28.0)	0.6‡ (0.3 – 1.2)
60 +	8.8 (7.3 – 10.5)	17.8 (15.5 – 20.4)	25.5 (23.7 – 27.4)	0.3‡ (0.1 – 0.6)
Race/Ethnicity				
Non-Hispanic Whites	3.6 (3.0 – 4.4)	9.4 (7.9 – 11.2)	18.1 (16.2 – 20.2)	1.2 (0.8 – 1.7)
Non-Hispanic Blacks	31.1 (27.4 – 35.1)	51.6 (46.7 – 56.5)	39.5 (37.3 – 41.7)	§
Mexican Americans	11.3 (8.7 – 14.6)	24.4 (20.1 – 29.3)	32.9 (29.6 – 36.4)	§

1 ng/ml = 2.5 nmol/L

* Serum 25(OH)D < 30 nmol/L = risk for deficiency
Serum 25(OH)D < 40 nmol/L = level set by IOM equal to EAR
Serum 25(OH)D between 30 -50 nmol/L = at risk of inadequacy
Serum 25(OH)D > 125 nmol/L = maybe reason for concern about excess

‡ Estimate flagged: 30% ≤ RSE < 40% for the prevalence estimate

§ Estimate suppressed: RSE ≥ 40% for the prevalence estimate

Source: Centers for Disease Control and Prevention. Second National Report on Biochemical Indicators of Diet and Nutrition in the U.S. Population. Atlanta, GA: Centers for Disease Control and Prevention, U.S. Department of Health and Human Services; 2012. Available from: http://www.cdc.gov/nutritionreport/pdf/Nutrition_Book_complete508_final.pdf.

Table D1.5 Vitamin D: Food sources ranked by amounts of vitamin D and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion[1]	Vitamin D in Standard Portion (µg)[1]	Calories per 100 grams[1]	Vitamin D per 100 grams (µg)[1]
Salmon, sockeye, canned	3 ounces	142	17.9	167	21.0
Trout, rainbow, farmed, cooked	3 ounces	143	16.2	168	19.0
Salmon, chinook, smoked	3 ounces	99	14.5	117	17.1
Swordfish, cooked	3 ounces	146	14.1	172	16.6
Sturgeon, mixed species, smoked	3 ounces	147	13.7	173	16.1
Salmon, pink, canned	3 ounces	117	12.3	138	14.5
Fish oil, cod liver	1 tsp	41	11.3	902	250
Cisco, smoked	3 ounces	150	11.3	177	13.3
Salmon, sockeye, cooked	3 ounces	144	11.1	169	13.1
Salmon, pink, cooked	3 ounces	130	11.1	153	13.0
Sturgeon, mixed species, cooked	3 ounces	115	11.0	135	12.9
Whitefish, mixed species, smoked	3 ounces	92	10.9	108	12.8
Mackerel, Pacific and jack, cooked	3 ounces	171	9.7	201	11.4
Salmon, coho, wild, cooked	3 ounces	118	9.6	139	11.3
Mushrooms, portabella, exposed to UV light, grilled	½ cup	18	7.9	29	13.1
Tuna, light, canned in oil, drained	3 ounces	168	5.7	198	6.7
Halibut, Atlantic and Pacific, cooked	3 ounces	94	4.9	111	5.8
Herring, Atlantic, cooked	3 ounces	173	4.6	203	5.4
Sardine, canned in oil, drained	3 ounces	177	4.1	208	4.8
Rockfish, Pacific, mixed species, cooked	3 ounces	93	3.9	109	4.6
Whole milk[2]	1 cup	149	3.2	61	1.3
Whole chocolate milk[2]	1 cup	208	3.2	83	1.3
Tilapia, cooked	3 ounces	109	3.1	128	3.7
Flatfish (flounder and sole), cooked	3 ounces	73	3.0	86	3.5
Reduced fat chocolate milk (2%)[2]	1 cup	190	3.0	76	1.2
Yogurt (various types and flavors)[2]	8 ounces	98-254	2.0-3.0	43-112	0.9-1.3
Milk (non-fat, 1% and 2%)[2]	1 cup	83-122	2.9	34-50	1.2
Soymilk[2]	1 cup	109	2.9	45	1.2
Low-fat chocolate milk (1%)[2]	1 cup	178	2.8	71	1.1
Fortified ready-to-eat cereals (various)[2]	1/3 -1 ¼ cup	74-247	0.2-2.5	248-443	0.8-8.6
Orange juice, fortified[2]	1 cup	117	2.5	47	1.0
Almond milk (all flavors)[2]	1 cup	91-120	2.4	38-50	1.0
Rice drink[2]	1 cup	113	2.4	47	1.0
Pork, cooked (various cuts)	3 ounces	122-390	0.2-2.2	143-459	0.2-2.6
Mushrooms, morel, raw	½ cup	10	1.7	31	5.1
Margarine (various)[2]	1 Tbsp	75-100	1.5	533-717	10.7
Mushrooms, Chanterelle, raw	½ cup	10	1.4	38	5.3
Egg, hard-boiled	1 large	78	1.1	155	2.2

[1]Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: http://www.ars.usda.gov/nutrientdata .
[2]Vitamin D fortified

Table D1.6. Calcium: Food sources ranked by amounts of calcium and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion[1]	Calcium in Standard Portion (mg)[1]	Calories per 100 grams[1]	Calcium per 100 grams (mg)[1]
Fortified ready-to-eat cereals (various)[2]	¾ -1 ¼ cup	70-197	137-1000	234-394	455-3333
Pasteurized process American cheese	2 ounces	210	593	371	1045
Parmesan cheese, hard	1.5 ounces	167	503	392	1184
Plain yogurt, nonfat	8 ounces	127	452	56	199
Romano cheese	1.5 ounces	165	452	387	1064
Almond milk (all flavors)[2]	1 cup	91-120	451	38-50	188
Pasteurized process Swiss cheese	2 ounces	189	438	334	772
Tofu, raw, regular, prepared with calcium sulfate	½ cup	94	434	76	350
Gruyere cheese	1.5 ounces	176	430	413	1011
Vanilla yogurt, low-fat	8 ounces	193	388	85	171
Plain yogurt, low-fat	8 ounces	143	415	63	183
Pasteurized process American cheese food	2 ounces	187	387	330	682
Fruit yogurt, low-fat	8 ounces	238	383	105	169
Orange juice, calcium fortified[2]	1 cup	117	349	47	140
Soymilk (all flavors)[2]	1 cup	109	340	45	140
Ricotta cheese, part skim	½ cup	171	337	138	272
Swiss cheese	1.5 ounces	162	336	380	791
Evaporated milk	½ cup	170	329	135	261
Sardines, canned in oil, drained	3 ounces	177	325	208	382
Provolone cheese	1.5 ounces	149	321	351	756
Monterey cheese	1.5 ounces	159	317	373	746
Mustard spinach (tendergreen), raw	1 cup	33	315	22	210
Muenster cheese	1.5 ounces	156	305	368	717
Low-fat milk (1%)	1 cup	102	305	42	125
Mozzarella cheese, part-skim	1.5 ounces	128	304	301	716
Skim milk (nonfat)	1 cup	83	299	34	122
Reduced fat milk (2%)	1 cup	122	293	50	120
Colby cheese	1.5 ounces	167	291	394	685
Low-fat chocolate milk (1%)	1 cup	178	290	71	116
Cheddar cheese	1.5 ounces	173	287	406	675
Rice drink[2]	1 cup	113	283	47	118
Whole buttermilk	1 cup	152	282	62	115
Whole chocolate milk	1 cup	208	280	83	112
Whole milk	1 cup	149	276	61	113
Reduced fat chocolate milk (2%)	1 cup	190	273	76	109
Ricotta cheese, whole milk	½ cup	216	257	174	207

[1]Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: http://www.ars.usda.gov/nutrientdata .
[2]Calcium fortified

Table D1.7. Potassium: Food sources ranked by amounts of potassium and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion[1]	Potassium in Standard Portion (mg)[1]	Calories per 100 grams[1]	Potassium per 100 grams (mg)[1]
Potato, baked, flesh and skin	1 medium	163	941	94	544
Prune juice, canned	1 cup	182	707	71	276
Carrot juice, canned	1 cup	94	689	40	292
Passion-fruit juice, yellow or purple	1 cup	126-148	687	51-60	278
Tomato paste, canned	¼ cup	54	669	82	1014
Beet greens, cooked from fresh	½ cup	19	654	27	909
Adzuki beans, cooked	½ cup	147	612	128	532
White beans, canned	½ cup	149	595	114	454
Plain yogurt, nonfat	1 cup	127	579	56	255
Tomato puree	½ cup	48	549	38	439
Sweet potato, baked in skin	1 medium	103	542	90	475
Salmon, Atlantic, wild, cooked	3 ounces	155	534	182	628
Clams, canned	3 ounces	121	534	142	628
Pomegranate juice	1 cup	134	533	54	214
Plain yogurt, low-fat	8 ounces	143	531	63	234
Tomato juice, canned	1 cup	41	527	17	217
Orange juice, fresh	1 cup	112	496	45	200
Soybeans, green, cooked	½ cup	127	485	141	539
Chard, swiss, cooked	½ cup	18	481	20	549
Lima beans, cooked	½ cup	108	478	115	508
Mackerel, various types, cooked	3 ounces	114-171	443-474	134-201	521-558
Vegetable juice, canned	1 cup	48	468	19	185
Chili with beans, canned	½ cup	144	467	112	365
Great northern beans, canned	½ cup	150	460	114	351
Yam, cooked	½ cup	79	456	116	670
Halibut, cooked	3 ounces	94	449	111	528
Tuna, yellowfin, cooked	3 ounces	111	448	130	527
Acorn squash, cooked	½ cup	58	448	56	437
Snapper, cooked	3 ounces	109	444	128	522
Soybeans, mature, cooked	½ cup	149	443	173	515
Tangerine juice, fresh	1 cup	106	440	43	178
Pink beans, cooked	½ cup	126	430	149	508
Chocolate milk (1%, 2% and whole)	1 cup	178-208	418-425	71-83	167-170
Amaranth leaves, cooked	½ cup	14	423	21	641
Banana	1 medium	105	422	89	358
Spinach cooked from fresh or canned	½ cup	21-25	370-419	23	346-466
Black turtle beans, cooked	½ cup	121	401	130	433
Peaches, dried, uncooked	¼ cup	96	399	239	996
Prunes, stewed	½ cup	133	398	107	321
Rockfish, Pacific, cooked	3 ounces	93	397	109	467
Rainbow trout, wild or farmed, cooked	3 ounces	128-143	381-383	150-168	448-450
Skim milk (nonfat)	1 cup	83	382	34	156
Refried beans, canned, traditional	½ cup	106	380	89	319

Table D1.7. Potassium, continued

Food	Standard Portion Size	Calories in Standard Portion[1]	Potassium in Standard Portion (mg)[1]	Calories per 100 grams[1]	Potassium per 100 grams (mg)[1]
Apricots, dried, uncooked	¼ cup	78	378	241	1162
Pinto beans, cooked	½ cup	123	373	143	436
Lentils, cooked	½ cup	115	365	116	369
Avocado	½ cup	120	364	160	485
Tomato sauce, canned	½ cup	30	364	24	297
Plantains, slices, cooked	½ cup	89	358	116	465
Kidney beans, cooked	½ cup	113	357	127	403
Navy beans, cooked	½ cup	128	354	140	389

[1]Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: http://www.ars.usda.gov/nutrientdata .

Table D1.8. Dietary fiber: Food sources ranked by amounts of dietary fiber and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion[1]	Dietary fiber in Standard Portion (g)[1]	Calories per 100 grams[1]	Dietary fiber per 100 grams (g)[1]
High fiber bran ready-to eat-cereal	1/3 – ¾ cup	60-81	9.1-14.3	200-260	29.3-47.5
Navy beans, cooked	½ cup	127	9.6	140	10.5
Small white beans, cooked	½ cup	127	9.3	142	10.4
Yellow beans, cooked	½ cup	127	9.2	144	10.4
Shredded wheat ready-to-eat cereal (various)	1-1 ¼ cup	155-220	5.0-9.0	321-373	9.6-15.0
Cranberry (roman) beans, cooked	½ cup	120	8.9	136	10.0
Adzuki beans, cooked	½ cup	147	8.4	128	7.3
French beans, cooked	½ cup	114	8.3	129	9.4
Split peas, cooked	½ cup	114	8.1	116	8.3
Chickpeas, canned	½ cup	176	8.1	139	6.4
Lentils, cooked	½ cup	115	7.8	116	7.9
Pinto beans, cooked	½ cup	122	7.7	143	9.0
Black turtle beans, cooked	½ cup	120	7.7	130	8.3
Mung beans, cooked	½ cup	106	7.7	105	7.6
Black beans, cooked	½ cup	114	7.5	132	8.7
Artichoke, globe or French, cooked	½ cup	45	7.2	53	8.6
Lima beans, cooked	½ cup	108	6.6	115	7.0
Great northern beans, canned	½ cup	149	6.4	114	4.9
White beans, canned	½ cup	149	6.3	114	4.8
Kidney beans, all types, cooked	½ cup	112	5.7	127	6.4
Pigeon peas, cooked	½ cup	102	5.6	121	6.7
Cowpeas, cooked	½ cup	99	5.6	116	6.5
Wheat bran flakes ready-to-eat cereal (various)	¾ cup	90-98	4.9-5.5	310-328	16.9-18.3
Pear	1 medium	101	5.5	57	3.1
Pumpkin seeds, whole, roasted	1 ounce	126	5.2	446	18.4
Baked beans, canned, plain	½ cup	119	5.2	94	4.1
Soybeans, cooked	½ cup	149	5.2	173	6.0
Plain rye wafer crackers	2 wafers	73	5.0	334	22.9
Avocado	½ cup	120	5.0	160	6.7
Broadbeans (fava beans), cooked	½ cup	94	4.6	110	5.4
Pink beans, cooked	½ cup	126	4.5	149	5.3
Apple, with skin	1 medium	95	4.4	52	2.4
Green peas, cooked (frsh, frzn, cnd)	½ cup	59-67	3.5-4.4	69-84	4.1-5.5
Refried beans, canned	½ cup	107	4.4	90	3.7
Chia seeds, dried	1 Tbsp	58	4.1	486	34.4
Bulgur, cooked	½ cup	76	4.1	83	4.5
Mixed vegetables, cooked from frozen	½ cup	59	4.0	65	4.4
Raspberries	½ cup	32	4.0	52	6.5
Blackberries	½ cup	31	3.8	43	5.3
Collards, cooked	½ cup	32	3.8	33	4.0

Table D1.8. Dietary fiber, continued

Food	Standard Portion Size	Calories in Standard Portion[1]	Dietary fiber in Standard Portion (g)[1]	Calories per 100 grams[1]	Dietary fiber per 100 grams (g)[1]
Soybeans, green, cooked	½ cup	127	3.8	141	4.2
Prunes, stewed	½ cup	133	3.8	107	3.1
Sweet potato, baked in skin	1 medium	103	3.8	90	3.3
Figs, dried	¼ cup	93	3.7	249	9.8
Pumpkin, canned	½ cup	42	3.6	34	2.9
Potato, baked, with skin	1 medium	163	3.6	94	2.1
Popcorn, air-popped	3 cups	93	3.5	387	14.5
Almonds	1 ounce	164	3.5	579	12.5
Pears, dried	¼ cup	118	3.4	262	7.5
Whole wheat spaghetti, cooked	½ cup	87	3.2	124	4.5
Parsnips, cooked	½ cup	55	3.1	71	4.0
Sunflower seed kernels, dry roasted	1 ounce	165	3.1	582	11.1
Orange	1 medium	69	3.1	49	2.2
Banana	1 medium	105	3.1	89	2.6
Guava	1 fruit	37	3.0	68	5.4
Oat bran muffin	1 small	178	3.0	270	4.6
Pearled barley, cooked	½ cup	97	3.0	123	3.8
Winter squash, cooked	½ cup	38	2.9	37	2.8
Dates	¼ cup	104	2.9	282	8.0
Pistachios, dry roasted	1 ounce	161	2.8	567	9.9
Pecans, oil roasted	1 ounce	203	2.7	715	9.5
Hazelnuts or filberts	1 ounce	178	2.7	628	9.7
Peanuts, oil roasted	1 ounce	170	2.7	599	9.4
Whole wheat paratha bread	1 ounce	92	2.7	326	9.6
Quinoa, cooked	½ cup	111	2.6	120	2.8

[1]Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: http://www.ars.usda.gov/nutrientdata .

Table D1.9. Iron: Food sources ranked by amounts of iron and energy per standard food portions and per 100 grams of foods

Food	Standard Portion Size	Calories in Standard Portion[1]	Iron in Standard Portion (mg)[1]	Calories per 100 grams[1]	Iron per 100 grams (mg)[1]
Organ meats (spleen, liver, giblets, heart, kidney or lung) various, cooked	3 ounces	84-235	4.5-33.5	99-277	5.3-39.4
Fortified ready-to-eat cereals (various)	½ -1 ½ cup	89-230	5.1-19.6	310-443	19.4-67.7
Fortified instant cereals (various), prepared	1 cup	174-241	5.1-14.7	62-96	2.1-6.7
Clams, cooked, breaded and fried	3 ounces	172	11.8	202	13.9
Octopus, cooked, moist heat	3 ounces	139	8.1	164	9.5
Coconut milk, canned	1 cup	445	7.5	197	3.3
Tofu, raw, regular, prep. w/ Ca sulfate	½ cup	94	6.6	76	5.4
Oysters, eastern, wild/farmed, cooked, dry heat	3 ounces	67	6.1-6.6	79	7.2-7.8
Oysters, cooked, breaded and fried	3 ounces	169	5.9	199	7.0
Mussels, blue, cooked, moist heat	3 ounces	146	5.7	172	6.7
Liverwurst spread	¼ cup	168	4.9	305	8.9
Soybeans, mature, cooked	½ cup	149	4.4	173	5.1
Chili with beans, canned	½ cup	128	4.4	112	3.4
Beef, plate steak, boneless, outside skirt, all grades, grilled[2]	3 ounces	240-248	4.3-4.4	282-292	5.1-5.2
Mushrooms, morel, raw	½ cup	10	4.0	31	12.2
White beans, canned or cooked	½ cup	125-149	3.3-3.9	114-139	3.0-3.7
Lentils, cooked	½ cup	115	3.3	116	3.3
Spinach, cooked from fresh, frzn or cnd	½ cup	21-32	1.9-3.2	23-34	2.0-3.6
Beef, shoulder pot roast, boneless, 0" fat, all grades, braised[2]	3 ounces	167-173	3.1	196-204	3.5-3.6
Beef, loin, tenderloin steak, boneless, 0" fat, all grades, grilled[2]	3 ounces	168-179	2.7-3.0	198-211	3.2-3.6
Ground beef (95% lean/5% fat), cooked	3 ounces	164	2.8	193	3.2
Black turtle beans, cooked	½ cup	121	2.7	130	2.9
Kidney beans, cooked	½ cup	113	2.6	127	2.9
Sardines, canned in oil, drained	3 ounces	177	2.5	208	2.9
Bagel, enriched	1 sm (3" dia)	182	2.5	264	3.6
Chickpeas, cooked	½ cup	134	2.4	164	2.9
Pumpkin/squash seed kernels, roasted	1 ounce	163	2.3	574	8.1
Adzuki beans, cooked	½ cup	147	2.3	128	2.0
Hearts of palm, canned	½ cup	21	2.3	28	3.1
Yardlong beans, cooked	½ cup	101	2.3	118	2.6
Lima beans, cooked	½ cup	108	2.3	115	2.4
Tomato puree, canned	½ cup	48	2.3	38	1.8
Navy beans, cooked	½ cup	127	2.2	140	2.4
Cowpeas, cooked	½ cup	100	2.2	116	2.5

[1]Source: U.S Department of Agriculture, Agricultural Research Service, Nutrient Data Laboratory. 2014. USDA National Nutrient Database for Standard Reference, Release 27. Available at: http://www.ars.usda.gov/nutrientdata.
[2]Lean and fat or lean only

Table D1.10. USDA Food Intake Patterns (Healthy U.S.-Style Patterns) recommended daily intake amounts, weekly amounts for vegetable and protein foods subgroups.

Energy Level of Pattern*	**1,000**	**1,200**	**1,400**	**1,600**	**1,800**	**2,000**	**2,200**	**2,400**	**2,600**	**2,800**	**3,000**	**3,200**
Food Group												
Fruits	1 c	1 c	1½ c	1½ c	1½ c	2 c	2 c	2 c	2 c	2½ c	2½ c	2½ c
Vegetables	1 c	1½ c	1½ c	2 c	2½ c	2½ c	3 c	3 c	3½ c	3½ c	4 c	4 c
Dark green vegetables (c/wk)	½	1	1	1½	1½	1½	2	2	2½	2½	2½	2½
Red/Orange vegetables (c/wk)	2½	3	3	4	5½	5½	6	6	7	7	7½	7½
Dry beans and peas(c/wk)	½	½	½	1	1½	1½	2	2	2½	2½	3	3
Starchy vegetables (c/wk)	2	3½	3½	4	5	5	6	6	7	7	8	8
Other vegetables (c/wk)	1½	2½	2½	3½	4	4	5	5	5½	5½	7	7
Grains	3 oz eq	4 oz eq	5 oz eq	5 oz eq	6 oz eq	6 oz eq	7 oz eq	8 oz eq	9 oz eq	10 oz eq	10 oz eq	10 oz eq
Whole grains	1½ oz eq	2 oz eq	2½ oz eq	3 oz eq	3 oz eq	3 oz eq	3½ oz eq	4 oz eq	4½ oz eq	5 oz eq	5 oz eq	5 oz eq
Other grains	1½ oz eq	2 oz eq	2½ oz eq	2 oz eq	3 oz eq	3 oz eq	3½ oz eq	4 oz eq	4½ oz eq	5 oz eq	5 oz eq	5 oz eq
Protein Foods	2 oz eq	3 oz eq	4 oz eq	5 oz eq	5 oz eq	5½ oz eq	6 oz eq	6½ oz eq	6½ oz eq	7 oz eq	7 oz eq	7 oz eq
Meat, poultry, eggs (oz/wk)	10	14	19	23	23	26	28	31	31	33	33	33
Seafood (oz/wk)	3	4	6	8	8	8	9	10	10	10	10	10
Nuts seeds, soy (oz/wk)	2	2	3	4	4	5	5	5	5	6	6	6
Dairy	2 c	2.5 c	2.5 c	3 c	3 c	3 c	3 c	3 c	3 c	3 c	3 c	3 c
Oils	15 g	17 g	17 g	22 g	24 g	27 g	29 g	31 g	34 g	36 g	44 g	51g
Limits for:												
Solid fats	10g	7g	7g	8g	11g	18g	18g	23g	25g	26g	31g	40g
Added Sugars	17g	12g	13g	14g	19g	30g	32g	39g	43g	45g	53g	69g

*Food group amounts shown in cup (c) or ounce equivalents (oz eq). Oils, solid fats, and added sugars are shown in grams (g).

Notes continue on next page.

Table D1.10. USDA Food Intake Patterns (Healthy U.S.-Style Patterns), continued

Quantity equivalents for each food group are:

- Grains, 1 ounce equivalent is: ½ cup cooked rice, pasta, or cooked cereal; 1 ounce dry pasta or rice; 1 slice bread; 1 small muffin (1 oz); 1 cup RTE cereal flakes.
- Fruits and vegetables, 1 cup equivalent is: 1 cup raw or cooked fruit or vegetable, 1 cup fruit or vegetable juice, 2 cups leafy salad greens.
- Protein Foods **,** 1 ounce equivalent is: 1 ounce lean meat, poultry, or fish; 1 egg; ¼ cup cooked dry beans or tofu; 1 Tbsp peanut butter; ½ ounce nuts or seeds.
- Milk, 1 cup equivalent is: 1 cup milk or yogurt, 1½ ounces natural cheese such as Cheddar cheese or 2 ounces of processed cheese.

Source: Center for Nutrition Policy and Promotion, USDA. USDA Food Patterns. For more information see Appendix E-3.1: Adequacy of the USDA Food Patterns

Table D1.11. Energy levels used for assignment of individuals to USDA Food Intake Patterns

Males, age	Sedentary[1] Male[s]	Moderately Active[2] Male	Active[3] Male	Females, age	Sedentary[1] Female	Moderately Active[2] Female	Active[3] Female
2	1000	1000	1000	2	1000	1000	1000
3	1000	1400	1400	3	1000	1200	1400
4	1200	1400	1600	4	1200	1400	1400
5	1200	1400	1600	5	1200	1400	1600
6	1400	1600	1800	6	1200	1400	1600
7	1400	1600	1800	7	1200	1600	1800
8	1400	1600	2000	8	1400	1600	1800
9	1600	1800	2000	9	1400	1600	1800
10	1600	1800	2200	10	1400	1800	2000
11	1800	2000	2200	11	1600	1800	2000
12	1800	2200	2400	12	1600	2000	2200
13	2000	2200	2600	13	1600	2000	2200
14	2000	2400	2800	14	1800	2000	2400
15	2200	2600	3000	15	1800	2000	2400
16	2400	2800	3200	16	1800	2000	2400
17	2400	2800	3200	17	1800	2000	2400
18	2400	2800	3200	18	1800	2000	2400
19-20	2600	2800	3000	19-20	2000	2200	2400
21-25	2400	2800	3000	21-25	2000	2200	2400
26-30	2400	2600	3000	26-30	1800	2000	2400
31-35	2400	2600	3000	31-35	1800	2000	2200
36-40	2400	2600	2800	36-40	1800	2000	2200
41-45	2200	2600	2800	41-45	1800	2000	2200
46-50	2200	2400	2800	46-50	1800	2000	2200
51-55	2200	2400	2800	51-55	1600	1800	2200
56-60	2200	2400	2600	56-60	1600	1800	2200
61-65	2000	2400	2600	61-65	1600	1800	2000
66-70	2000	2200	2600	66-70	1600	1800	2000
71-75	2000	2200	2600	71-75	1600	1800	2000
76 and up	2000	2200	2400	76 and up	1600	1800	2000

[1]Sedentary means a lifestyle that includes only the physical activity of independent living.
[2]Moderately Active means a lifestyle that includes physical activity equivalent to walking about 1.5 to 3 miles per day at 3 to 4 miles per hour, in addition to the activities of independent living.
[3]Active means a lifestyle that includes physical activity equivalent to walking more than 3 miles per day at 3 to 4 miles per hour, in addition to the activities of independent living.
Source: Center for Nutrition Policy and Promotion, USDA. USDA Food Patterns. Available at http://www.cnpp.usda.gov/sites/default/files/usda_food_patterns/EstimatedCalorieNeedsPerDayTable.pdf

Table D1.12. Percent of total energy intake from the 32 as-consumed food subcategories,* NHANES 2009-10.

Subcategory	% of total energy consumption	Cumulative %
BURGERS, SANDWICHES, and TACOS	13.8	13.8
DESSERTS and SWEET SNACKS	8.5	22.3
SUGAR-SWEETENED and DIET BEVERAGES	6.5	28.8
RICE, PASTA, GRAIN-BASED MIXED DISHES	5.5	34.3
CHIPS, CRACKERS, and SAVORY SNACKS	4.6	38.9
PIZZA	4.3	43.2
MEAT, POULTRY, SEAFOOD MIXED DISHES	3.9	47.1
VEGETABLES (Incl. Beans and Peas, not Starchy)	3.8	50.9
ALCOHOLIC BEVERAGES	3.8	54.8
STARCHY VEGETABLES	3.8	58.6
YEAST BREADS AND TORTILLAS	3.8	62.4
HIGHER FAT MILK/YOGURT	3.5	65.8
BREAKFAST CEREALS AND BARS	3.5	69.3
POULTRY (Not incl. Deli and Mixed Dishes)	3.3	72.6
CANDY AND SUGARS	3.1	75.6
FRUIT (non-juice)	2.7	78.4
MEATS (Not incl. Deli and Mixed Dishes)	2.1	80.5
LOWFAT MILK/YOGURT	1.9	82.4
QUICK BREADS (Biscuits, Muffins, Pancakes, Waffles)	1.9	84.4
100% FRUIT JUICE	1.8	86.2
NUTS, SEEDS, AND SOY	1.7	87.9
EGGS	1.5	89.4
RICE AND PASTA	1.5	90.8
COFFEE AND TEA	1.4	92.3
SPREADS	1.3	93.6
SOUPS	1.3	95.0
DELI/CURED PRODUCTS (Meat and Poultry)	1.3	96.3
CHEESE	1.3	97.6
SEAFOOD (Not incl. Mixed Dishes)	1.1	98.7
CONDIMENTS AND GRAVIES	0.7	99.4
SALAD DRESSINGS	0.3	99.7
WATERS	0.0	99.7

*Collapsed from the 150 WWEIA Food Categories.

Note: does not total to 100% because baby foods and formulas are not included.

Source: Analysis of What We Eat in America (WWEIA) Food categories for NHANES 2009-10, population ages 2+. (see ***Appendix E-2.9)***

Table D1.13. Percent of individuals consuming 1, 2, or 3 meals per day, and number of snacks consumed, by age/sex groups, NHANES 2009-2010

	3 meals total	3 meals + ≤1 snack	3 meals + 2-3 snacks	3 meals +≥4 snacks	2 meals total	2 meals + ≤1 snack	2 meals + 2-3 snacks	2 meals + ≥4 snacks	1 meal total	1 meal + ≤1 snack	1 meal + 2-3 snacks	1 meal + ≥4 snacks
	%	%	%	%	%	%	%	%	%	%	%	%
Males:												
Ages 2-5	84	9	42	32	16	1	8	7	1	0	0	1
Ages 6-11	73	17	37	19	22	4	10	8	5	1	2	1
Ages 12-19	57	14	27	15	36	8	17	11	8	2	3	2
Ages 20-29	49	10	28	11	39	9	16	14	12	1	6	4
Ages 30-39	59	10	27	22	34	7	17	10	7	2	4	1
Ages 40-49	60	10	32	18	33	4	18	11	6	1	1	4
Ages 50-59	64	11	31	21	31	5	14	13	5	1	3	1
Ages 60-69	72	13	38	21	24	5	13	6	4	0	0	1
Ages 70+	64	18	34	12	32	7	18	7	3	1	1	2
20+	60	12	31	18	33	6	16	11	7	0	1	2
Females:												
Ages 2-5	84	9	38	36	15	1	7	7	1	0	0	0
Ages 6-11	68	15	40	13	30	4	14	12	3	0	1	2
Ages 12-19	49	11	27	10	41	13	19	9	10	1	5	4
Ages 20-29	55	13	23	18	38	8	18	12	7	1	4	3
Ages 30-39	63	9	30	24	34	7	19	7	3	1	1	1
Ages 40-49	64	14	31	20	29	5	12	12	7	1	2	4
Ages 50-59	69	14	28	26	29	4	11	14	3	1	1	1
Ages 60-69	72	8	36	28	26	3	14	9	2	0	1	1
Ages 70+	70	19	32	18	29	7	14	8	1	0	1	0
20+	65	13	30	22	31	6	15	11	4	1	2	2
M/F 2+	63	12	31	20	31	6	15	10	5	1	2	2

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.14. Percent of individuals skipping specific meals, by age/sex groups, NHANES 2009-2010

Age/sex	% skipping breakfast	% skipping lunch	% skipping dinner
Males:			
Ages 2-5	6	7	4
Ages 6-11	13	13	6
Ages 12-19	26	19	7
Ages 20-29	28	23	12
Ages 30-39	19	22	8
Ages 40-49	16	25	6
Ages 50-59	12	23	7
Ages 60-69	9	18	6
Ages 70+	5	28	7
Females:			
Ages 2-5	5	7	5
Ages 6-11	14	16	5
Ages 12-19	25	25	11
Ages 20-29	22	24	7
Ages 30-39	14	17	9
Ages 40-49	13	22	8
Ages 50-59	8	19	8
Ages 60-69	6	18	6
Ages 70+	4	21	6
Males and Females ages 2+	15	20	7

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.15. Meal and snack intake over time—percent reporting consumption of each meal, by age/sex group, NHANES 2005-2006 to 2009-2010

	Breakfast 2005-2006	Breakfast 2007-2008	Breakfast 2009-2010	Lunch 2005-2006	Lunch 2007-2008	Lunch 2009-2010	Dinner 2005-2006	Dinner 2007-2008	Dinner 2009-2010	Snacks 2005-2006	Snacks 2007-2008	Snacks 2009-2010
	%	%	%	%	%	%	%	%	%	%	%	%
Males:												
Ages 2-5	96	94	94	92	91	93	96	96	96	99	98	97
Ages 6-11	91	87	87	88	90	87	97	94	94	98	95	96
Ages 12-19	71	74	74	78	81	81	92	88	93	93	95	92
Ages 20-29	69	72	72	73	82	77	88	91	88	98	94	96
Ages 30-39	82	81	81	85	77	78	90	89	92	95	95	96
Ages 40-49	83	84	84	79	79	75	94	94	94	99	97	97
Ages 50-59	88	88	88	79	80	77	92	91	93	95	98	97
Ages 60-69	91	91	91	74	74	82	95	91	94	94	95	94
Ages 70+	95	95	95	74	70	72	92	94	93	94	93	94
Ages 20+	83	84	84	78	78	77	92	92	92	96	95	96
Females:												
Ages 2-5	97	95	95	91	90	93	95	95	95	96	97	97
Ages 6-11	90	86	86	88	91	84	96	94	95	97	98	98
Ages 12-19	71	75	75	80	82	75	92	89	89	94	95	94
Ages 20-29	74	78	78	79	81	76	89	94	93	94	96	95
Ages 30-39	88	86	86	83	77	83	92	92	91	97	95	97
Ages 40-49	85	87	87	79	82	78	93	94	92	97	98	94
Ages 50-59	92	92	92	81	83	81	94	95	92	98	98	97
Ages 60-69	93	94	94	79	76	82	95	94	94	98	99	97
Ages 70+	96	96	96	79	78	79	93	93	94	93	94	94
Ages 20+	87	88	88	80	80	80	93	94	93	96	97	96
M/F Ages 2+	85	85	85	80	81	80	93	92	93	96	96	96

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2005-06, 2007-08, 2009-2010. For standard errors and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.16. Percent of energy from each meal and snack occasion over time, by age/sex group, NHANES 2005-2006 to 2009-2010

	Breakfast 2005-2006	**Breakfast 2007-2008**	**Breakfast 2009-2010**	**Lunch 2005-2006**	**Lunch 2007-2008**	**Lunch 2009-2010**	**Dinner 2005-2006**	**Dinner 2007-2008**	**Dinner 2009-2010**	**Snacks 2005-2006**	**Snacks 2007-2008**	**Snacks 2009-2010**
	%	%	%	%	%	%	%	%	%	%	%	%
Males:												
Ages 2-5	19	20	20	26	24	26	27	27	26	28	28	28
Ages 6-11	17	19	19	26	27	26	30	29	31	26	25	25
Ages 12-19	14	15	15	26	26	25	35	33	33	26	26	26
Ages 20-29	15	15	15	24	26	25	34	34	34	28	26	26
Ages 30-39	15	15	15	29	25	24	32	35	36	24	22	25
Ages 40-49	15	15	15	22	24	22	39	37	37	24	23	25
Ages 50-59	16	16	16	23	25	22	38	36	37	23	23	25
Ages 60-69	19	19	19	21	21	23	39	37	39	21	24	20
Ages 70+	22	22	22	21	19	20	38	38	39	18	20	19
Ages 20+	16	16	16	24	24	23	36	36	36	24	23	24
Females:												
Ages 2-5	20	19	19	24	23	24	26	26	27	30	29	29
Ages 6-11	19	19	19	26	27	24	31	30	33	24	26	24
Ages 12-19	14	16	16	25	27	25	35	30	33	26	28	26
Ages 20-29	15	16	16	26	25	23	33	36	35	26	25	25
Ages 30-39	17	18	18	26	23	25	34	35	33	23	25	24
Ages 40-49	16	17	17	24	24	23	37	36	35	23	25	24
Ages 50-59	18	18	18	25	24	23	37	37	36	21	23	23
Ages 60-69	19	18	18	22	22	22	39	36	37	20	23	23
Ages 70+	22	21	21	22	24	24	36	37	38	20	19	18
Ages 20+	17	18	18	24	24	23	35	36	35	23	24	23
M/F Ages 2+	17	17	17	25	25	24	35	35	35	24	24	24

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2005-06, 2007-08, 2009-2010. For standard errors and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.17. Percent of nutrient intake from snacks by age/sex group, NHANES 2009-2010

Age/sex	Food energy %	Protein %	Dietary fiber %	Folate %	Vitamin D %	Calcium %	Iron %	Potas-sium %	Sodium* %	Caffeine %	Saturated Fat* %
Males:											
Ages 2-5	28	19	25	18	24	27	18	26	18	36	26
Ages 6-11	25	15	22	17	21	23	18	22	16	41	24
Ages 12-19	26	14	23	17	17	23	18	21	16	60	23
Ages 20-29	26	14	22	21	22	28	20	24	15	48	18
Ages 30-39	25	12	19	17	17	24	17	21	13	45	17
Ages 40-49	25	14	21	19	20	25	17	22	14	48	21
Ages 50-59	25	14	21	18	17	24	17	21	13	43	23
Ages 60-69	20	11	16	13	14	22	13	18	11	37	17
Ages 70+	19	10	16	11	9	19	11	17	9	41	18
Females:											
Ages 2-5	29	21	28	17	29	32	19	29	18	44	30
Ages 6-11	24	14	25	17	14	19	19	20	16	39	23
Ages 12-19	26	16	26	20	19	26	21	24	19	47	24
Ages 20-29	25	14	21	16	18	25	17	22	15	39	23
Ages 30-39	24	13	22	14	16	24	15	22	14	42	20
Ages 40-49	24	14	19	18	17	28	18	22	14	40	24
Ages 50-59	23	13	20	17	15	23	17	20	13	42	22
Ages 60-69	23	14	19	14	16	26	15	21	13	42	24
Ages 70+	18	10	15	11	13	20	11	16	10	35	18

*Overconsumed nutrient

Source: Food Surveys Research Group, Beltsville Human Nutrition Research Center, Agricultural Research Service, USDA. WWEIA Data Tables, NHANES 2009-2010. For standard errors and documentation, see: http://seprl.ars.usda.gov/Services/docs.htm?docid=18349

Table D1.18. Vegetable density (cup equivalents per 1000 calorie) for all vegetable subgroups, by point of purchase, NHANES 2003-2004 to 2009-2010

Point of purchase	2003-2004	2005-2006	2007-2008	2009-2010
DARK GREEN VEGETABLES (cup eq/1000 calorie)				
Store	0.04	0.05	0.05	0.06
Restaurant	0.07	0.08	0.09	0.09
Quick serve restaurant	0.02	0.02	0.03	0.03
School/day care	0.01	0.02	0.01	0.02
Other	0.05	0.08	0.07	0.07
RED AND ORANGE VEGETABLES (cup eq/1000 calorie)				
Store	n/a	0.16	0.16	0.16
Restaurant	n/a	0.23	0.23	0.20
Quick serve restaurant	n/a	0.22	0.17	0.17
School/day care	n/a	0.19	0.17	0.14
Other	n/a	0.23	0.22	0.22
STARCHY VEGETABLES (cup eq/1000 calorie)				
Store	0.20	0.18	0.20	0.19
Restaurant	0.23	0.24	0.26	0.24
Quick serve restaurant	0.24	0.22	0.23	0.23
School/day care	0.16	0.17	0.21	0.12
Other	0.22	0.23	0.25	0.25
OTHER VEGETABLES (cup eq/1000 calorie)				
Store	0.20	0.20	0.20	0.22
Restaurant	0.44	0.42	0.42	0.38
Quick serve restaurant	0.26	0.28	0.23	0.25
School/day care	0.16	0.16	0.13	0.12
Other	0.32	0.33	0.27	0.35

Source: Analysis of food group content, expressed as Food Pattern Equivalents, by point of purchase for What We Eat in America, NHANES 2003-2004, 2005-2006, 2007-2008, 2009-2010, population ages 2+ (see ***Appendix E-2.15).***

Table D1.19. Body mass index (BMI)*, by sex, age, and race/ethnicity, adults ages 20 years and older, NHANES 2009-2012

	Normal weight % (SE)	Overweight % (SE)	Obese % (SE)
All adults ages 20 y and older	29.6 (0.9)	33.3 (0.8)	35.3 (0.8)
Men	26.5 (1.1)	38.1 (0.9)	34.5 (1.1)
Women	32.6 (1.0)	28.8 (1.1)	36.0 (1.0)
Age group (years)			
20-39	36.8 (1.8)	29.5 (1.2)	31.5 (1.3)
40-59	24.5 (1.0)	35.9 (1.2)	38.0 (1.0)
≥60	25.4 (1.1)	35.7 (1.1)	37.5 (1.3)
Race/ethnicity**			
Non-Hispanic White	31.2 (1.2)	33.5 (1.1)	33.4 (1.1)
Non-Hispanic Black	21.7 (0.9)	27.7 (1.1)	48.7 (1.4)
Hispanic	21.0 (1.0)	37.5 (1.2)	40.8 (1.2)
Race/ethnicity by sex			
Men			
Non-Hispanic White	26.7 (1.5)	38.4 (1.1)	34.3 (1.3)
Non-Hispanic Black	28.5 (1.1)	31.7 (1.5)	37.9 (1.5)
Hispanic	19.4 (1.4)	41.5 (1.5)	38.5 (1.5)
Women			
Non-Hispanic White	35.7 (1.4)	28.8 (1.7)	32.5 (1.5)
Non-Hispanic Black	16.2 (1.2)	24.5 (1.4)	57.5 (1.7)
Hispanic	22.7 (1.1)	33.5 (1.4)	43.0 (1.5)

* Normal weight = 18.5 ≤ BMI <25 kg/m^2; Overweight = 25≤ BMI <30 kg/m^2; Obese= BMI = ≥30 kg/m^2

Estimates are age-adjusted to the year 2000 standard population using three age groups: 20–39 years, 40–59 years, and 60 years and over; estimates are weighted; all pregnant women excluded from analysis. SE = standard error.

**Participants with a race-Hispanic origin categorized as “other” are included in overall estimates but are not separately reported.

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Body Mass Index, Adults 20 y and over, NHANES 2009 -2012.

Table D1.20. Percent of overweight and obesity* by income in relation to poverty level, adults ages 20 years and older

Income as % of poverty level	% Overweight 1988-1994	% Obese 1988-1994	% Overweight 1999-2002	% Obese 1999-2002	% Overweight 2003-2006	% Obese 2003-2006	% Overweight 2007-2010	% Obese 2007-2010
Below 100%	31.5	28.1	30	34.7	30.7	35	32.5	37.2
100%-199%	31.9	26.1	33.2	34.1	30.6	35.9	33.2	37.3
200%-399%	33.3	22.7	36.5	32.1	33.3	35.7	31.8	36.8
400% or more	33.7	18.7	36.7	25.5	35.8	28.9	35.6	31.3

*Overweight = 25≤ BMI <30 kg/m^2; Obese= BMI = ≥30 kg/m^2 .

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. U.S. Department of Health and Human Services. Table 74. Healthy weight, overweight, and obesity among persons 20 years of age and over, by selected characteristics: United States, selected years 1960–1962 through 2007–2010. Health, United States, 2011. 2011. Available from: http://www.cdc.gov/nchs/data/hus/2011/074.pdf.

Table D1.21. Trends in prevalence of abdominal obesity[@] among adults, by age, sex, and race/ethnicity, NHANES*

		1999-2000	2001-2002	2003-2004	2005-2006	2007-2008	2009-2010	2011-2012
	Overall	46.4	43.4	52.1	51.6	52.7	52.8	54.2
	Men	37.1	39.1	42.5	44.8	43.4	43	43.5
	Women	55.4	57.1	61.3	58.2	61.6	62.3	64.7
Age group (years)**	**Men**							
	20 - 39	25.3	26.5	28.7	29.9	28.5	NA	NA
	40 - 59	41.8	43.9	49.8	52.7	49.4	NA	NA
	60 +	52.8	55	57.2	60.9	60.4	NA	NA
	Women							
	20 - 39	43.8	45.6	48.5	46.2	51.3	NA	NA
	40 - 59	60.3	59.9	66.7	63.5	65.5	NA	NA
	60 +	69.1	73.5	76.3	72.4	73.8	NA	NA
Race/ethnicity Overall	Non-Hispanic White	45.8	48.4	51.8	51.2	53.3	52.3	53.8
	Non-Hispanic Black	52.4	52.3	57.5	57.1	57.4	60.2	60.9
	Mexican American	48.1	49.9	55	51.4	55.5	58.4	57.4
	Men							
	Non-Hispanic White	38.6	42.4	45.1	46.2	46.6	45.3	44.5
	Non-Hispanic Black	31.5	30.6	35.1	40	38.9	39.5	41.5
	Mexican American	35.8	34.5	38	34.8	41.6	43.4	43.2
	Women							
	Non-Hispanic White	52.9	54.1	57.9	56.3	59.7	59.3	63.3
	Non-Hispanic Black	69.7	70.1	75.7	71	72.3	77.7	75.9
	Mexican American	60.2	66.9	73.8	70.5	71	75.5	71.6

[@]Abdominal obesity, as measured by waist circumference (WC) is defined as WC >102 cm in men and >88 cm in women

*All data from 1999 -2012, except age group –source: Ford ES, Maynard LM, Li C. Trends in mean waist circumference and abdominal obesity among US adults, 1999-2012. JAMA. 2014;312(11):1151-3. PMID: 25226482. http://www.ncbi.nlm.nih.gov/pubmed/25226482.

**Age group data only available from 1999 -2008 – source: Ford ES, Li C, Zhao G, Tsai J. Trends in obesity and abdominal obesity among adults in the United States from 1999-2008. Int J Obes (Lond). 2011;35(5):736-43. PMID: 20820173. http://www.ncbi.nlm.nih.gov/pubmed/20820173.

Age adjustment was performed using the direct method using the projected year 2000 US population aged 20 years or older.

NA = data not available.

Table D1.22. Body mass index (BMI) * among children and adolescents ages 2 to 19 years, NHANES 2009-2012

	Normal weight % (SE)	Overweight % (SE)	Obese % (SE)
Total	64.8 (0.8)	14.9 (0.6)	16.9 (0.6)
Sex			
Boys	63.7 (1.0)	14.9 (0.8)	17.6 (0.9)
Girls	65.9 (1.3)	14.9 (0.8)	16.1 (0.7)
Age group (years)			
2- 5	72.1 (1.5)	14.5 (1.3)	10.2 (0.9)
6-11	62.7 (1.1)	15.5 (0.8)	17.9 (0.9)
12-19	62.7 (1.2)	14.6 (0.8)	19.4 (1.1)
Race/ethnicity**			
Non-Hispanic White	68.2 (1.2)	14.1 (1.0)	14.0 (1.0)
Non-Hispanic Black	60.0 (1.4)	14.9 (0.7)	22.1 (1.2)
Hispanic	58.4 (0.9)	17.2 (0.7)	21.8 (0.6)
Boys			
Non-Hispanic White	66.8 (1.6)	14.5 (1.5)	14.4 (1.5)
Non-Hispanic Black	61.2 (1.8)	13.6 (1.1)	21.9 (1.4)
Hispanic	57.1 (1.3)	16.4 (0.9)	23.7 (1.0)
Girls			
Non-Hispanic White	69.8 (1.9)	13.7 (1.4)	13.6 (1.2)
Non-Hispanic Black	58.7 (2.0)	16.3 (1.3)	22.3 (2.0)
Hispanic	59.7 (1.2)	18.0 (0.9)	19.8 (1.1)

*5th - 84th percentile = normal weight; 85th - 94th percentile = overweight; ≥95th percentile = obese.

**Race-Hispanic origin classified as "other" not separately reported by included in overall estimates. Analyses based on age at the time of exam and exclude pregnant women.

SE = standard error.

Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Body Mass Index Among Children and Adolescents Ages 2 – 19 years, NHANES 2009 -2012.

Table D1.23. Hypertension, lipid profile, and diabetes by body mass index (BMI) and waist circumference, adults ages 20 years and older, NHANES 2009-2012

	Total cholesterol[£] % (SE) ≥ 240 mg/dl	HDL-C[£] % (SE) < 40 mg/dl	LDL-C[∫] % (SE) ≥ 160 mg/dl	Triglycerides[∫] %(SE) ≥ 200 mg/dl	Hypertension*[@ ∂] % (SE)	Diabetes**[Ω] % (SE)
BMI[€]						
Normal weight	12.1 (0.8)	8.5 (0.7)	8 (0.8)	4.8 (0.7)	20.0 (1.1)	5.5 (0.8)
Over weight	15.2 (1)	18.8 (1)	12 (1.2)	12 (0.8)	26.4 (0.8)	9.0 (0.9)
Obese	11.7 (0.6)	30.2 (1.3)	11.2 (0.8)	17.2 (1.6)	39.2 (0.8)	20.3 (1.2)
Waist Circumference (cm)[&]						
Men ≤102, Women ≤ 88	12.1 (0.8)	13.7 (0.8)	8 (0.9)	7.6 (0.8)	21.2 (0.9)	6.0 (0.9)
Men >102, Women >88	13.4 (0.6)	24.9 (1.1)	12.1 (0.9)	14.8 (1.3)	34.6 (0.6)	16.2 (0.9)
BMI, waist circumference (cm) by sex						
Men						
Normal weight	9.7 (1.1)	14.2 (1)	8.3 (1.3)	7 (1.4)	20.1 (1.2)	8.8 (1.6)
Over weight	13.7 (1)	26.8 (1.7)	11 (1.5)	15.6 (1.4)	28.1 (1.3)	10.0 (1.3)
Obese	10.9 (0.9)	42.2 (1.7)	10.2 (1.1)	20.2 (1.9)	39.1 (1.2)	21.6 (1.6)
≤102 cm	12 (1)	20.4 (1.1)	9.3 (0.9)	10.8 (1.2)	23.3 (1)	8.3 (1.2)
>102 cm	11.3 (1)	40.3 (1.6)	11 (1.3)	20.4 (2)	37.2 (1)	19.6 (1.3)
Women						
Normal weight	13.6 (1.1)	4.3 (0.7)	7.7 (0.9)	3.2 (0.7)	19.9 (1.3)	3.2 (0.7)
Over weight	16.7 (1.4)	8.6 (0.9)	12.8 (1.5)	7 (1.1)	24.3 (1)	7.8 (0.8)
Obese	12.3 (0.8)	18.9 (1.4)	11.9 (1.2)	14.2 (1.9)	39.2 (1)	19.2 (1.1)
≤ 88 cm	12.1 (1.1)	3.6 (0.5)	5.9 (1.2)	2.4 (0.6)	17.8 (1.3)	2.6 (0.6)
> 88 cm	14.9 (0.7)	14.9 (1)	12.8 (0.9)	11.2 (1.2)	32.9 (0.7)	13.9 (0.9)

* Adults ages 18 years and older.

[@] Hypertension is defined as having measured systolic pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication. Estimates are based on the average of up to 3 measurements.

**Total diabetes is the sum of self-reported diabetes and undiagnosed diabetes. Diagnosed diabetes was obtained by self-report and excludes women who reported having diabetes only during pregnancy. Undiagnosed diabetes is defined as fasting plasma glucose (FPG) of at least 126 mg/dL or a hemoglobin A1c of at least 6.5% and no reported physician diagnosis. Respondents had fasted for at least 8 hours and less than 24 hours. The definition of undiagnosed diabetes was based on recommendations from the American Diabetes Association. For more information, see Standards of medical care in diabetes – 2010. Diabetes Care 2010: 33 (suppl 1): S11-S61.

Notes continue on next page

Table D1.23, continued

[€]BMI= 18.5-24.9 kg/m^2 = normal weight; BMI =25-29.9 kg/m^2= overweight; BMI = ≥30 kg/m^2= obese.

[&]Abdominal obesity, as measured by waist circumference (WC) is defined as WC >102 cm in men and >88 cm in women

SE = standard error.

Source –
[£]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total cholesterol and high density lipoprotein cholesterol (HDL), adult 20 years and over, NHANES 2009 -2012.
[∫]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Low density lipoprotein cholesterol (LDL-C) and triglycerides, adults 20 years and over, NHANES 2009-2012.
[∂]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Prevalence of high blood pressure, adults 18 years and over, NHANES 2009-2012.
[Ω]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total diabetes, in adults 20 years and over, NHANES 2009 -2012.

Table D1.24. Lipid profile by weight status, among children and adolescents, NHANES 2009-2012

	Total cholesterol*[$£] ***≥ 200 mg/dL*** ***% (SE)***	**HDL-C*[£]** ***< 40 mg/dL*** ***% (SE)***	**LDL-C**[# Ω]** ***≥ 130 mg/dL*** ***% (SE)***	**Triglycerides**[$ Ω]** ***≥ 130 mg/dL*** ***% (SE)***
Body mass index (BMI)				
Normal weight	6.9 (0.7)	7.7 (0.6)	6.7 (1.4)	6.5 (1.2)
Overweight	7.1 (1.2)	16.4 (2.3)	8.0 (2.1)	11.4 (2.7)
Obese	11.3 (1.5)	30.5 (2.5)	6.8 (1.8)	24.1 (3.4)
Weight Status by Sex				
Boys				
Normal weight	5.1 (0.7)	8.8 (1.1)	6.1 (2.0)[@]	5.8 (1.4)
Overweight	5.3 (1.4)	16.9 (3.2)	7.5 (2.7)[@]	11.6 (2.9)
Obese	13.2 (2.4)	35.1 (2.6)	8.8 (3.0)[@]	38.6 (5.0)
Girls				
Normal weight	8.7 (1.1)	6.5 (0.9)	7.3 (1.8)	7.2 (2.5)[@]
Overweight	9.1 (2.1)	15.8 (2.6)	+	11.2 (4.4)[@]
Obese	9.1 (1.9)	25.5 (3.7)	4.6 (1.8)[@]	7.9 (2.4)

Analyses based on age at exam and exclude pregnant adolescents. Estimates are weighted.

[$]Cut-point criteria based on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents .

[*]Data for children and adolescents ages 6 to 19 years old.

[**]Data for children and adolescents ages 12 – 19 years old.

[#]LDL-C calculated using the Friedewald equation (which is valid when triglyceride <400 mg/dL).

Normal weight = 5th-84th percentile; overweight = 85th-94th percentile; obese = ≥95th percentile.

[@]Relative standard error (RSE)≥30 but < 40; + = RSE≥40.

SE = standard error.

Sources:

[£]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total cholesterol, high density lipoprotein cholesterol (HDL), and non-HDL-cholesterol among children and adolescents ages 6 –19 years, NHANES 2009 -2012.

[Ω]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Low density lipoprotein cholesterol (LDL-C) and triglycerides among adolescents ages 12-19 years, NHANES 2009-2012.

Table D1.25. Prevalence of high and borderline high blood pressure (BP) in children, 2009-2012

	High BP* % (SE)	Borderline high BP* % (SE)
Total	1.7 (0.2)	8.3 (0.7)
Boys	1.7 (0.4)	12.0 (1.3)
Girls	1.6 (0.2)	4.6 (0.8)
Age group (years)		
8 - 12	1.8 (0.4)	3.8 (0.7)
13 -17	1.5 (0.4)	12.4 (1.1)
Race/Ethnicity**		
Non-Hispanic White	1.4 (0.3)	7.2 (0.9)
Non-Hispanic Black	2.3 (0.5)	12.1 (1.3)
Hispanic	1.8 (0.6)[@]	8.5 (1.4)
Body Mass Index (BMI)		
Normal weight	1.4 (0.3)	5.4 (0.8)
Overweight	+	10.9 (1.6)
Obese	1.8 (0.6)[@]	16.2 (1.8)
Race/Ethnicity by Sex		
Boys		
Non-Hispanic White	**	10.8 (1.8)
Non-Hispanic Black	2.5 (0.7)	16.6 (2.0)
Hispanic	+	12.7 (2.3)
Girls		
Non-Hispanic White	1.8 (0.4)	3.8 (1.1)
Non-Hispanic Black	+	7.5 (1.6)
Hispanic	1.5 (0.6)[@]	4.3(1.0)
BMI by Sex		
Boys		
Normal weight	1.8 (0.5)	8.6 (1.5)
Overweight	+	16.3 (2.8)
Obese	1.8 (0.6)[@]	20.1 (3.0)
Girls		
Normal weight	1.0 (0.3)	2.4 (0.8)[@]
Overweight	+	5.3 (1.2)
Obese	+	12.0 (2.7)

Analyses based on age at exam and exclude pregnant adolescents. Estimates are weighted. SE = standard error.
*Borderline high BP was defined as a systolic or diastolic BP ≥90th percentile but <95th percentile or BP levels ≥120/80 mm Hg and high BP was defined as a systolic or diastolic BP ≥95th percentile. Definitions are based on the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescent. Estimates are based on the average of up to 3 measurements.
**Race-Hispanic origin classified as "other" not separately reported but included in overall estimates
Normal weight = 5^{th} - 84^{th} percentile; overweight = 85^{th} - 94^{th} percentile; obese = ≥95^{th} percentile
[@] Relative standard error (RSE)≥30 but < 40; + = RSE≥40.
Source: Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Prevalence of high and borderline high blood pressure (BP), children and adolescents, Ages 8-17 years, NHANES 2009-2012.

Table D1.26. Prevalence of overweight and obesity among youth ages 3 to 19* years with type 2 diabetes by race and ethnicity , compared to youth without type 2 diabetes, SEARCH population, 2001-2004

Children ages 3 to 19 years with type 2 diabetes who are:	N	% (95% CI)	Children ages 3 to 19 without diabetes** who are:	% (95% CI)
Overweight €			**Overweight** €	
All	50	10.4 (6.7,15.9)	All	16.1 (15.0,17.3)
Non-Hispanic White	10	13.9 (6.3,28)	Non-Hispanic White	15.9 (14.3,17.6)
Non-Hispanic Black	15	8 (3.2,18.4)	Non-Hispanic Black	14.8 (13.4,16.3)
Hispanic	11	10.5 (4.2,23.8)	Hispanic	18.8 (16.6,21.1)
Asian Pacific Islander	7	14.9 (4.4,39.9)	Asian Pacific Islander	--
American Indian	7	3.3(0.4,20.7)	American Indian	--
Obese &			**Obese** &	
All	331	79.4 (72.8, 84.8)	All	16.9 (15.8,18.0)
Non-Hispanic White	64	68.8 (53.2,81)	Non-Hispanic White	15.8 (14.3,17.5)
Non-Hispanic Black	111	91.1 (81,96.1)	Non-Hispanic Black	20.2 (18.6,21.9)
Hispanic	63	75 (59.8,85.7)	Hispanic	18.3 (16.2,20.5)
Asian Pacific Islander	34	68.2 (43.4,85.7)	Asian Pacific Islander	--
American Indian	59	88 (67.9, 96.2)	American Indian	--

* 93% of children with type 2 diabetes are 12 -19 years old.

** US population estimates based on non-diabetic youth (NHANES 2001–2004).

-- NHANES does not contain large enough samples of Asian Pacific Islander I and American Indian to provide comparable estimates.

€ Overweight defined as BMI from the 85th to <95th percentile for age and sex

& Obesity defined as BMI ≥ 95th percentile.

Source: Liu LL, Lawrence JM, Davis C, Liese AD, Pettitt DJ, Pihoker C, et al. Prevalence of overweight and obesity in youth with diabetes in USA: the SEARCH for Diabetes in Youth study. Pediatr Diabetes. 2010;11(1):4-11. PMID: 19473302. http://www.ncbi.nlm.nih.gov/pubmed/19473302.

Table D1.27. Prevalence of hypertension and diabetes in US adults, NHANES 2009-2012

	Hypertension*[£] % (SE)	Total Diabetes**, [Ω] % (SE)
Overall	29.1 (0.6)	12.3 (0.8)
Men	29.8 (0.8)	14.0 (1.0)
Women	28.3 (0.6)	10.8 (0.8)
Age group (years)		
18-39[&]	7.1 (0.4)	3.2 (0.5)
40-59	31.7 (1.2)	13.5 (1.3)
≥60	66.3 (1.3)	26 (1.7)
Race/ethnicity[@]		
Non-Hispanic white	27.9 (0.7)	9.8 (0.8)
Non-Hispanic black	41.5 (0.9)	18.4 (1.3)
Hispanic	26.1 (0.9)	19.3 (1.5)
Race/ethnicity by sex		
Men		
Non-Hispanic White	28.9 (1.1)	11.7 (1.3)
Non-Hispanic Black	40.5 (1.1)	18.8 (1.8)
Hispanic	26.2 (1.4)	21 (1.7)
Women		
Non-Hispanic White	26.8 (0.8)	8.0 (0.9)
Non-Hispanic Black	42.1 (1.3)	18.1 (1.5)
Hispanic	25.8 (0.8)	17.6 (1.9)

Estimates are age-adjusted to the year 2000 standard population. Estimates are weighted. All pregnant women excluded from analysis.

SE = standard error.

*Hypertension is reported for adults ages 18 yrs and older and is defined as having measured systolic pressure of at least 140 mm Hg or diastolic pressure of at least 90 mm Hg and/or taking antihypertensive medication. Estimates are based on the average of up to 3 measurements.

**Total diabetes is reported for adults ages 20 years and older and is the sum of self-reported diabetes and undiagnosed diabetes. Diagnosed diabetes was obtained by self-report and excludes women who reported having diabetes only during pregnancy. Undiagnosed diabetes is defined as fasting plasma glucose (FPG) of at least 126 mg/dLor a hemoglobin A1c of at least 6.5% and no reported physician diagnosis. Respondents had fasted for at least 8 hours and less than 24 hours.

[&] Data for diabetes is reported for adults ages 20 to 39 years old.

[@]Participants with a race-Hispanic origin categorized as “other” are included in overall estimates but are not separately reported.

Sources:

[£]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Prevalence of high blood pressure, adults 18 years and over, NHANES 2009-2012.

[Ω]Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination survey (NHANES). Total diabetes, in adults 20 years and over, NHANES 2009 -2012

Table D1.28. Prevalence of type 2 diabetes by sex, age, and race/ethnicity in children and adolescents*

	Cases with type 2 diabetes	Prevalence /1000 youth (95% CI)
Overall (< 20 years old)	819	0.46 (0.43 - 0.49)
Sex		
Boys	314	0.35 (0.31 - 0.39)
Girls	505	0.58 (0.53 - 0.63)
Age group (years)		
10 to 14	198	0.23 (0.2 - 0.26)
15 to 19	621	0.68 (0.63 - 0.74)
Race/ethnicity		
Non-Hispanic White	172	0.17 (0.15 -0.2)
Non-Hispanic Black	209	1.06 (0.93 - 1.22)
Hispanic	317	0.79 (0.7 - 0.88)
Asian Pacific Islander	46	0.34 (0.26 - 0.46)
American Indian	75	1.2 (0.96 - 1.51)

*2009 SEARCH population

Source: Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. JAMA. 2014;311(17):1778-86. PMID: 24794371. http://www.ncbi.nlm.nih.gov/pubmed/24794371.

Table D1.29. Cancer incidence and death rates per 100,000 persons by age category, sex and race and ethnicity, United States, 2007 -2011*

Rates per 100,000 persons	Incidence Breast	Death Breast	Incidence Prostate	Death Prostate	Incidence Colorectal	Death Colorectal	Incidence Lung & Bronchus	Death Lung & Bronchus
Age (years), men and women								
<20	0	0	0	0	0.1	0	0	0
20-34	1.8	0.9	0	0	1.2	0.6	0.3	0.1
35-44	9.3	5.2	0.6	0.1	4.1	2.5	1.3	1
45-54	22	14.5	9.7	1.6	14.2	9.1	8.6	7.7
55-64	25.5	21.7	32.7	8.5	21.2	17.6	21.4	19.7
65-74	21.3	20.6	36.3	20.1	23.9	21.9	31.7	30.6
75-84	14.4	21	16.8	36.8	23.2	27.3	27.9	29.8
>84	5.7	16.2	3.8	33	12.1	20.9	8.9	11.2
Men								
all race/ethnicities	–	–	147.8	22.3	50.6	19.1	72.2	61.6
Non-Hispanic White	–	–	139.9	20.6	49.6	18.5	72.4	61.4
Non-Hispanic Black	–	–	223.9	48.9	62.3	27.7	93	75.7
Hispanic	–	–	121.8	18.5	44.3	15.8	39.6	30.5
Asian/Pacific Islander	–	–	79.3	10	43.1	13.1	49.4	34.7
American Indian/Alaska Native	–	–	71.5	21.2	45.5	19.2	49.5	50
Women								
all race/ethnicities	124.6	22.2	–	–	38.2	13.5	51.1	38.5
Non-Hispanic White	128	21.7	–	–	37.3	13	53.8	39.8
Non-Hispanic Black	122.8	30.6	–	–	47.5	18.5	51.2	36.5
Hispanic	91.3	14.5	–	–	30.6	9.9	25.5	14
Asian/Pacific Isldander	93.6	11.3	–	–	32	9.5	28.1	18.4
American Indian/Alaska Native	79.3	15.2	–	–	35.5	15.6	34.7	32.4

*SEER 18, 2007 -2011; rates (numbers) of new cases and deaths are per 100,000 persons and are age-adjusted to the 2000 U.S. standard population. Data are from selected statewide and metropolitan area cancer registries that meet the data quality criteria for all invasive cancer sites combined. Rates cover approximately 95% of the U.S. population.

Source: Data are from NCI factsheets, and can be found in the SEER Cancer Statistics Review (http://seer.cancer.gov/csr/1975_2011/)
Breast cancer - http://seer.cancer.gov/statfacts/html/breast.html , Prostate Cancer - http://seer.cancer.gov/statfacts/html/prost.html ,
Colon and Rectum Cancer - http://seer.cancer.gov/statfacts/html/colorect.html ,Lung and Bronchus Cancer - http://seer.cancer.gov/statfacts/html/lungb.html

Table D1.30. Estimates of the prevalence and number of US adults ages 50 years and older with osteoporosis (OP) and low bone mass (LBM) at either the femoral neck or lumber spine (NHANES 2005-2010)

	OP Prevalence * % (SE)	OP N (95% CI)**	BM Prevalence * % (SE)	LBM, N (95% CI)**
Both Sexes				
Overall (ages 50 above)	10.3 (0.37)	10.2 (9.4,10.9)	43.9 (0.72)	43.4 (42.0,44.8)
Men				
Overall	4.3 (0.40)	2.0 (1.6,2.3)	35.2 (0.93)	16.1 (15.3,17.0)
Age group (years)				
50-59	3.4 (0.68)	0.7 (0.4,1.0)	30.7 (1.78)	6.3 (5.6,7.0)
60-69	3.3 (0.73)	0.5 (0.3,0.7)	32.9 (1.82)	4.6 (4.1,5.1)
70-79	5.0 (0.78)	0.4 (0.3,0.5)	41.8 (2.51)	3.1 (2.7,3.5)
80+	10.9 (1.7)	0.4 (0.3,0.6)	53.1 (2.82)	2.2 (1.9,2.4)
Race/ethnicity [@]				
Non-Hispanic White	3.9 (0.39)	1.4 (1.1,1.6)	36.0 (1.13)	12.7 (11.9,13.4)
Non-Hispanic Black	1.3* (0.40)	0.1 (0.02,0.1)	21.3 (1.75)	0.9 (0.8,1.1)
Mexican American	5.9 (1.08)	0.1 (0.1,0.2)	38.3 (2.55)	0.9 (0.7,1.0)
Women				
Overall	15.4 (0.63)	8.2 (7.5,8.9)	51.4 (0.93)	27.3 (26.3,28.3)
Age group (years)				
50-59	6.8 (0.83)	1.5 (1.1,1.8)	49.3 (1.69)	10.6 (9.9,11.3)
60-69	12.3 (1.44)	1.9 (1.5,2.3)	53.4 (1.54)	8.2 (7.7,8.6)
70-79	25.7 (1.56)	2.4 (2.1,2.6)	51.8 (1.70)	4.7 (4.4,5.1)
80+	34.9 (2.44)	2.5 (2.2,2.8)	52.7 (3.07)	3.8 (3.3,4.2)
Race/ethnicity [c]				
Non-Hispanic White	15.8 (0.81)	6.3 (5.7,7.0)	52.6 (1.17)	21.1 (20.2,22.0)
Non-Hispanic Black	7.7 (1.10)	0.4 (0.3,0.5)	36.2 (2.03)	2.0 (1.8,2.2)
Mexican American	20.4 (1.70)	0.5 (0.4,0.6)	47.8 (2.33)	1.1 (1.0,1.2)

* Prevalence from NHANES 2005-2010 has been adjusted to the age, sex, and race/ethnic distribution of the US population at the time of the 2010 Census using the direct method.
**Count expressed in millions; 95% CI=95% confidence limits
[@] Other races not shown separately
OP = osteoporosis; LBM= low bone mass; NH= non-Hispanic. SE = standard error.
Osteoporosis and low bone mass were defined using the WHO criteria. Specifically, osteoporosis was defined as a T-score ≤ -2.5 at either the femoral neck or the lumbar spine. Among those without osteoporosis, low bone mass was defined as those with T-scores between -1.0 and -2.5 at either skeletal site. The reference group for calculation of the scores at the femoral neck for both men and women, consisted of 20-29 non-Hispanic White females from NHANES III. As there is no internationally recommended reference group for the lumbar spine, the reference group for calculation of these scores at the lumbar spine consisted of 30-year old White females from the DXA manufacturer reference database. These reference groups were used to calculate T-scores for all race/ethnic groups and for both sexes.
Source: Wright NC, Looker AC, Saag KG, Curtis JR, Delzell ES, Randall S, et al. The Recent Prevalence of Osteoporosis and Low Bone Mass in the United States Based on Bone Mineral Density at the Femoral Neck or Lumbar Spine. J Bone Miner Res. 2014. PMID: 24771492. http://www.ncbi.nlm.nih.gov/pubmed/24771492.

Table D1.31 Studies included in the analysis of Dietary Patterns Composition. Abbreviations listed below are used in Figures D1.56 to D1.60

Abbreviation Used in Figures	Study/Cohort	Citation
Interventions—feeding studies		
DASH	DASH – Dietary Approaches to Stop Hypertension Trial	Karanja et al. 1999[97]
OMNI CHO	OmniHeart trial – Carbohydrate-rich pattern	Swain et al. 2008[101]
OMNI PRO	OmniHeart trial – higher-protein pattern	
OMNI UNSAT	OmniHeart trial – higher unsaturated fat pattern	
Interventions—other		
EVOO	PREDIMED (Prevención con Dieta Mediterránea) trial. Extra Virgin Olive Oil group	Estruch et al. 2013[94]
NUTS	PREDIMED Mixed nuts group	
Cohorts--Med Diet score		
SUN F (CVD endpoint)	Seguimiento Universidad de Navarra (SUN) project. Female subjects	Martínez-González et al. 2010[98]
SUN M (CVD endpoint)	SUN project. Male subjects	
SUN (blood pressure endpoint)	Seguimiento Universidad de Navarra (SUN) project	Núñez-Córdoba et al.. 2009[99]
NHS (CVD endpoint)	Nurses' Health Study	Fung et al. 2009[95]
EPIC PAN F	European Prospective Investigation into Cancer and Nutrition – Physical Activity, Nutrition, Alcohol, Cessation of Smoking, Eating Out of Home and Obesity project (EPIC-PANACEA) Female subjects	Romaguera et al. 2009[100]
EPIC PAN M	EPIC-PANACEA Male subjects	
EPIC SPAIN	EPIC Spanish Cohort	Buckland et al. 2011[93]
WAICAP	Washington Heights-Inwood Columbia Aging Project (WHICAP)	Scarmeas et al. 2006[112]
NHS (cognitive decline endpoint)	Nurses' Health Study	Samieri et al. 2013[111]
Cohorts/Other scores		
WHI	Women's Health Initiative	George et al. 2014[96]
HPFS	Health Professionals Follow-up Study	McCullough et al. 2000[114]
EPIC POT F	EPIC Potsdam (Germany) study Female Subjects	von Ruesten et al. 2010[109]
EPIC POT M	EPIC Potsdam (Germany) study Male Subjects	

Table D1.31, continued

Abbreviation Used in Figures	Study/Cohort	Citation
Factor/Cluster Analyses		
NHS (type 2 diabetes endpoint)	Nurses' Health Study	Fung et al. 2004[103]
NHS (CHD endpoint)	Nurses' Health Study	Fung et al. 2001[104]
HPFS	Health Professionals Follow-up Study	Hu et al. 2000[105]
FOS	Framingham Offspring Study	McKeown et al. 2002[107]
WHITEHALL	Whitehall II study	Brunner et al. 2008[102]
SHANGHAI	Shanghai Women's Health Study	Villegas et al. 2010[108]
SINGAPORE	Singapore Chinese Health Study	Butler 2010[110]

Table D1.32. Composition of three USDA Food Patterns (Healthy U.S.-Style, Healthy Vegetarian, and Healthy Mediterranean-style) at the 2000 calorie level. Daily or weekly amounts from selected food groups, subgroups, and components.

Food group	Healthy US-style Pattern	Healthy Vegetarian Pattern	Healthy Med-style Pattern
Fruit	2 c per day	2 c per day	2 ½ c per day
Vegetables	2 ½ c per day	2 ½ c per day	2 ½ c per day
-Legumes	1 ½ c per wk	3 c per wk	1 ½ c per wk
Whole Grains	3 oz eq per day	3 oz eq per day	3 oz eq per day
Dairy	3 c per day	3 c per day	2 c per day
Protein Foods	5 ½ oz eq per day	3 ½ oz eq per day	6 ½ oz eq per day
--Meat	12 ½ oz eq/wk	--	12 ½ oz eq/wk
--Poultry	10 ½ oz eq/wk	--	10 ½ oz eq/wk
--Seafood	8 oz eq/wk	--	15 oz eq/wk
--Eggs	3 oz eq/wk	3 oz eq/wk	3 oz eq/wk
--Nuts/seeds	4 oz eq/wk	7 oz eq/wk	4 oz eq/wk
--Processed soy	½ oz eq/wk	8 oz eq/wk	½ oz eq/wk
Oils	27 g per day	27 g per day	27 g per day

Source: Food Pattern Modeling report: ***Appendix E-3.7 Developing Vegetarian and Mediterranean-style Food Patterns***

Table D1.33. Nutrients in the three USDA Food Patterns (Healthy US Style, Healthy Vegetarian, and Healthy Mediterranean-style) at the 2000 calorie level as a percent of the goal or limit for a 19 to 30 year old woman.

Nutrient	Healthy US-style Pattern % goal/limit	Healthy Vegetarian Pattern % goal/limit	Healthy Med-style Pattern % goal/limit
Protein -%RDA	198	155	194
Protein -%calorie	18	14	18
Fat-%calorie	33	34	32
Saturated fat* - %calorie	8	8	8
CHO-%RDA	197	211	199
CHO-%calorie	51	55	52
Fiber -% goal	`109	126	112
Calcium-%RDA	127	133	100
Iron-%RDA	93	96	95
Vitamin D-%RDA	46	37	42
Potassium-%AI	71	70	71
Sodium*-%UL	78	61	73

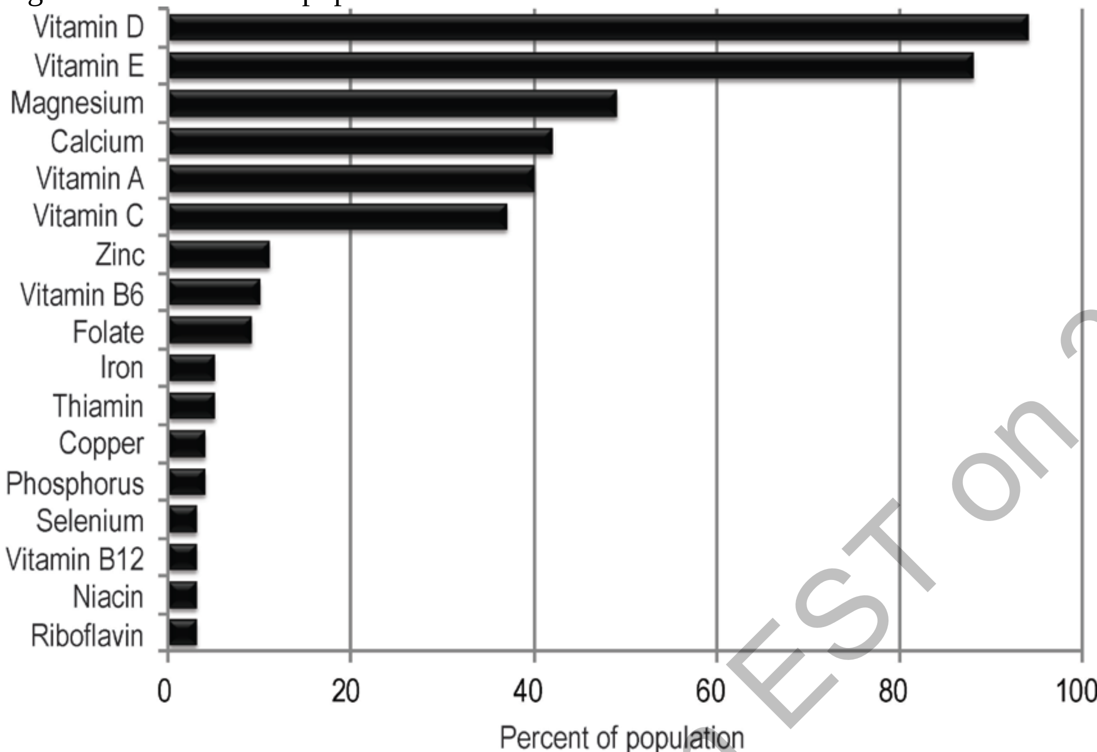
*overconsumed nutrient

Source: Food Pattern Modeling report: Developing Vegetarian and Mediterranean-style Food Patterns (see ***Appendix E-3.7)***

Part D Chapter 1. Figures

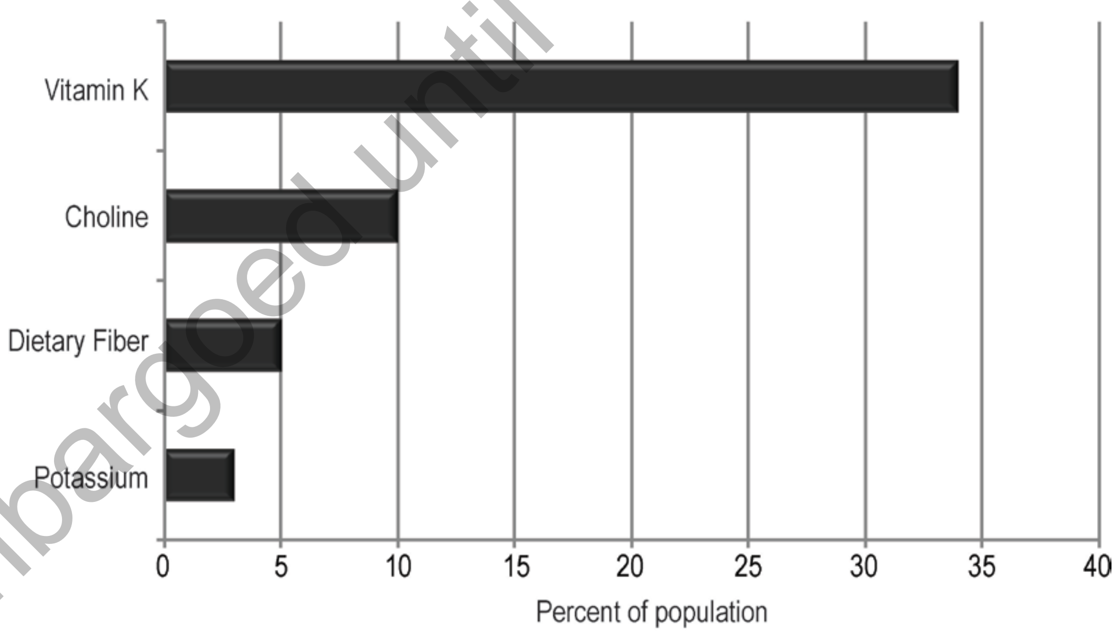
Figure D1.62	Intake from Protein Foods subgroups by self-identified vegetarians in comparison to non-vegetarian and amounts in USDA Food Pattern at 2000 calories.

Figure D1.1 Percent of population with usual intakes below EAR



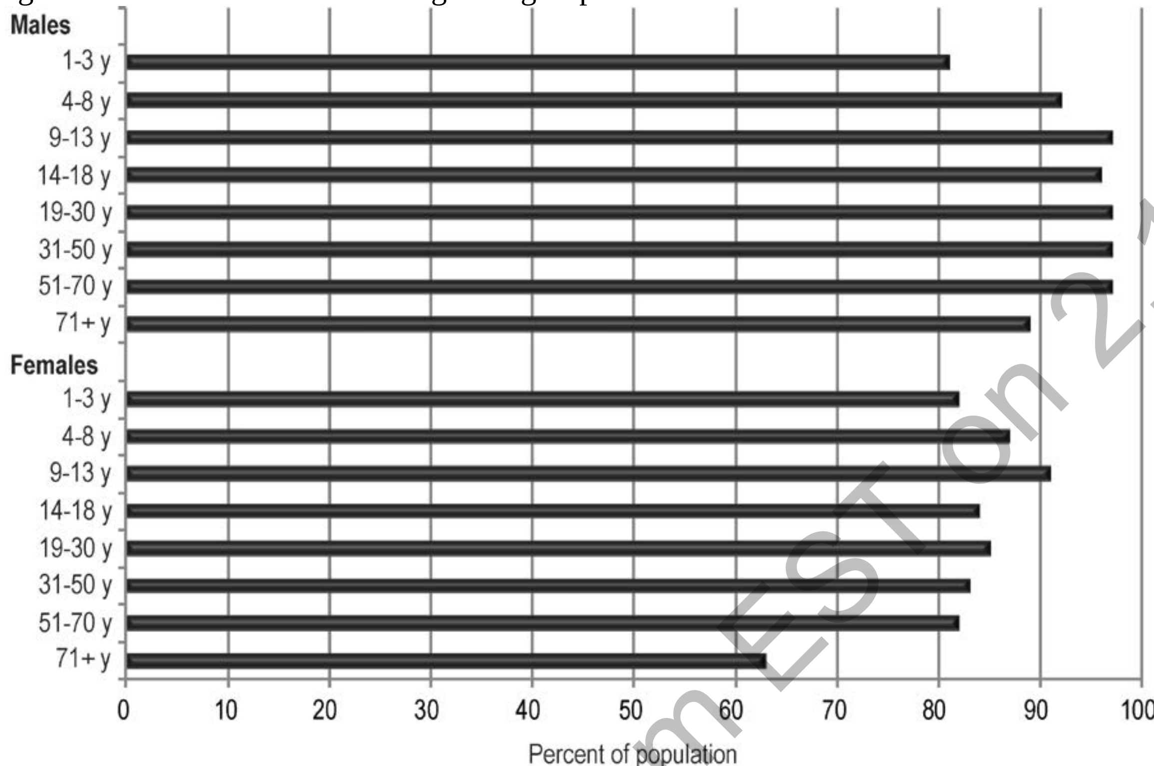
Source: What We Eat in America, NHANES 2007-2010

Figure D1.2 Percent of population with usual intakes above AI



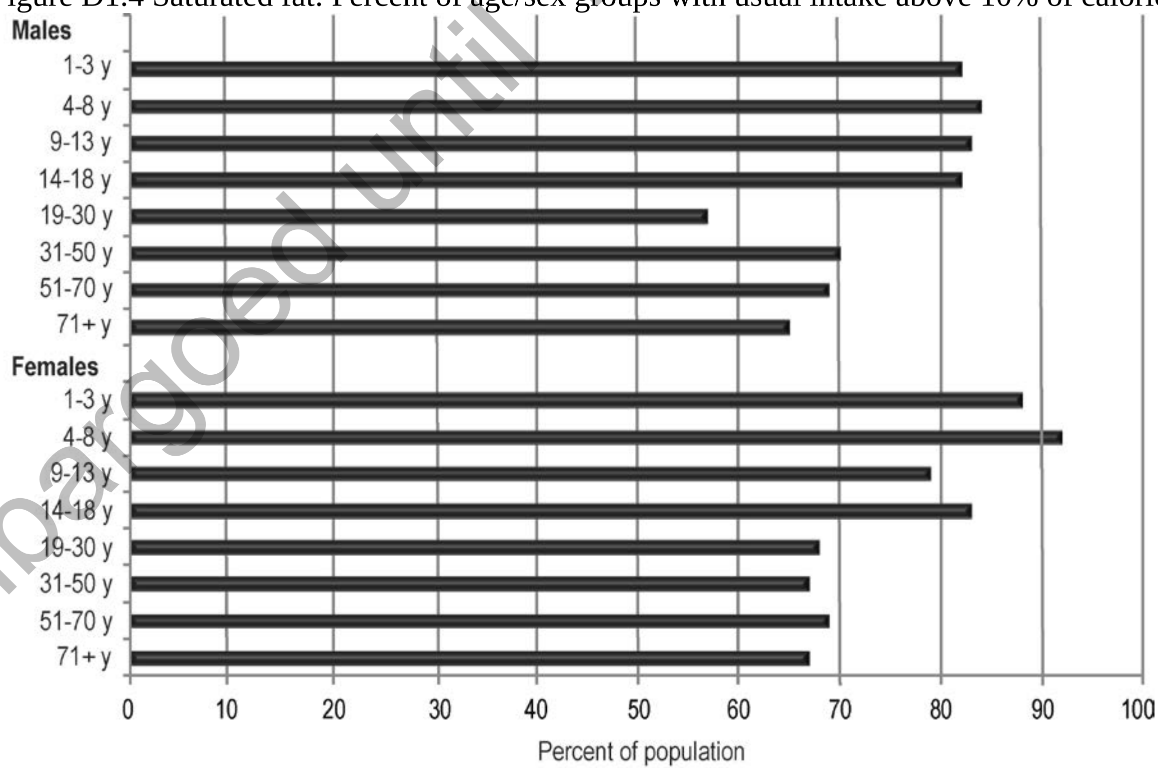
Source: What We Eat in America, NHANES 2007-2010

Figure D1.3 Sodium: Percent of age/sex groups with usual intakes above UL



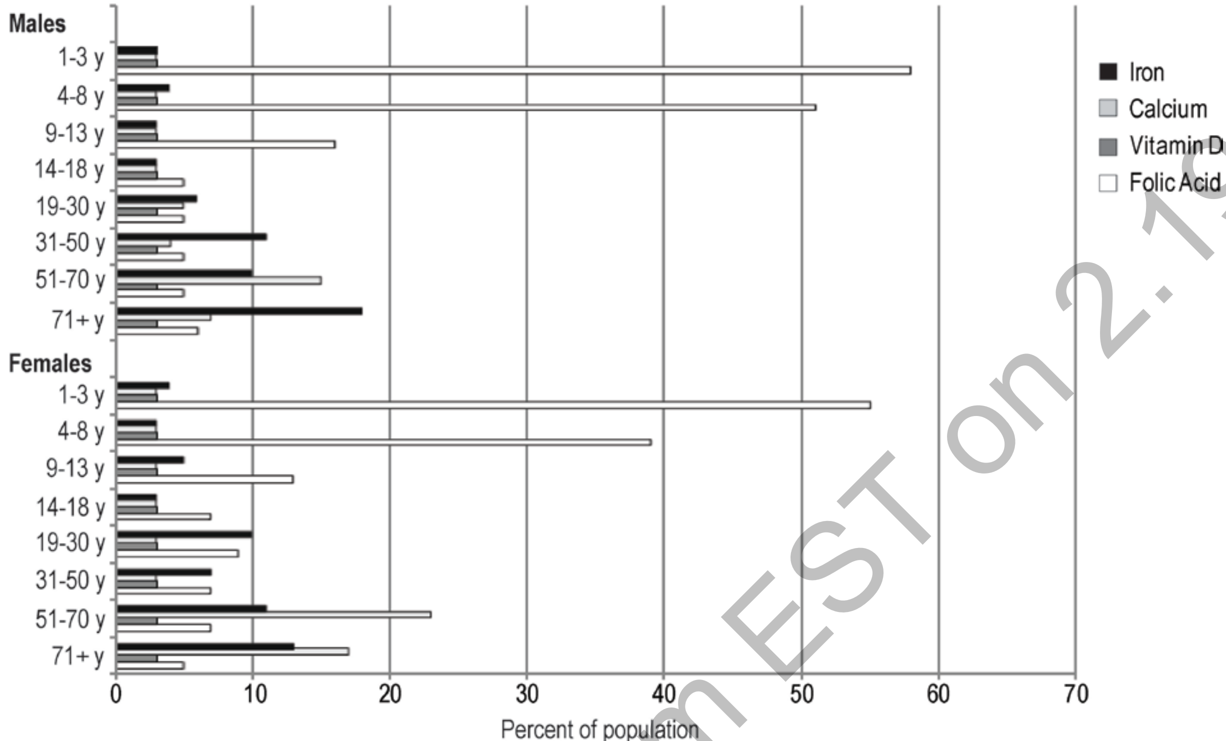
Source: What We Eat in America, NHANES 2007-2010

Figure D1.4 Saturated fat: Percent of age/sex groups with usual intake above 10% of calories



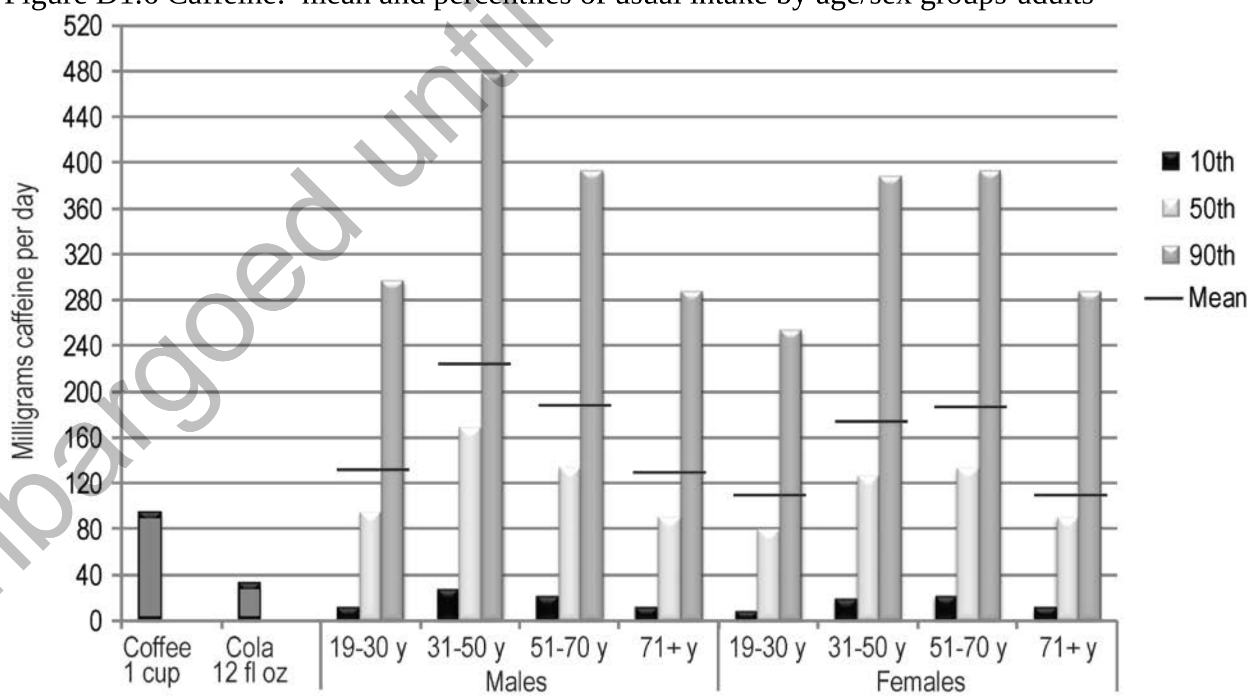
Source: What We Eat in America, NHANES 2007-2010

Figure D1.5 Supplement users: Percent with usual intakes from foods, beverages, and supplements greater than the UL



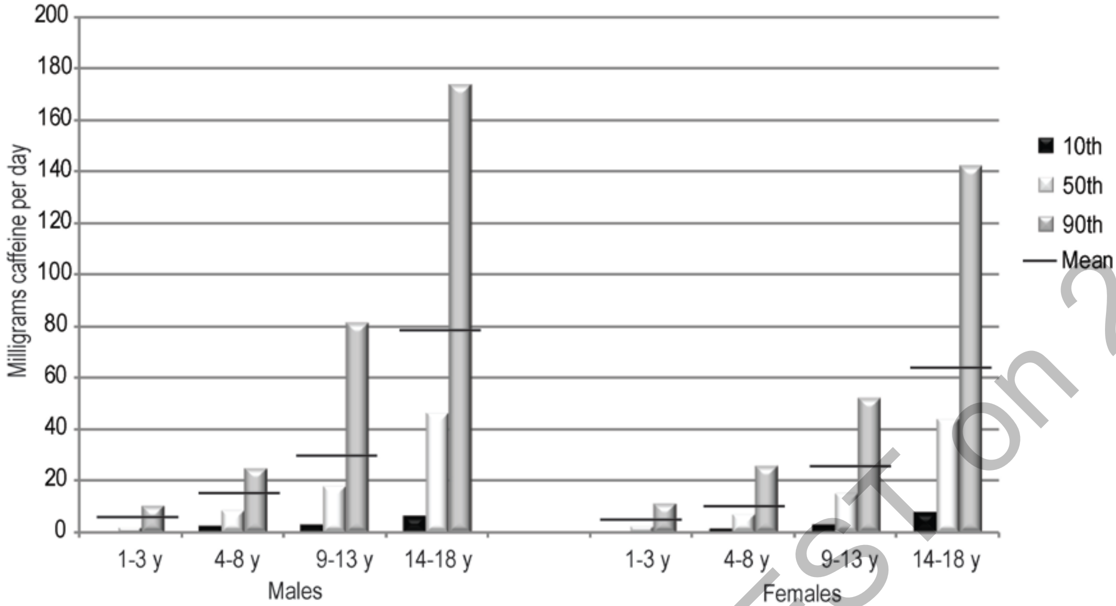
Source: What We Eat in America, NHANES 2007-2010

Figure D1.6 Caffeine: mean and percentiles of usual intake by age/sex groups-adults



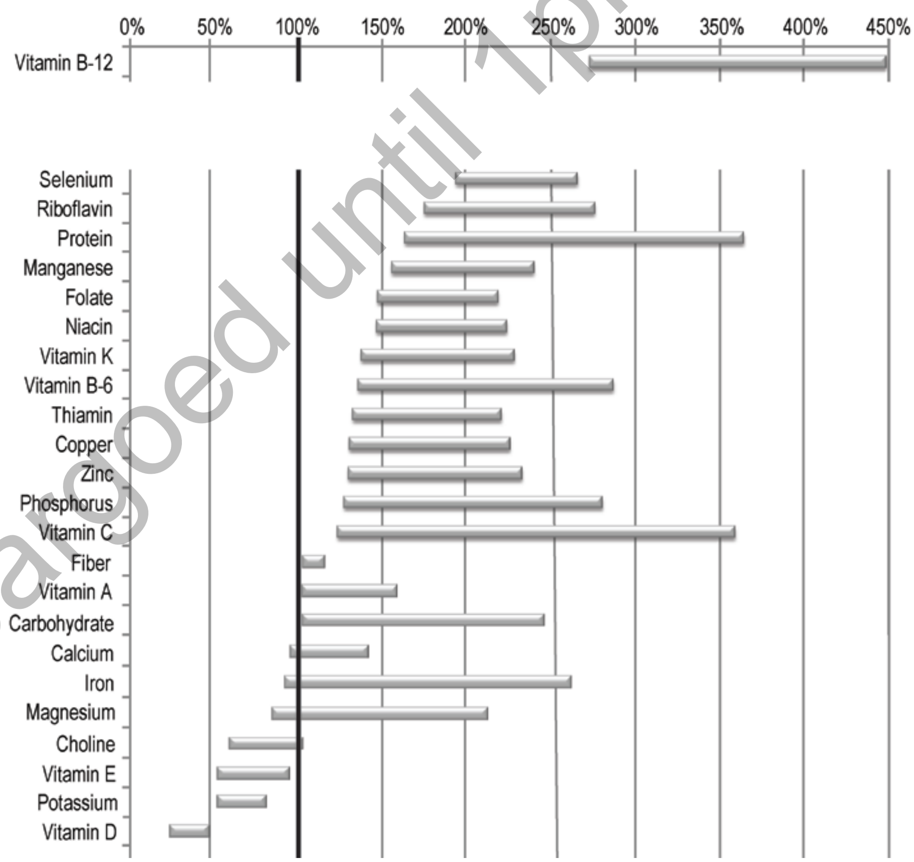
Source: What We Eat in America, NHANES 2007-2010

Figure D1.7 Caffeine: mean and percentiles of usual intake by age/sex groups-children and adolescents



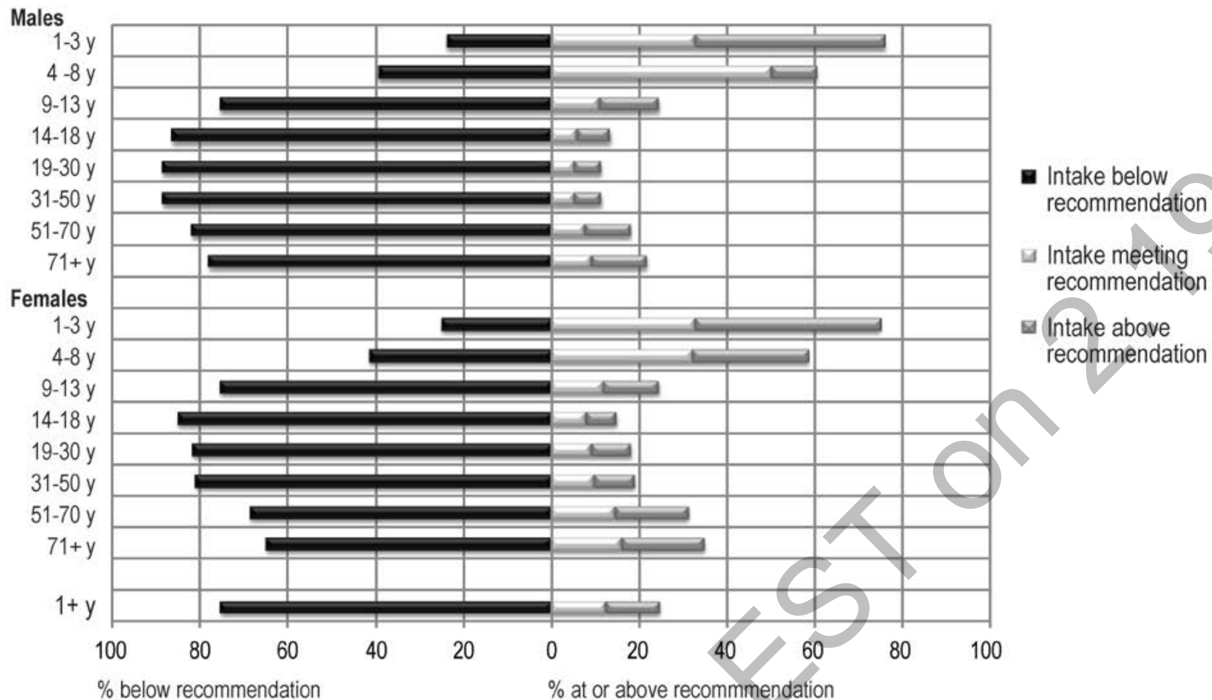
Source: What We Eat in America, NHANES 2007-2010

Figure D1.8 USDA Food Patterns: Range of nutrients in patterns as a percent of the target levels for all age/gender groups



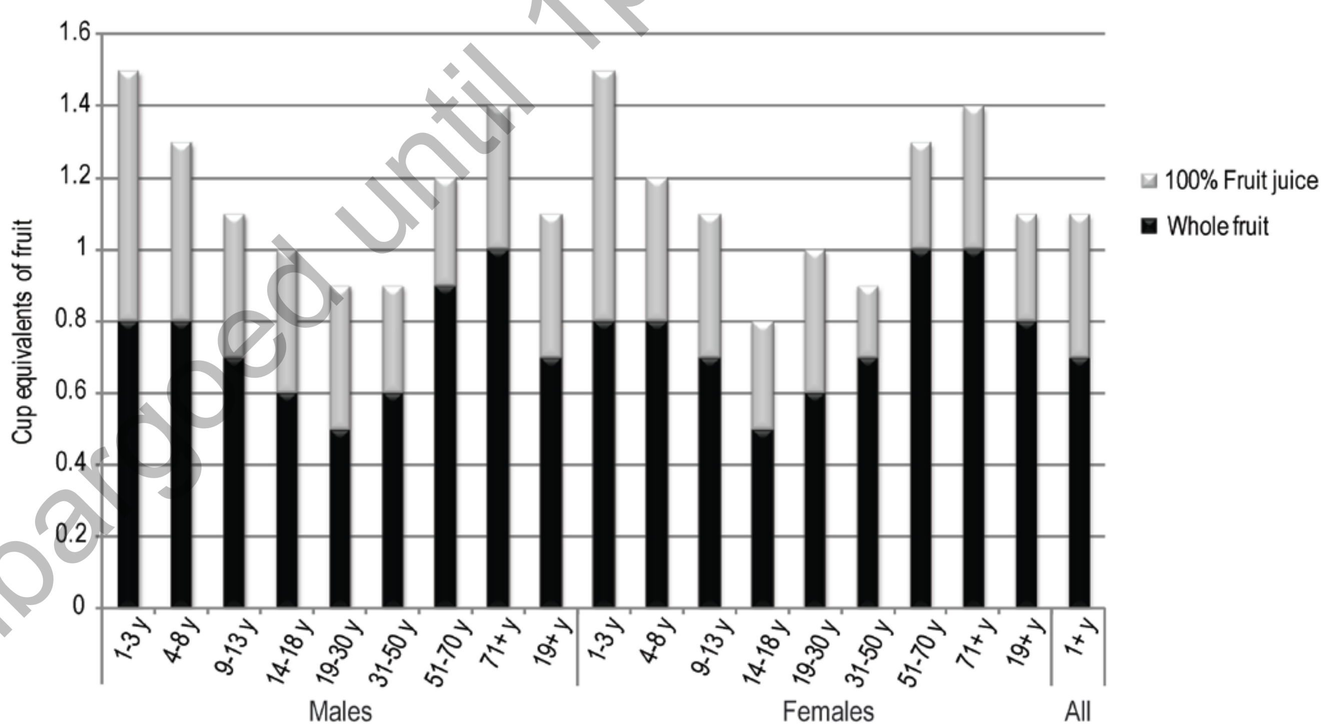
Source: What We Eat in America, NHANES 2007-2010

Figure D1.9 Total Fruit: Estimated percent of persons below, at, or above recommendation



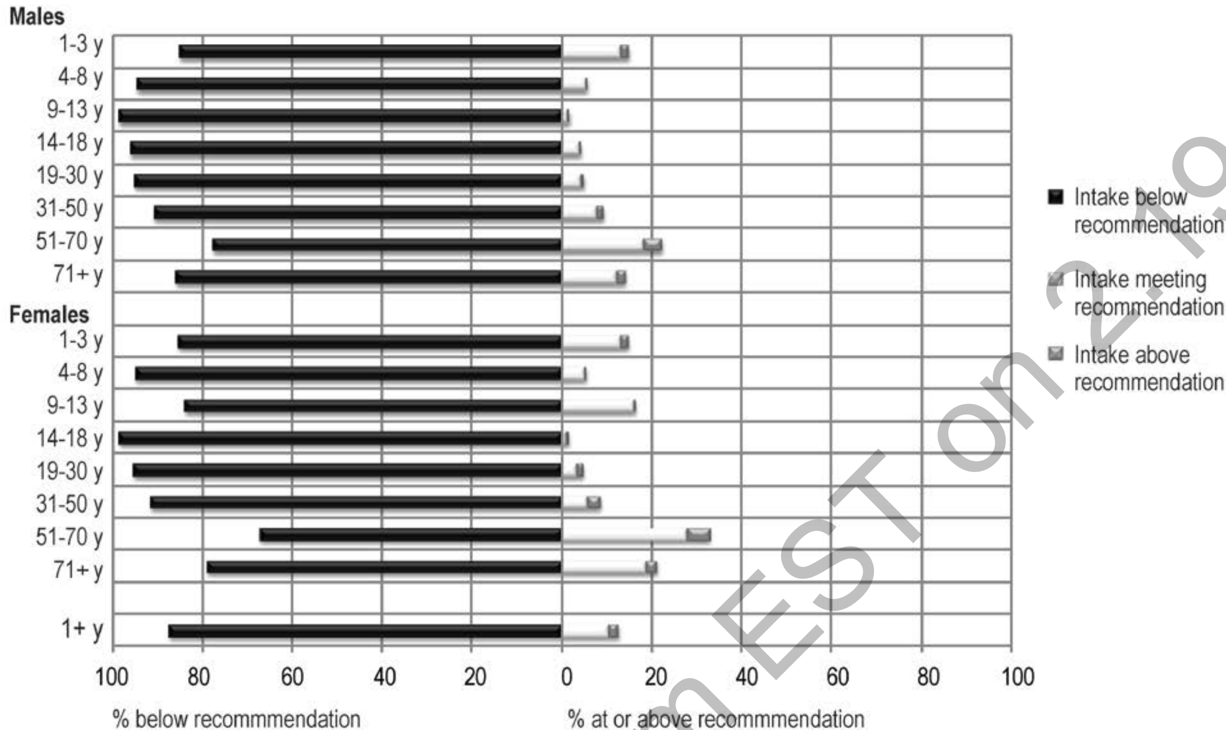
Source: What We Eat in America, NHANES 2007-2010

Figure D1.10 Whole fruit vs. fruit juice consumption by age/sex groups



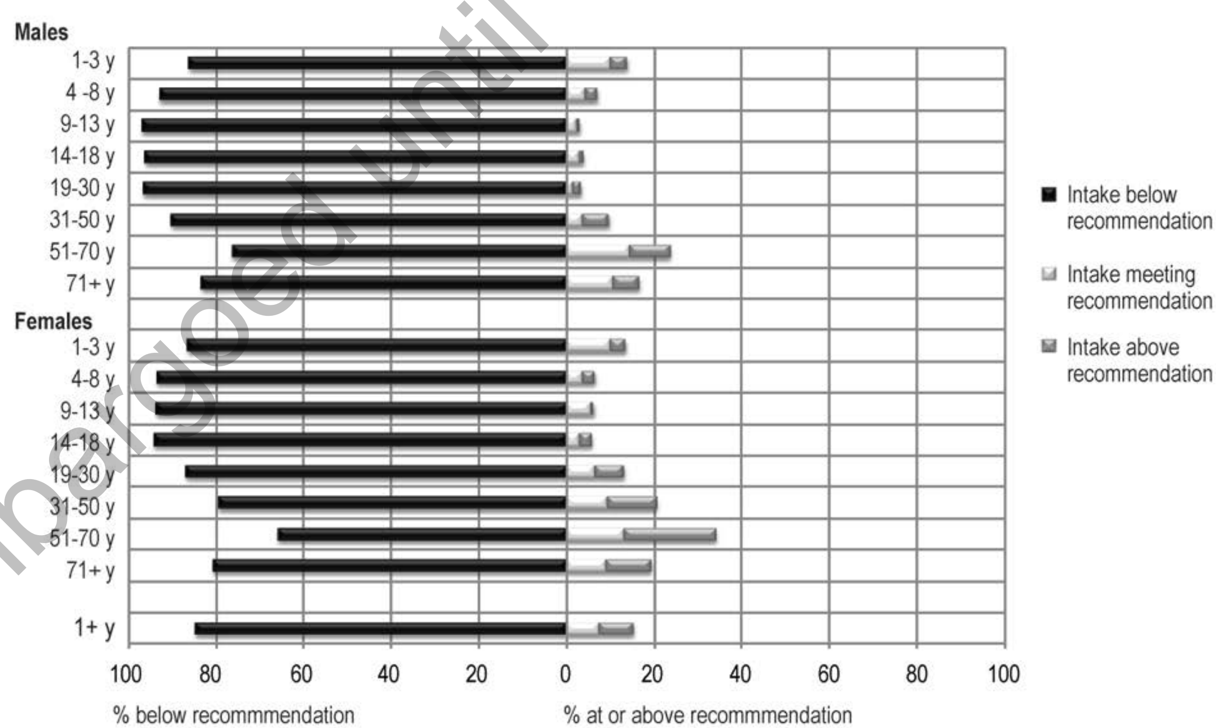
Source: What We Eat in America, NHANES 2007-2010

Figure D1.11 Total Vegetables: Estimated percent of persons below, at, or above recommendation



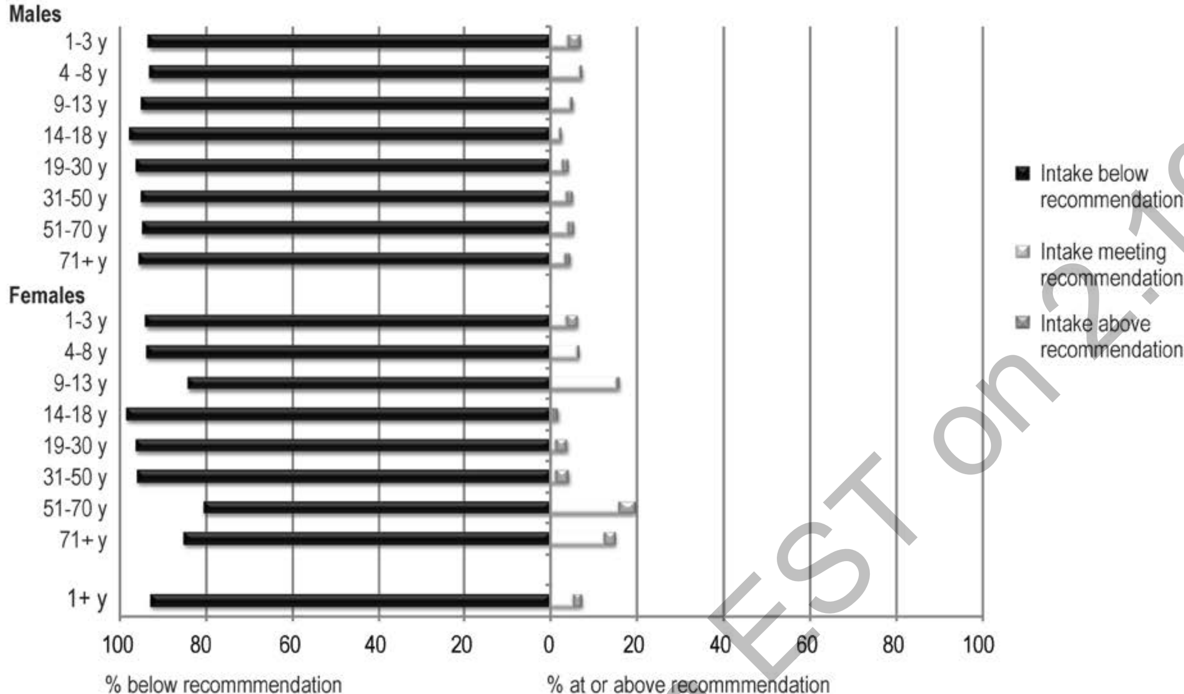
Source: What We Eat in America, NHANES 2007-2010

Figure D1.12 Dark Green vegetables: Estimated percent of persons below, at, or above recommendation



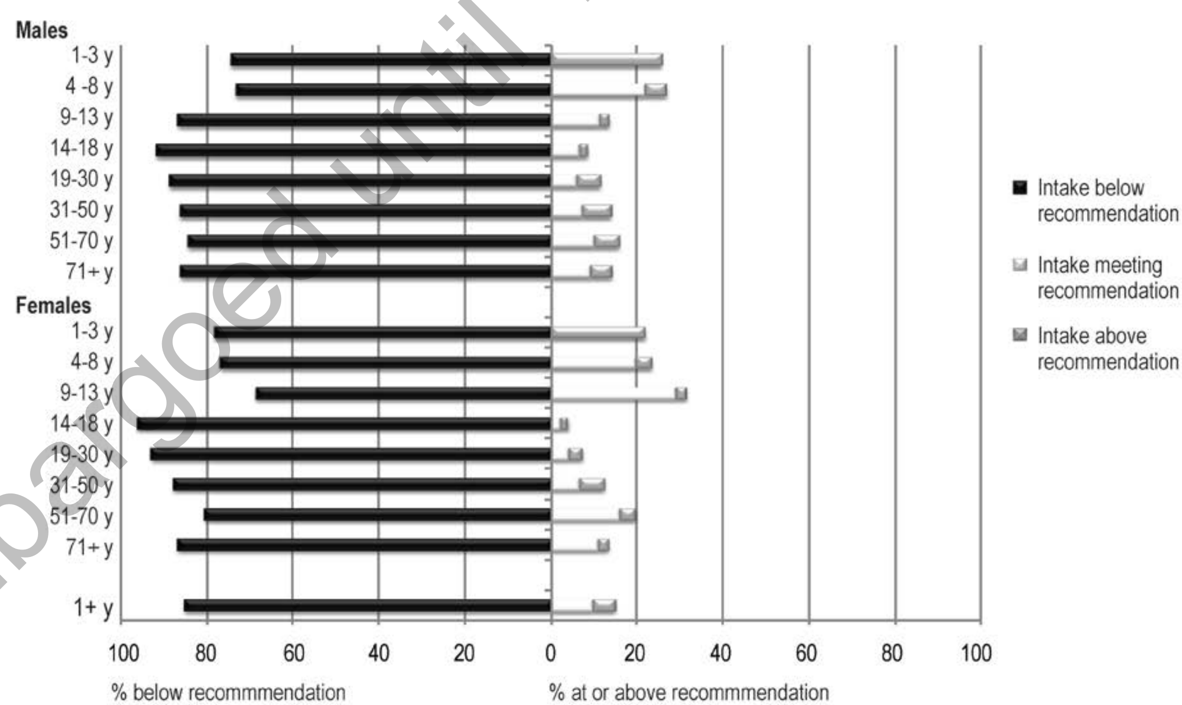
Source: What We Eat in America, NHANES 2007-2010

Figure D1.13 Red and Orange vegetables: Estimated percent of persons below, at, or above recommendation



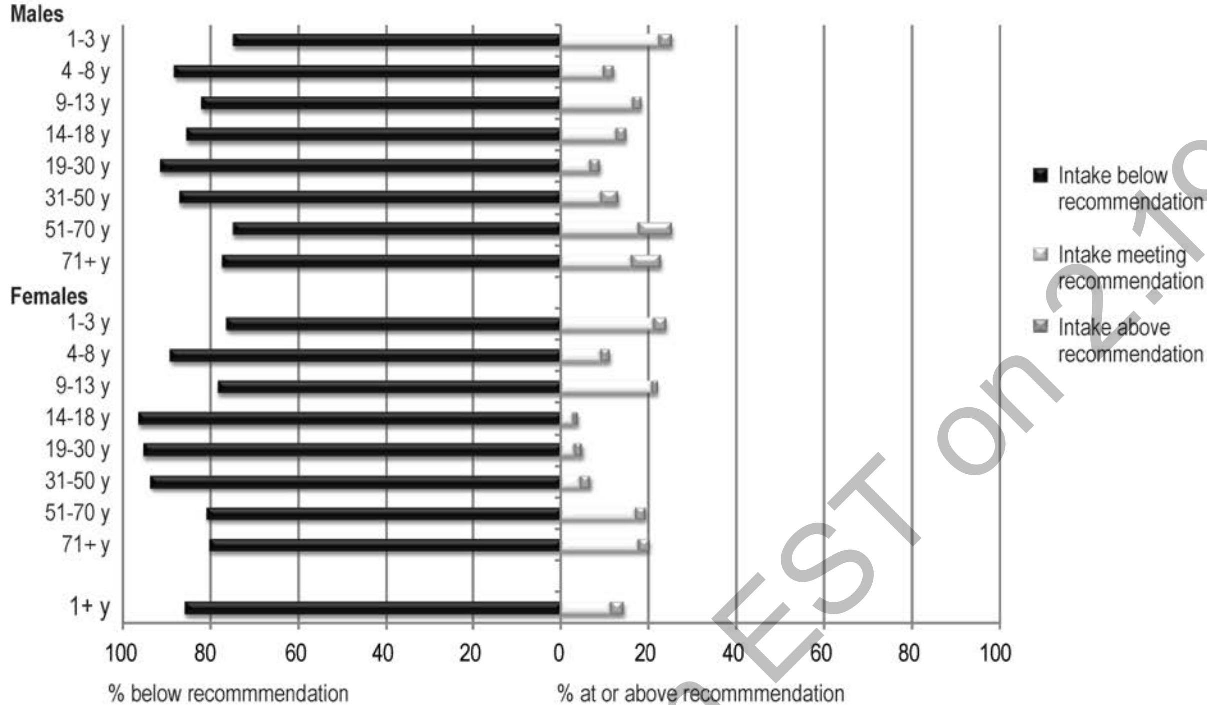
Source: What We Eat in America, NHANES 2007-2010

Figure D1.14 Beans and Peas: Estimated percent of persons below, at, or above recommendation



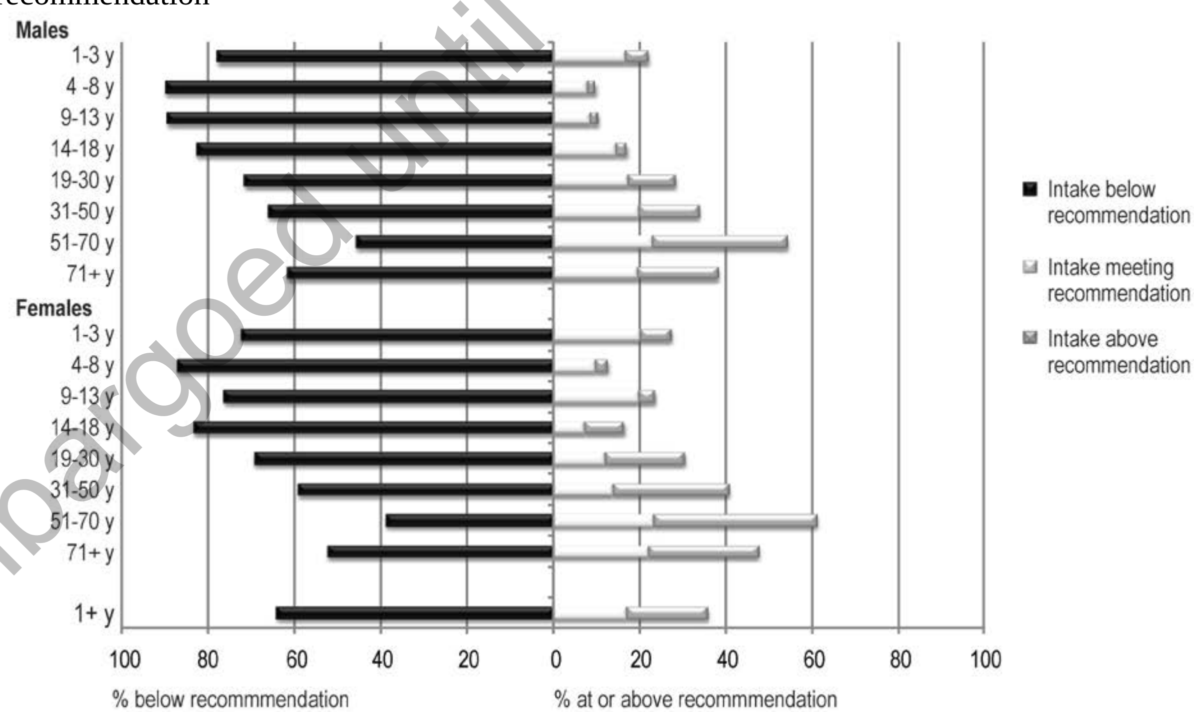
Source: What We Eat in America, NHANES 2007-2010

Figure D1.15 Starchy vegetables: Estimated percent of persons below, at, or above recommendation



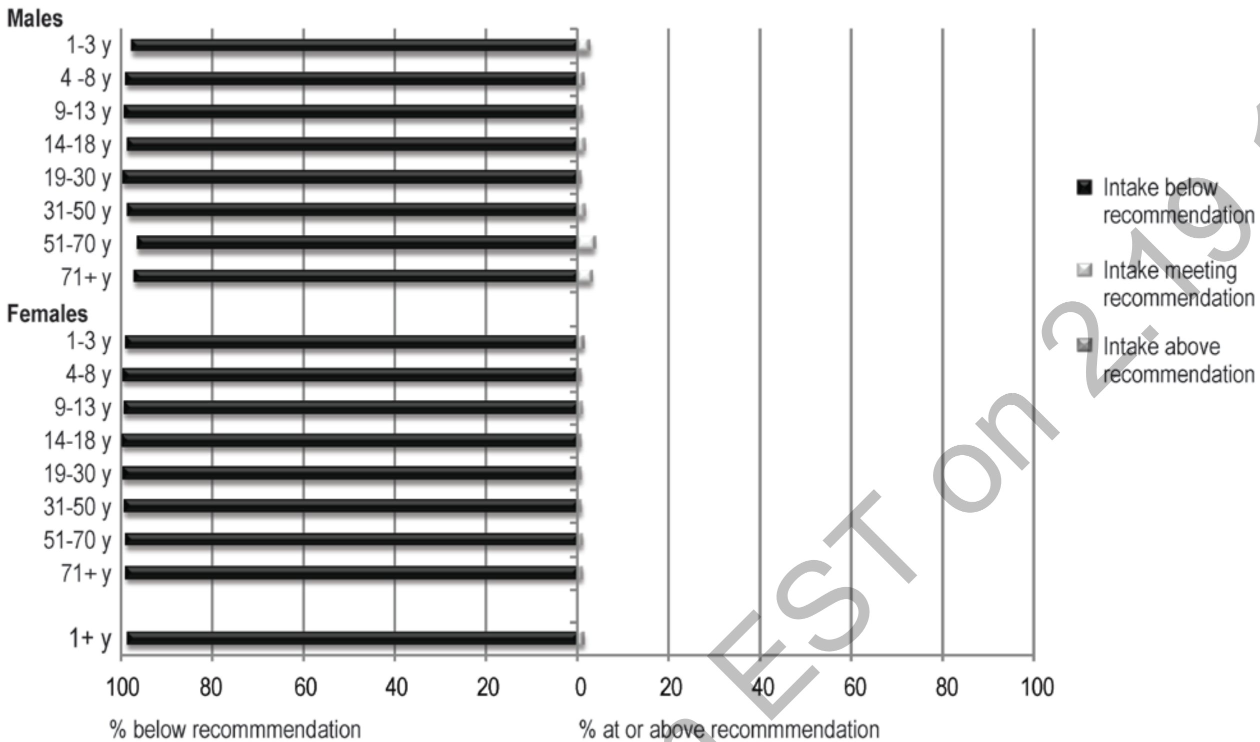
Source: What We Eat in America, NHANES 2007-2010

Figure D1.16 Other vegetables: Estimated percent of persons below, at, or above recommendation



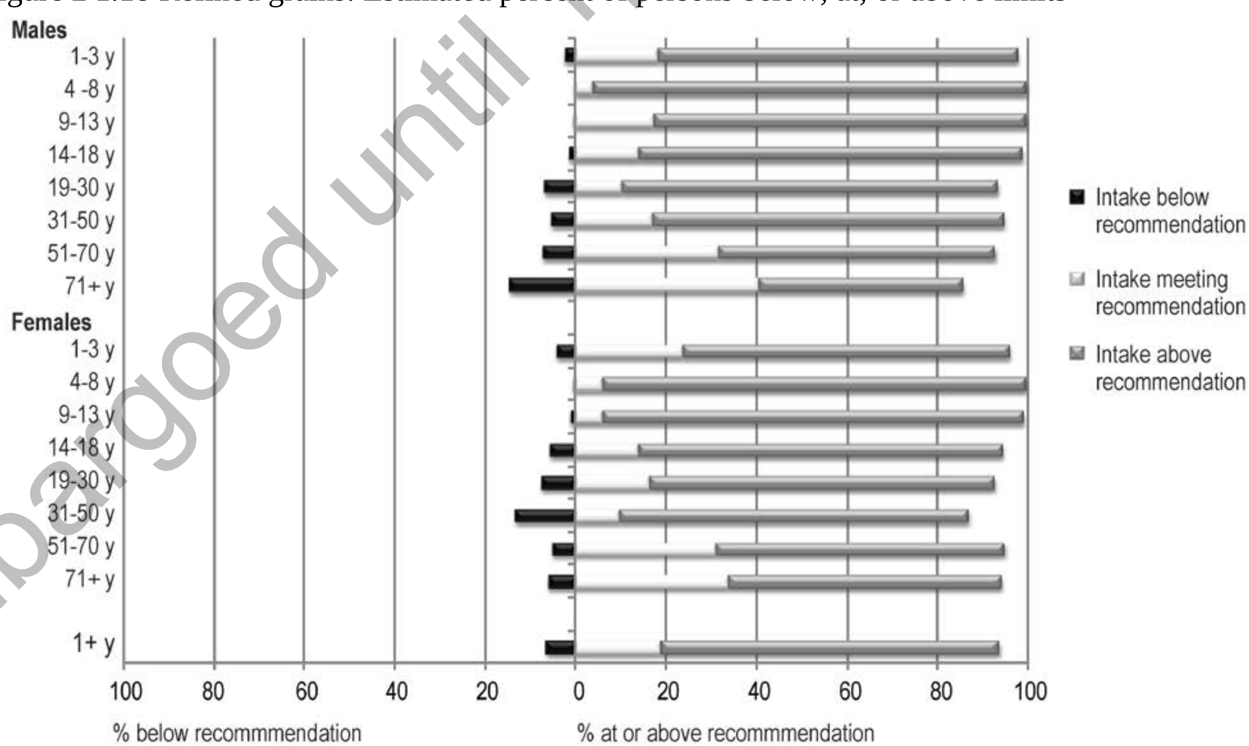
Source: What We Eat in America, NHANES 2007-2010

Figure D1.17 Whole grains: Estimated percent of persons below, at, or above recommendation



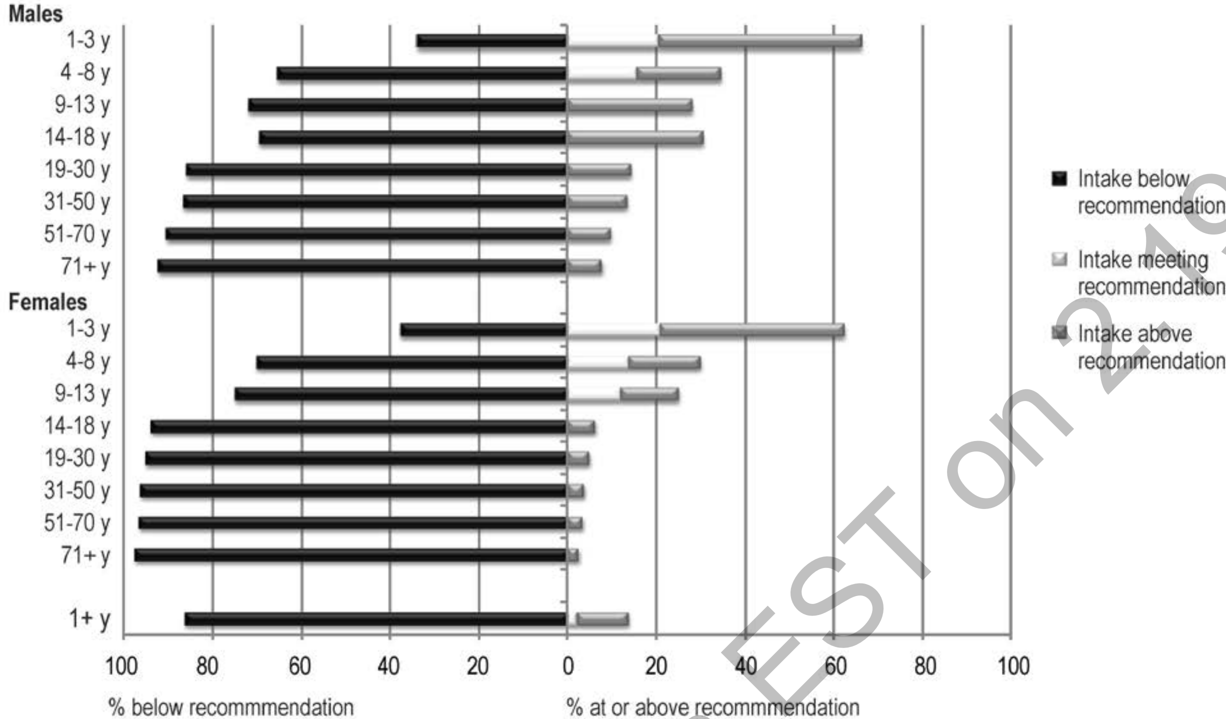
Source: What We Eat in America, NHANES 2007-2010

Figure D1.18 Refined grains: Estimated percent of persons below, at, or above limits



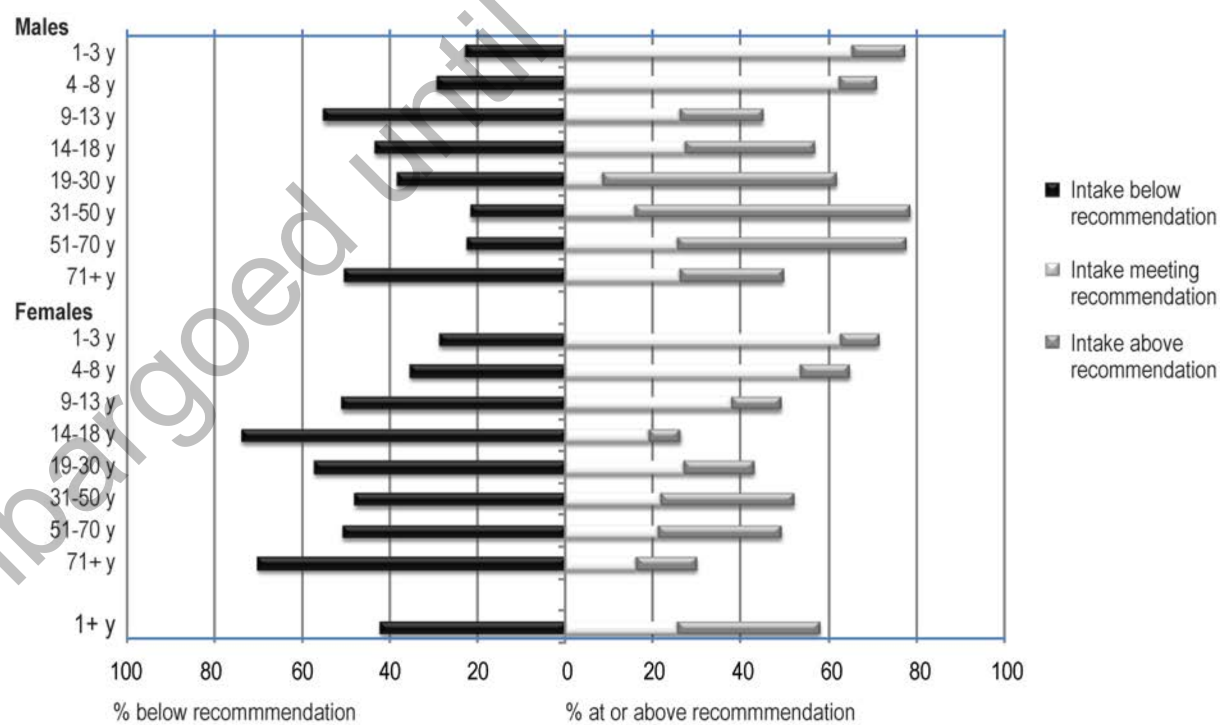
Source: What We Eat in America, NHANES 2007-2010

Figure D1.19 Dairy: Estimated percent of persons below, at, or above recommendation



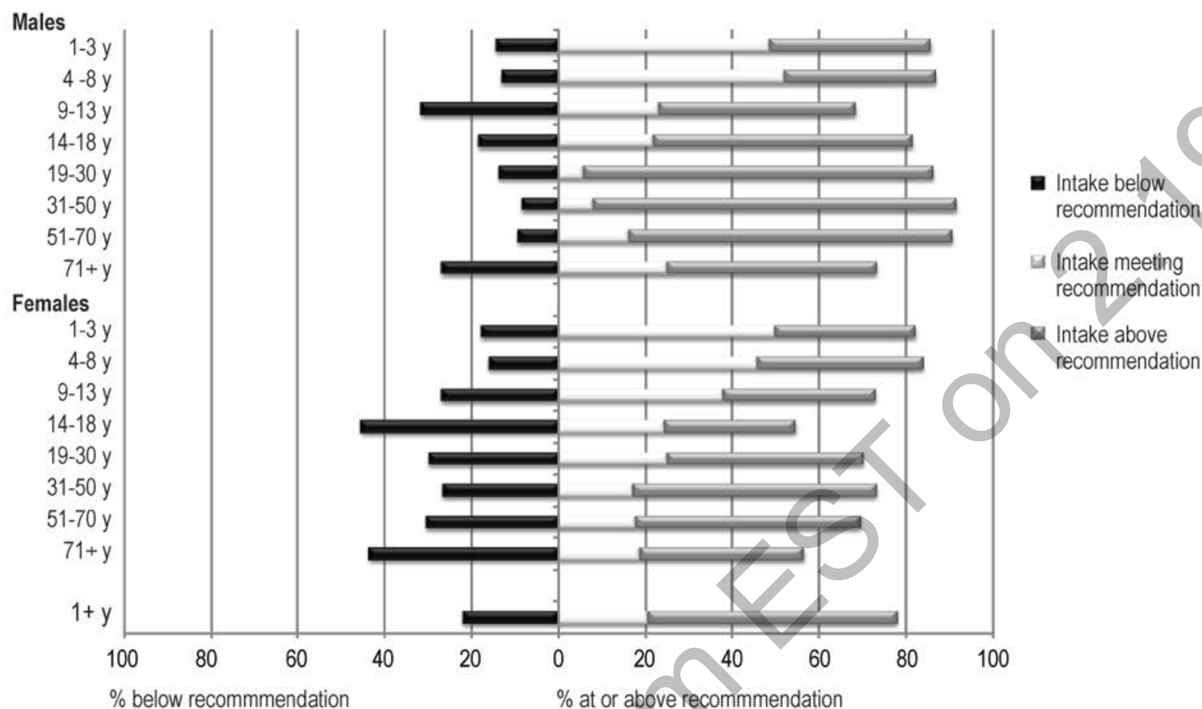
Source: What We Eat in America, NHANES 2007-2010

Figure D1.20 Total Protein foods: Estimated percent of persons below, at, or above recommendation



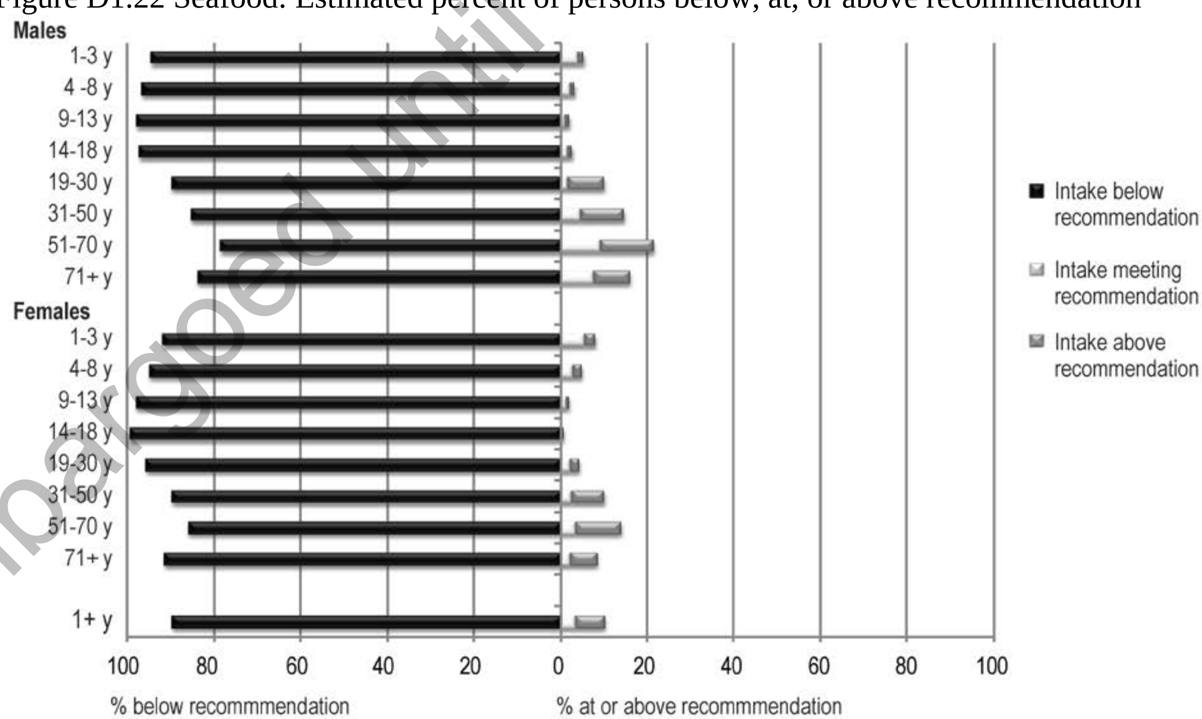
Source: What We Eat in America, NHANES 2007-2010

Figure D1.21 Meat, poultry, eggs: Estimated percent of persons below, at, or above recommendation



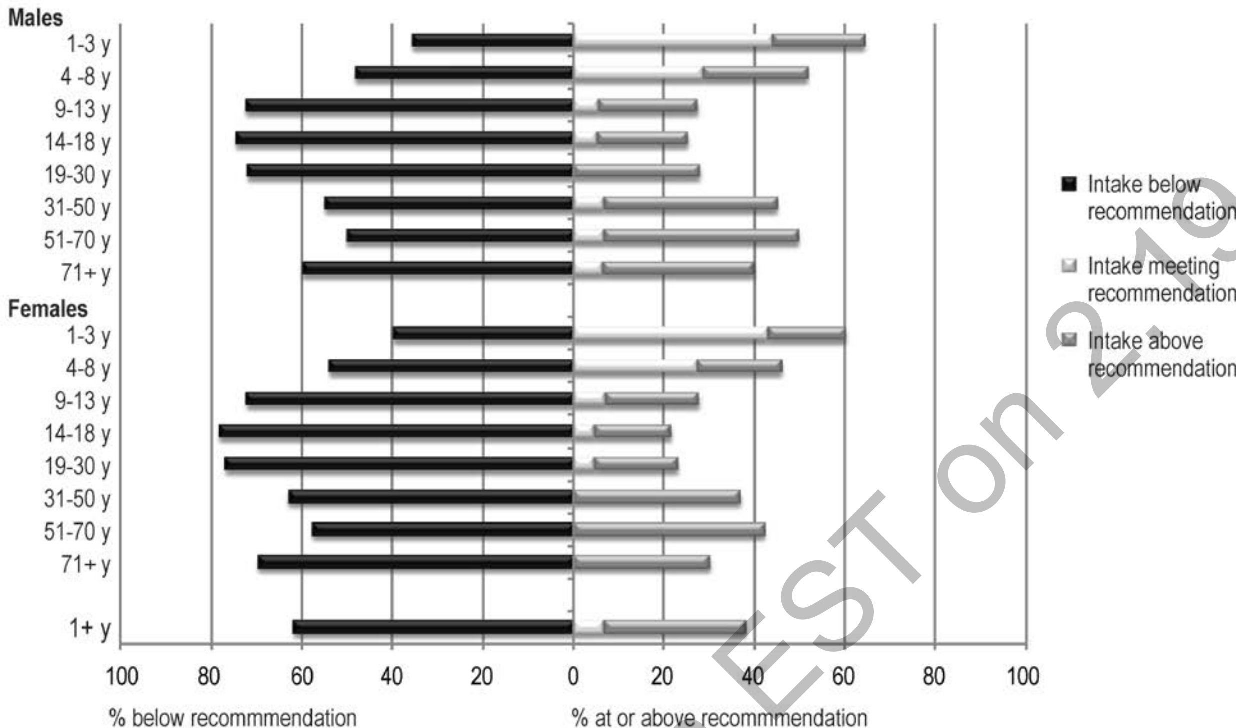
Source: What We Eat in America, NHANES 2007-2010

Figure D1.22 Seafood: Estimated percent of persons below, at, or above recommendation



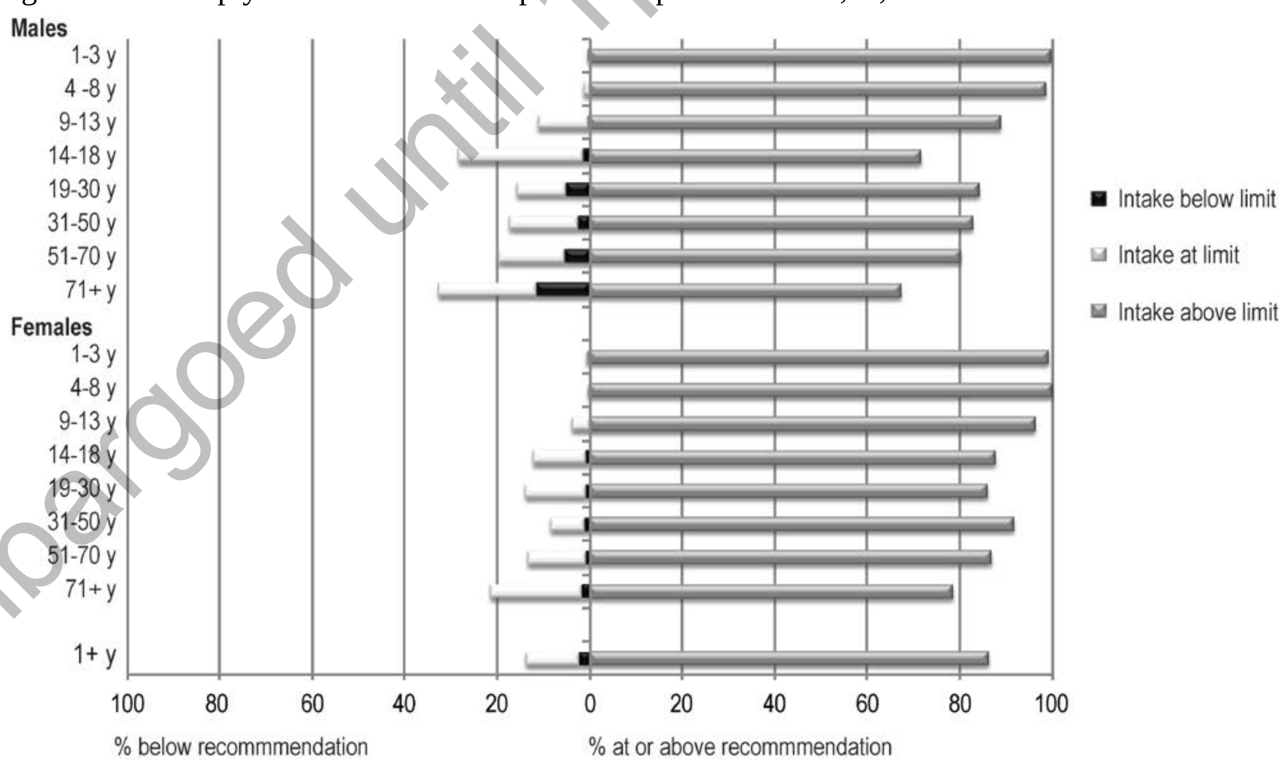
Source: What We Eat in America, NHANES 2007-2010

Figure D1.23 Nuts, seeds, soy: Estimated percent of persons below, at, or above recommendation



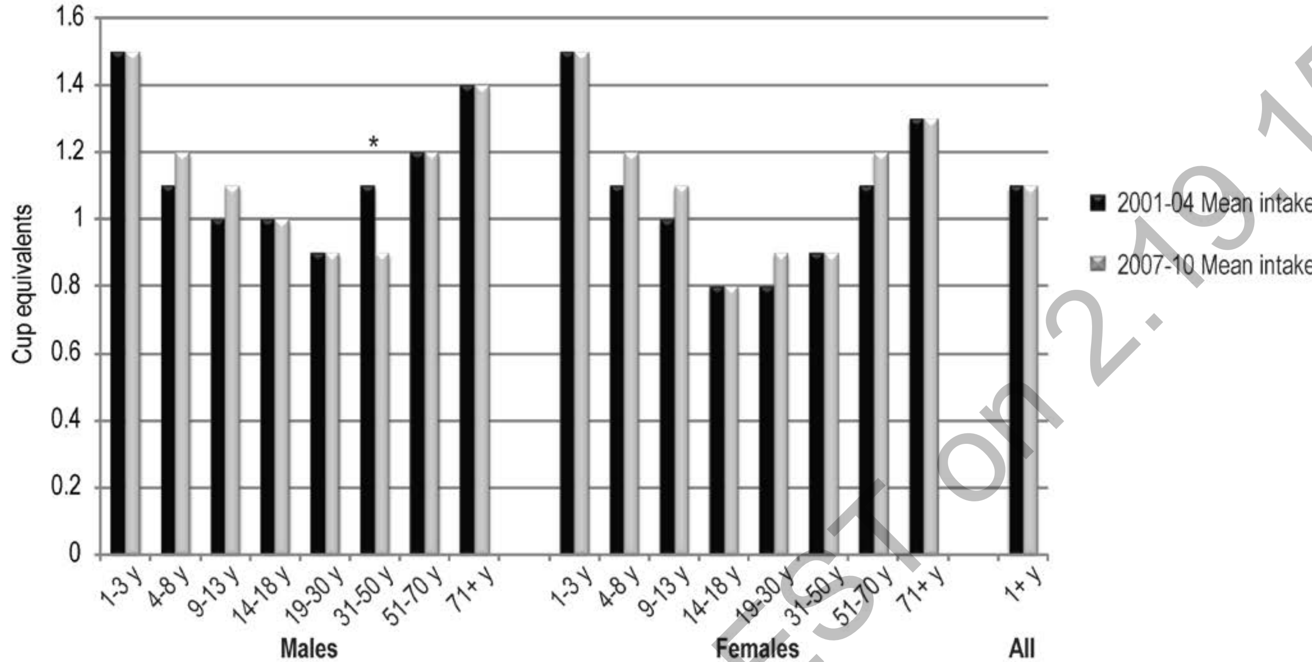
Source: What We Eat in America, NHANES 2007-2010

Figure D1.24 Empty calories: Estimated percent of persons below, at, or above limits



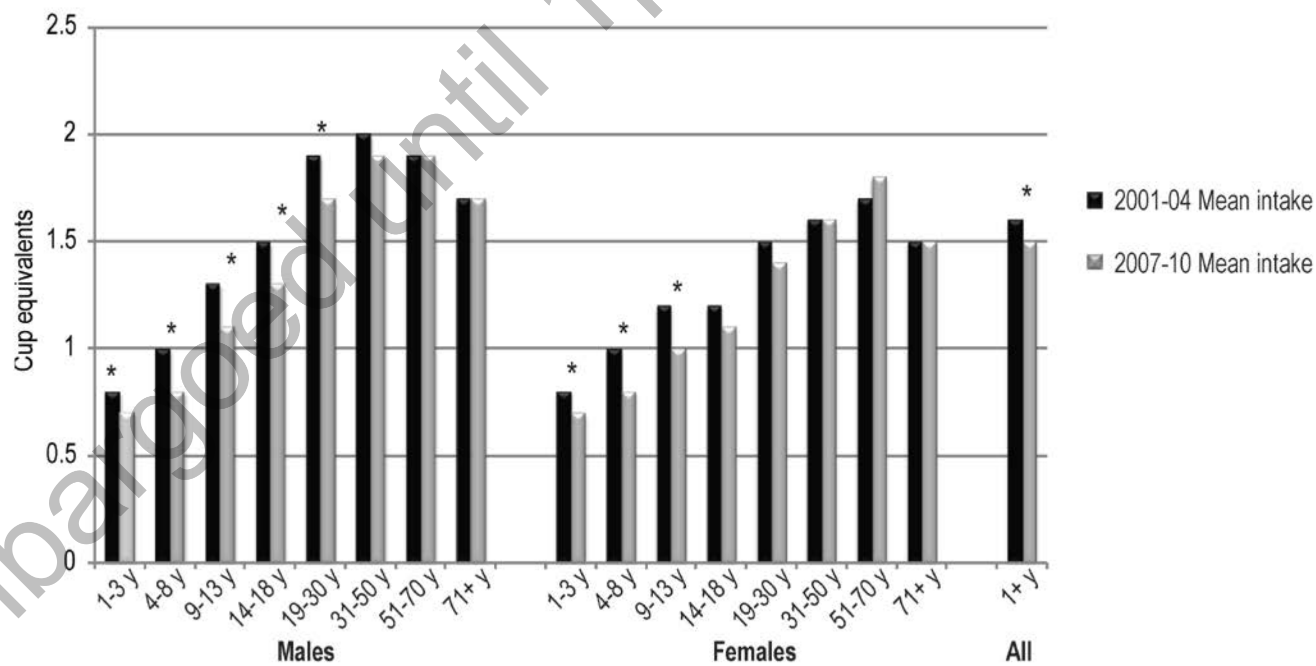
Source: What We Eat in America, NHANES 2007-2010

Figure D1.25 Fruit: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



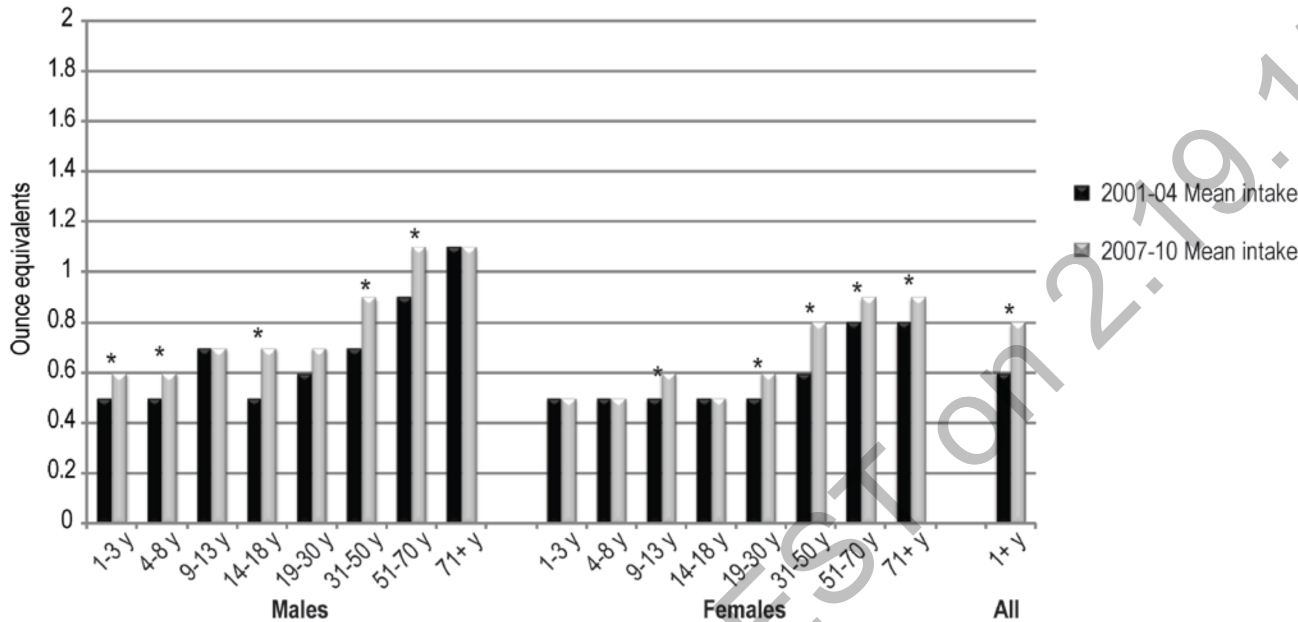
*p<.05
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

Figure D1.26 Vegetables: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



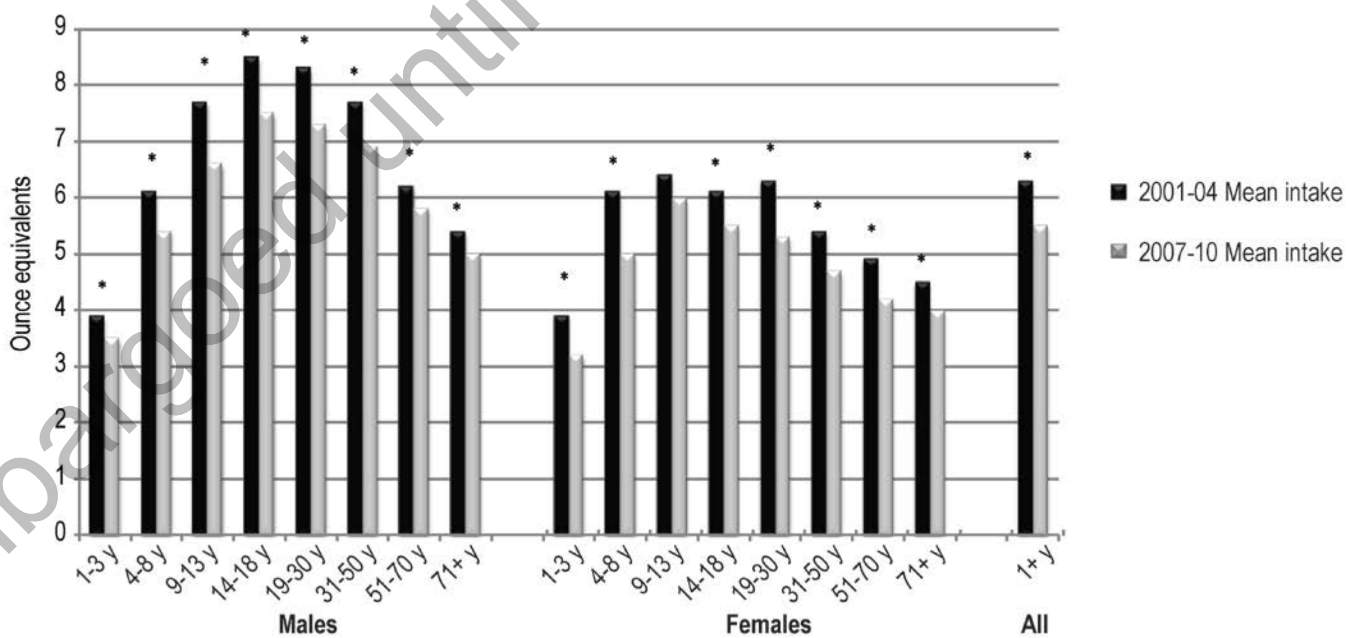
*p<.05
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

Figure D1.27 Whole grains: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



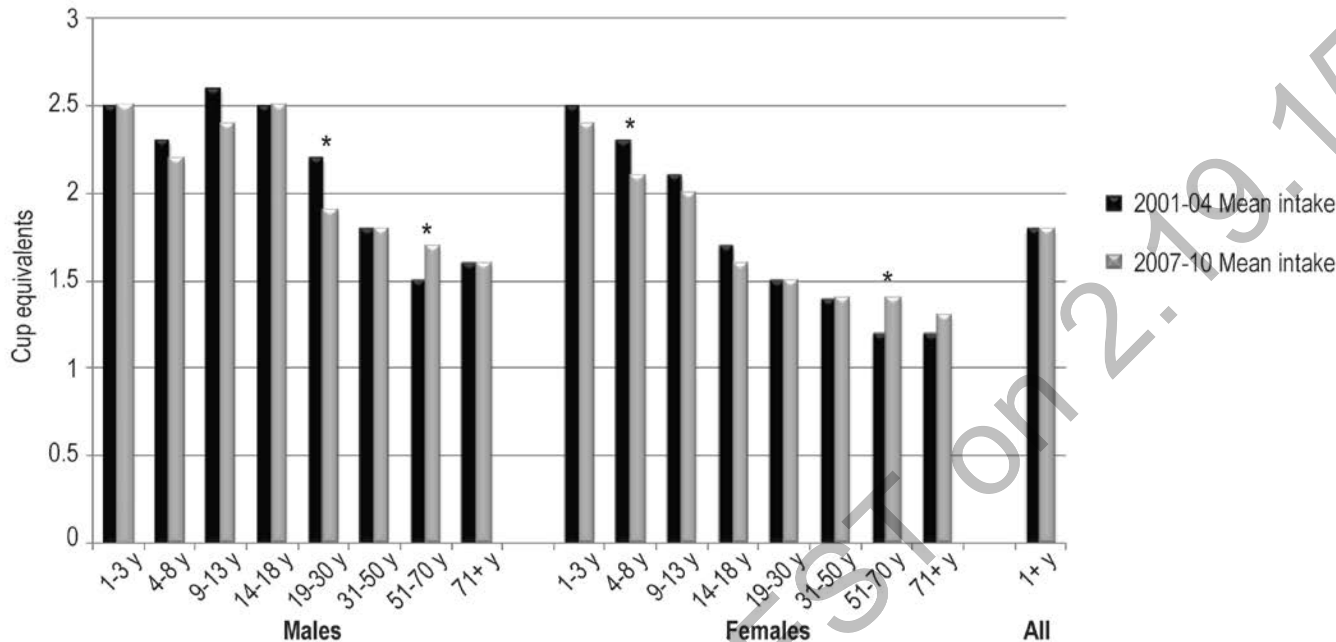
*p<.05
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

Figure D1.28 Refined grains: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

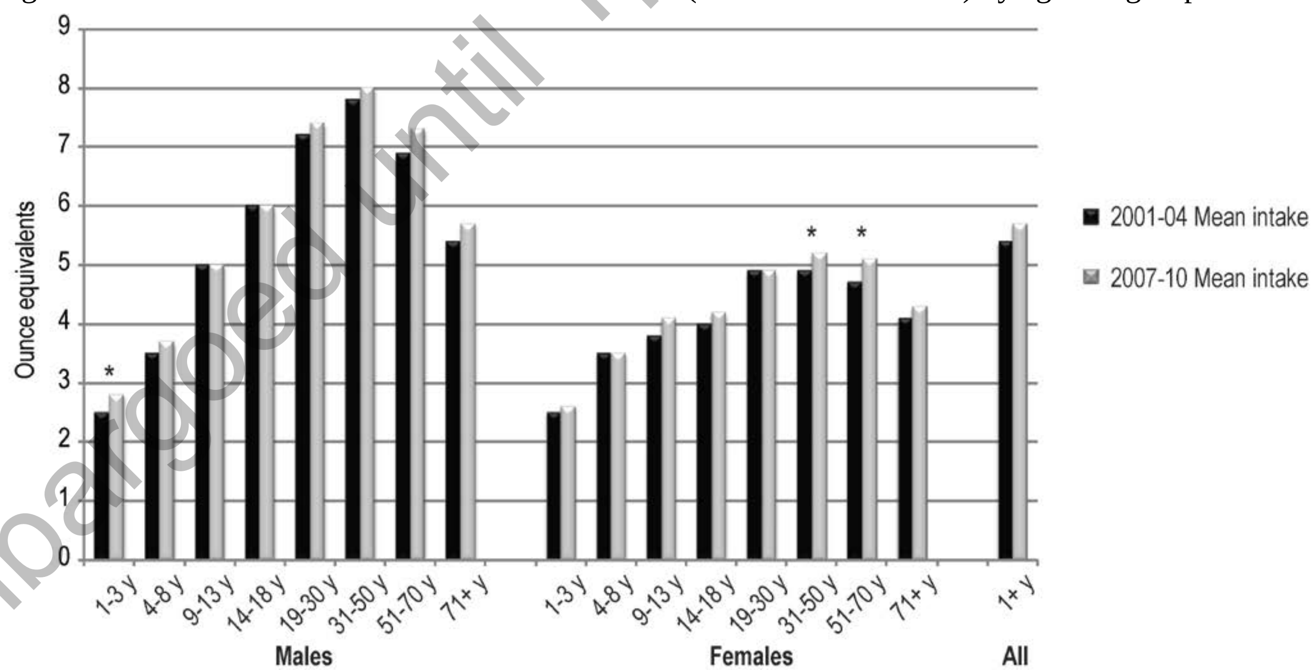
Figure D1.29 Dairy: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

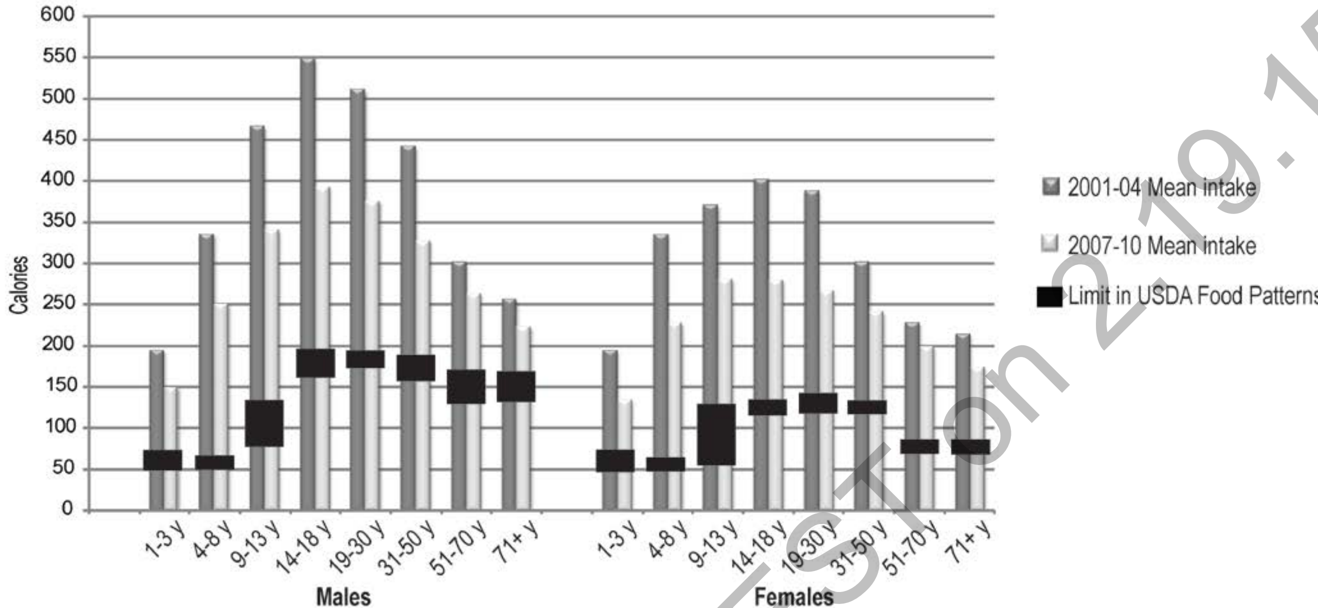
Figure D1.30 Protein Foods: Mean intakes over time (2001-04 vs. 2007-10) by age/sex group



*p<.05

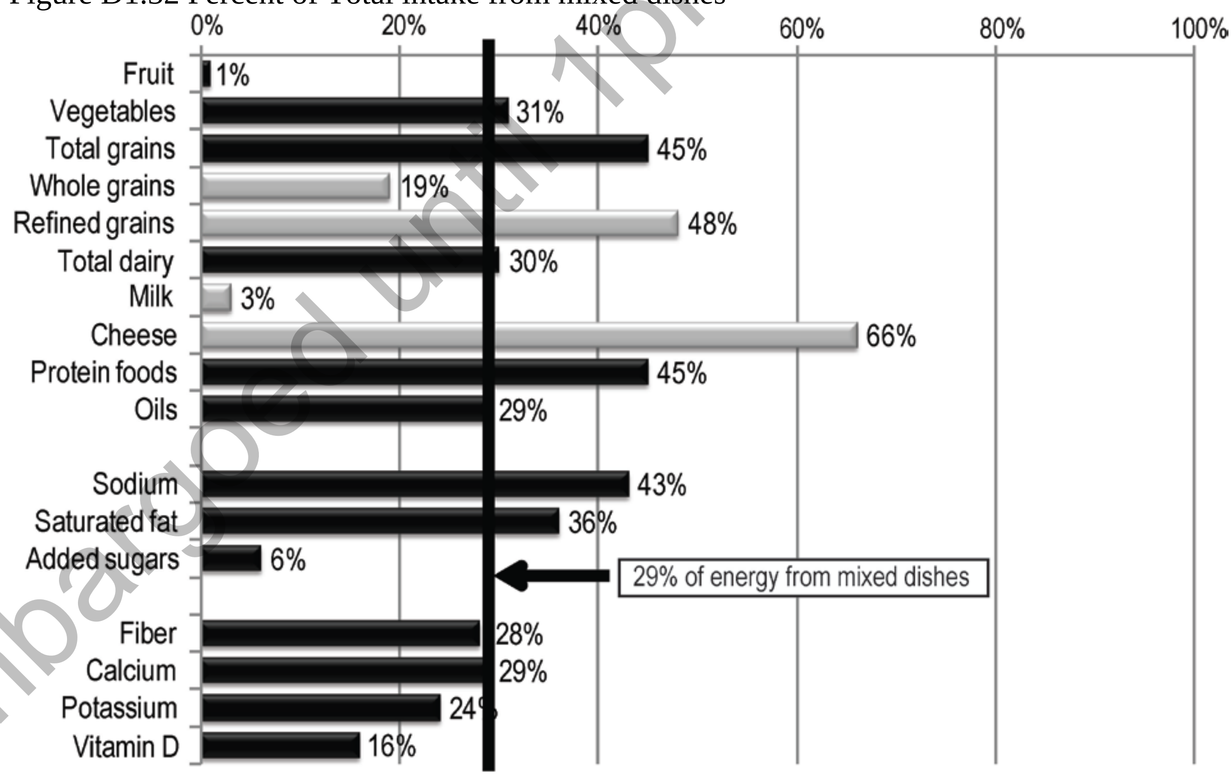
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

Figure D1.31 Added sugars intakes in 2001-04 and 2007-10 by age/sex groups in comparison to added sugars limits in the USDA Food Patterns



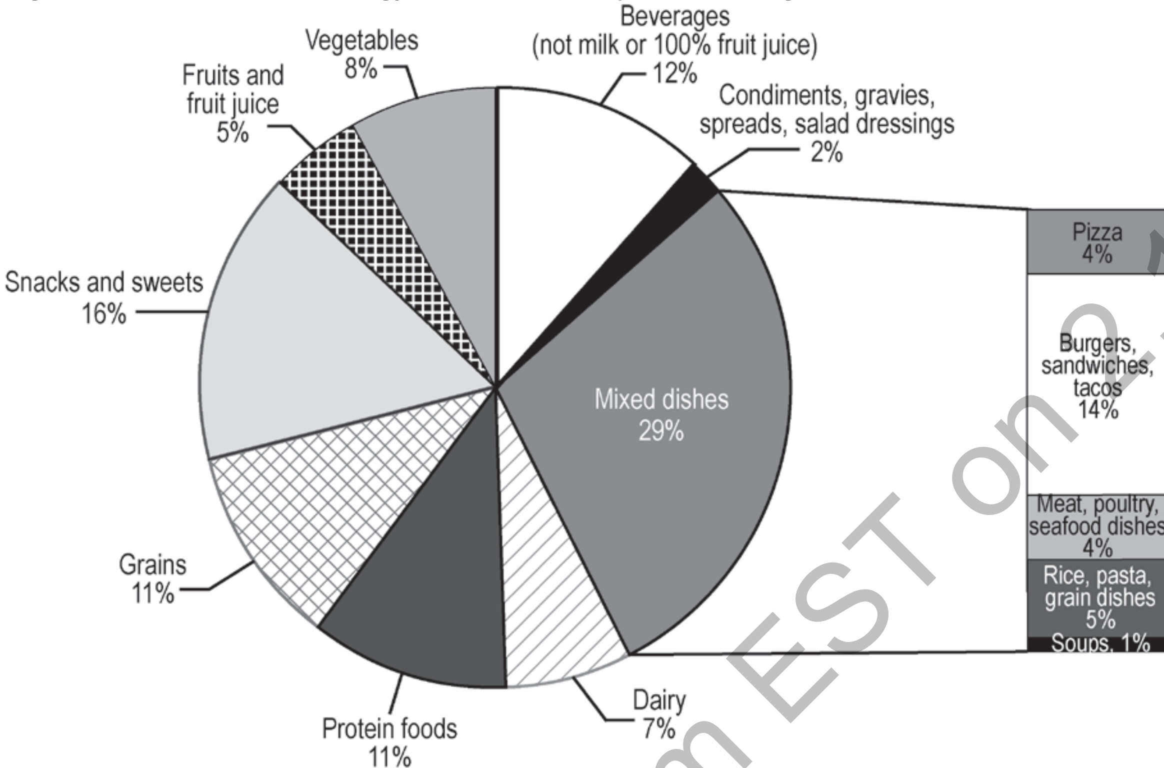
Source: What We Eat in America, NHANES 2001-2004 and 2007-2010

Figure D1.32 Percent of Total intake from mixed dishes



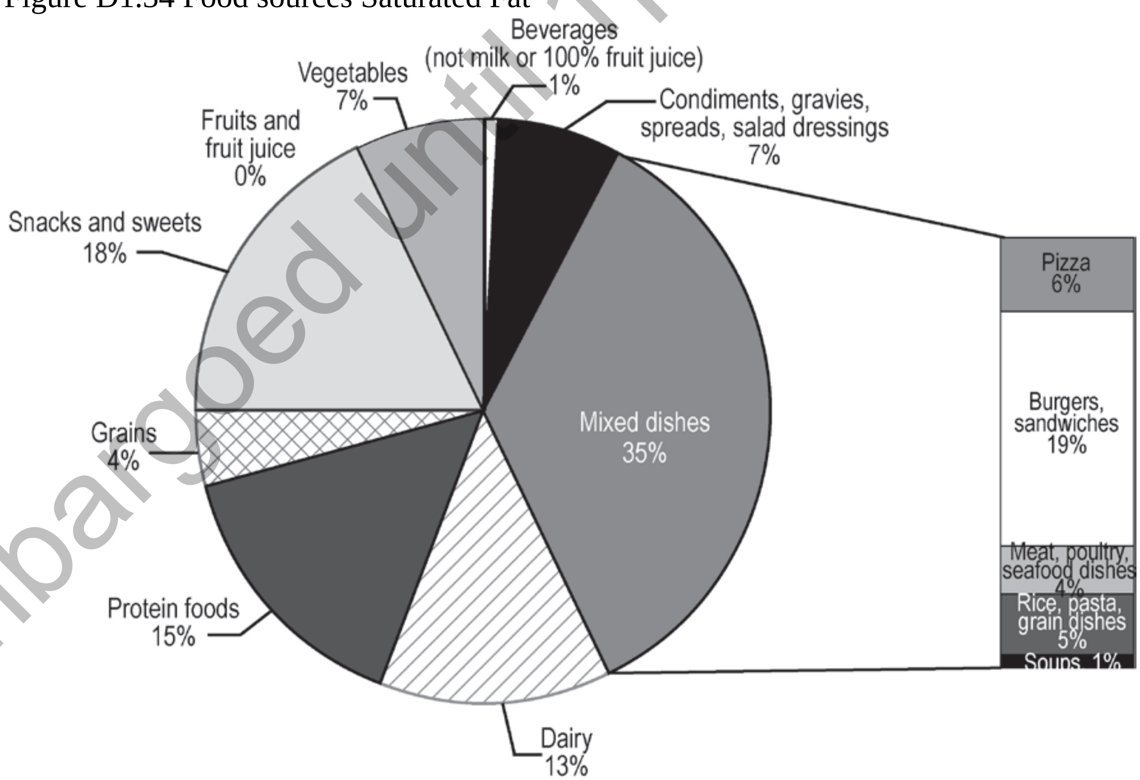
Note: Bars in lighter shades are for subgroups that "break out" the food group above them.
Source: What We Eat in America, NHANES 2009-2010

Figure D1.33 Percent of Energy Intake from Major food categories



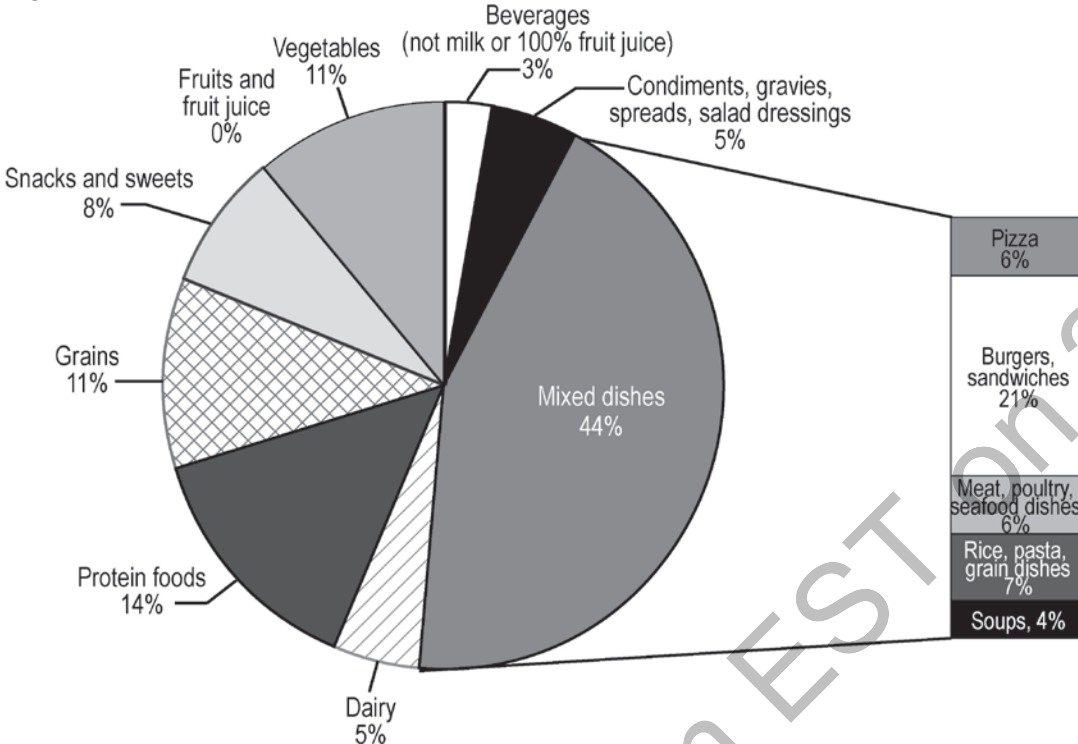
Source: What We Eat in America, NHANES 2009-2010

Figure D1.34 Food sources Saturated Fat



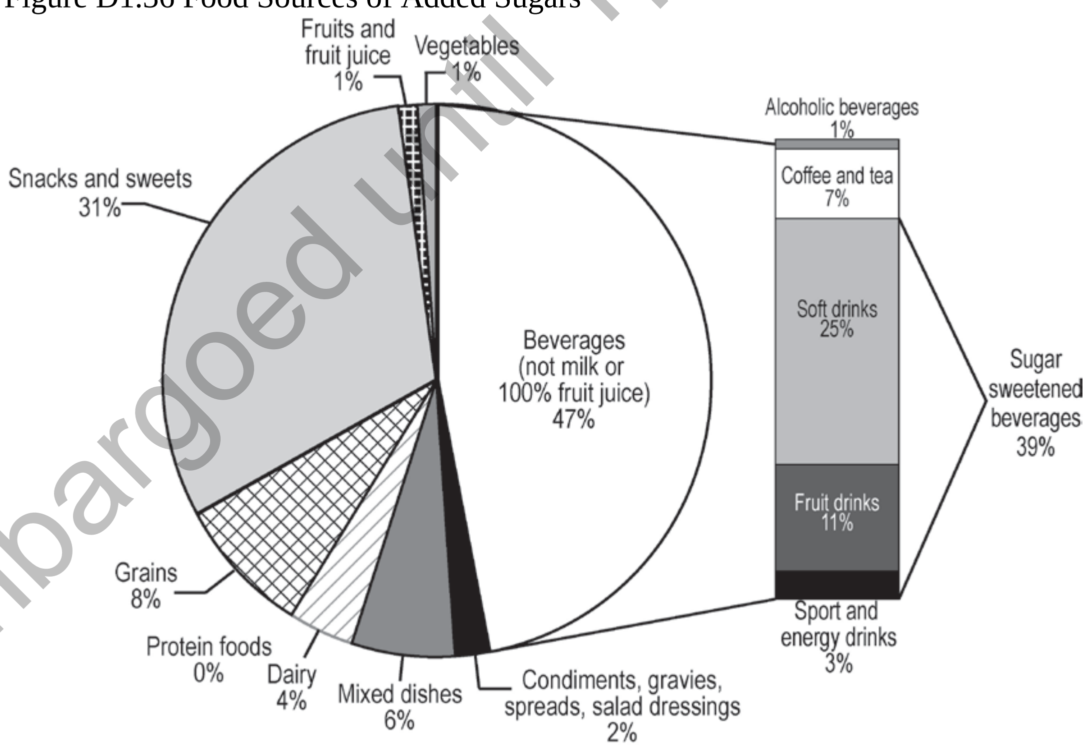
Source: What We Eat in America, NHANES 2009-2010

Figure D1.35 Food Sources of Sodium



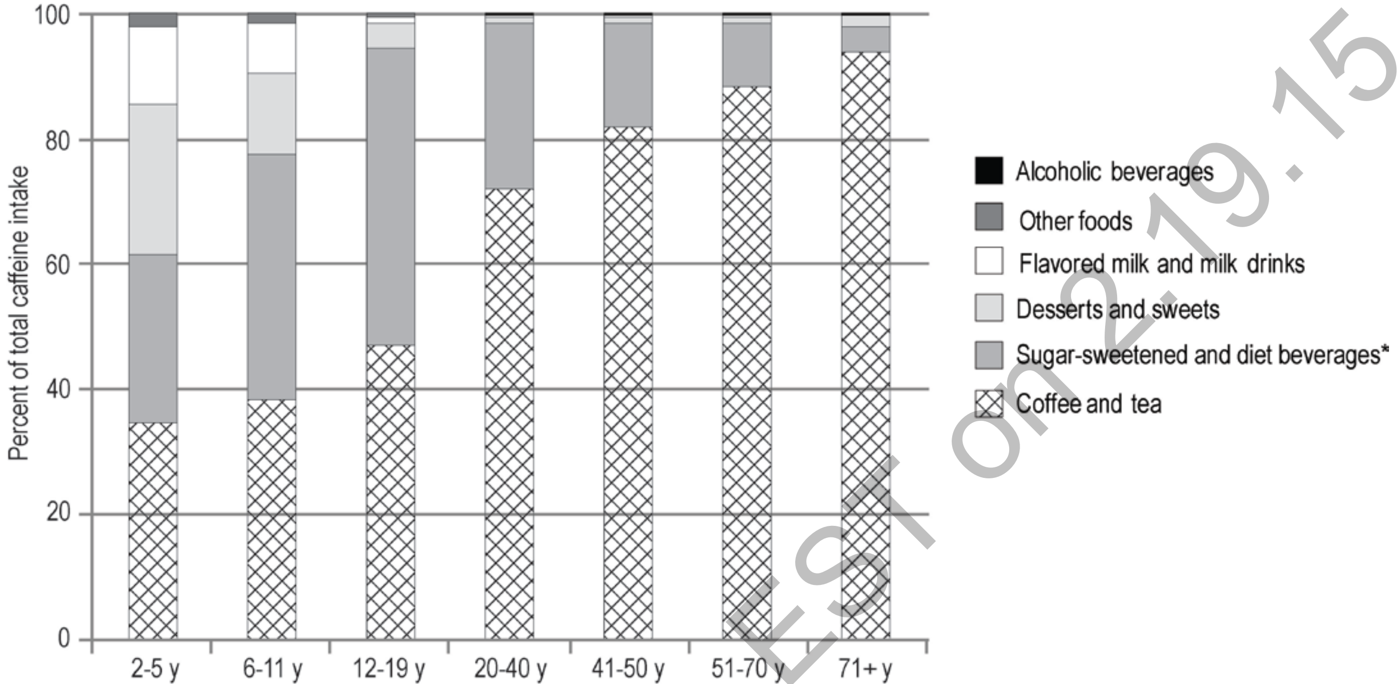
Source: What We Eat in America, NHANES 2009-2010

Figure D1.36 Food Sources of Added Sugars



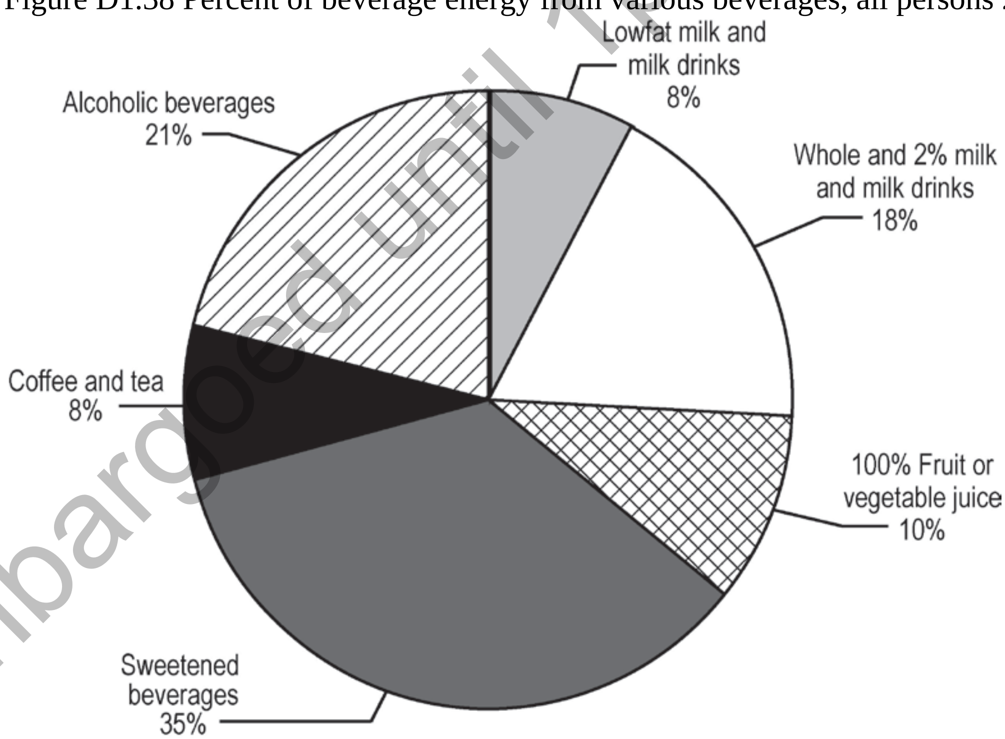
Source: What We Eat in America, NHANES 2009-2010

Figure D1.37 Caffeine sources by age group



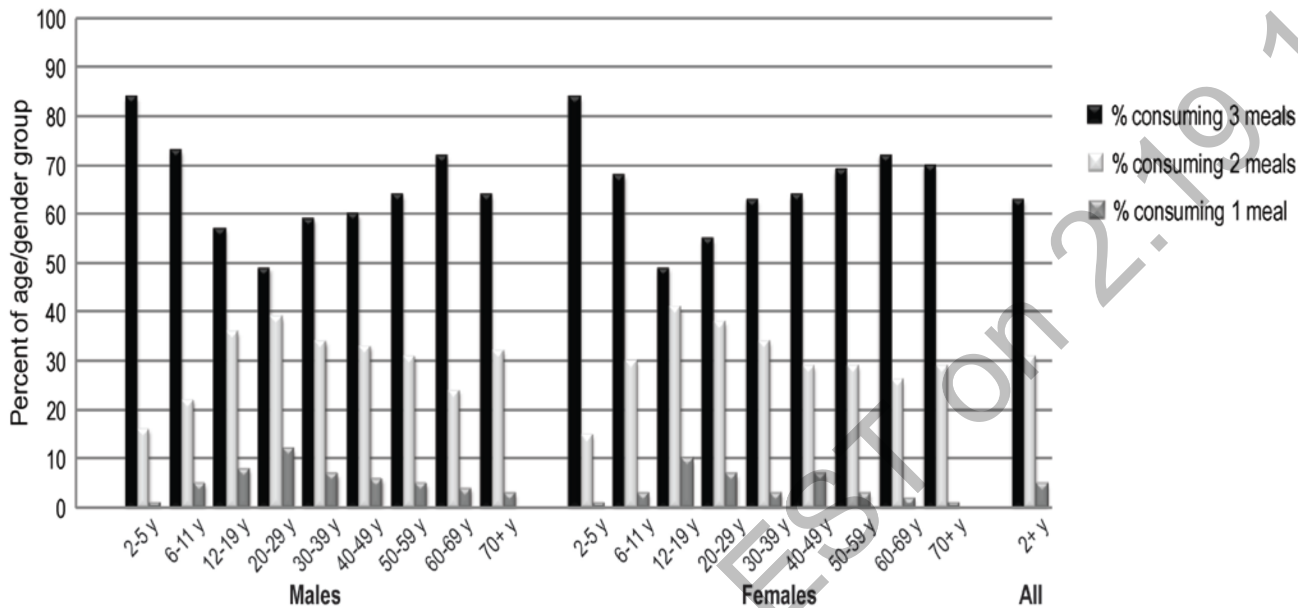
Source: What We Eat in America, NHANES 2009-2010

Figure D1.38 Percent of beverage energy from various beverages, all persons 2+



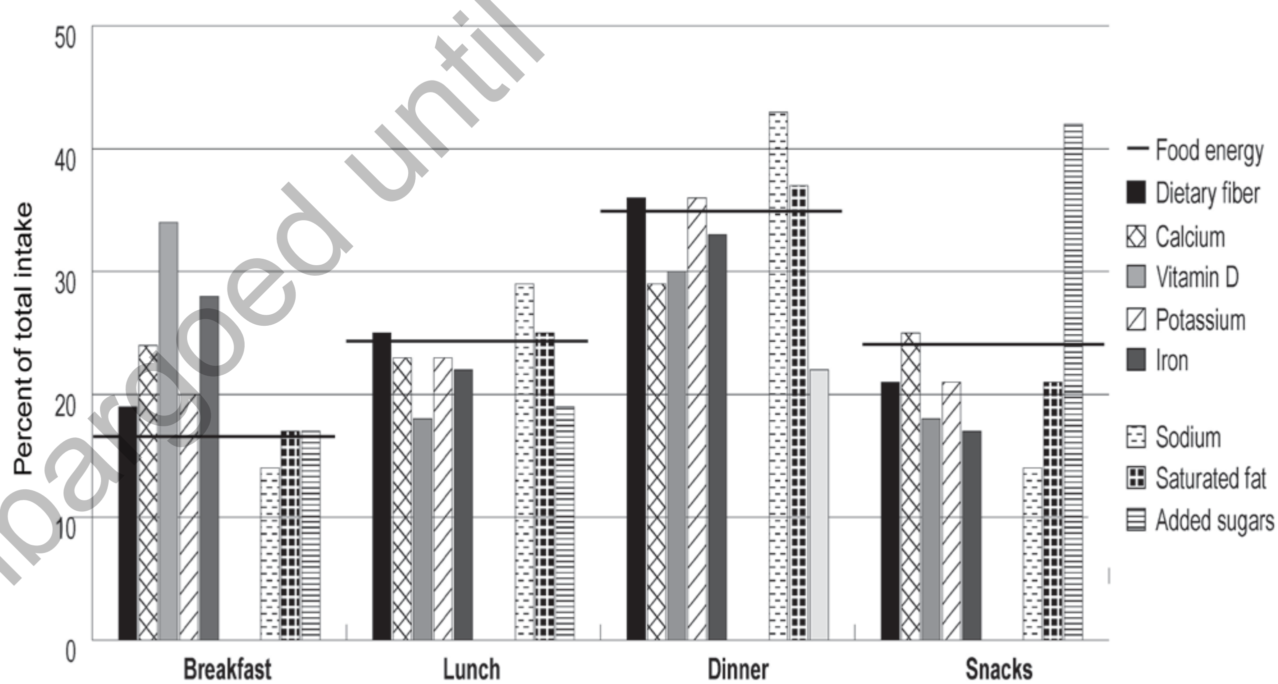
Source: What We Eat in America, NHANES 2009-2010

Figure D1.39 Number of meals reported per day by age/sex group



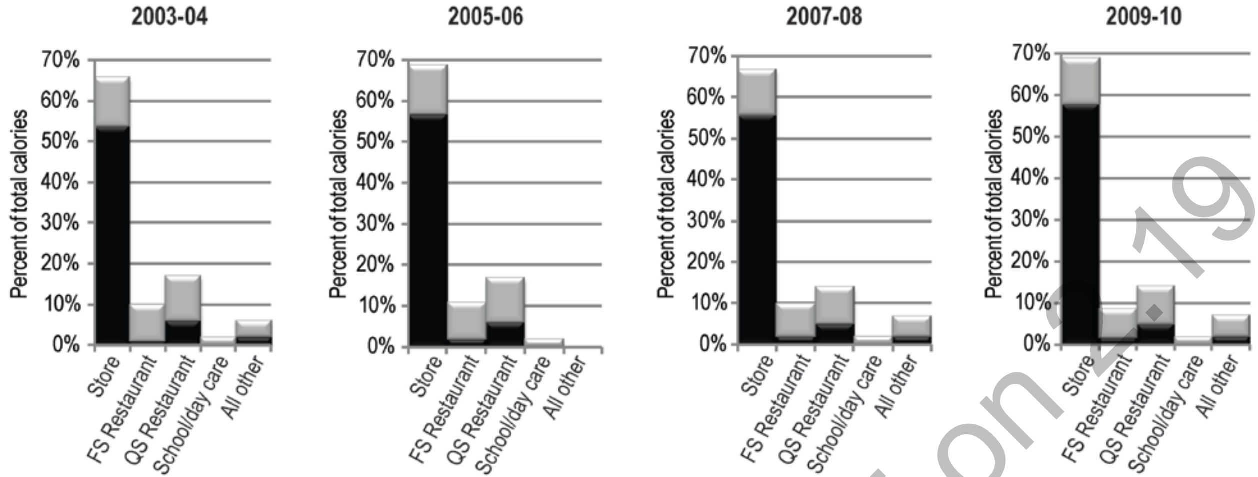
Source: What We Eat in America, NHANES 2009-2010

Figure D1.40 Percent of total daily intake of nutrients of concern from each eating occasion, for the population 2+



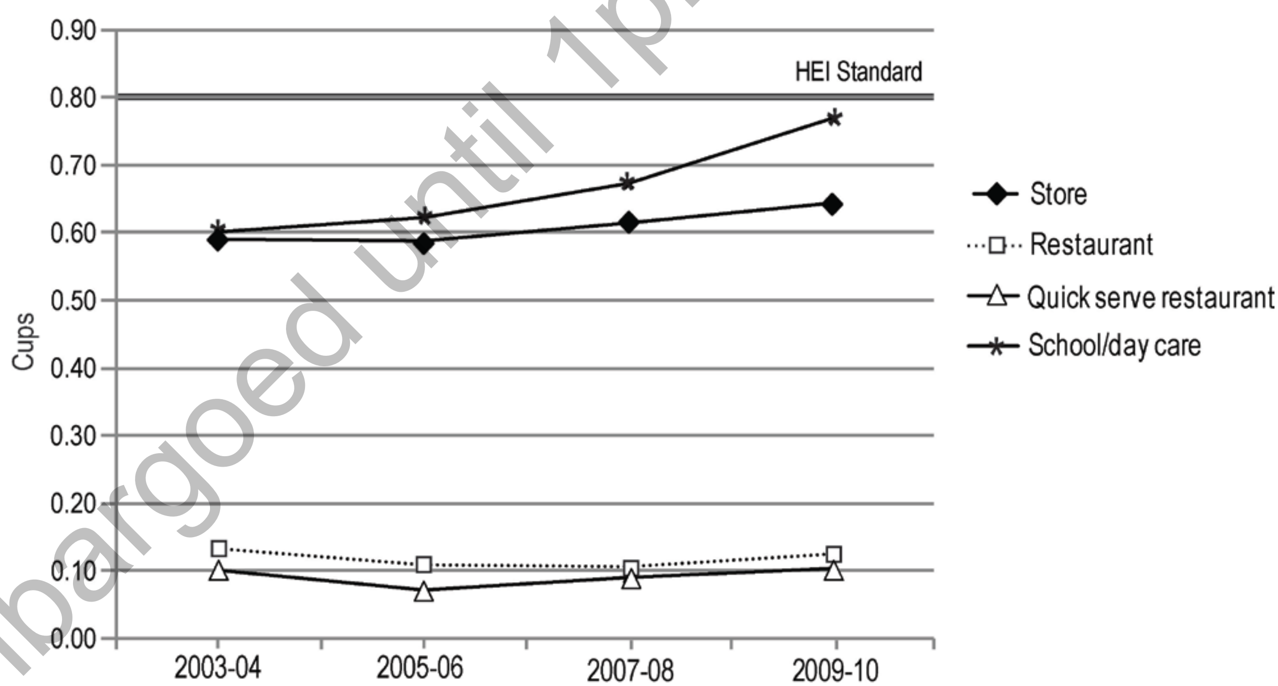
Source: What We Eat in America, NHANES 2009-2010

Figure D1.41 Percent of calories by where food was obtained and consumed



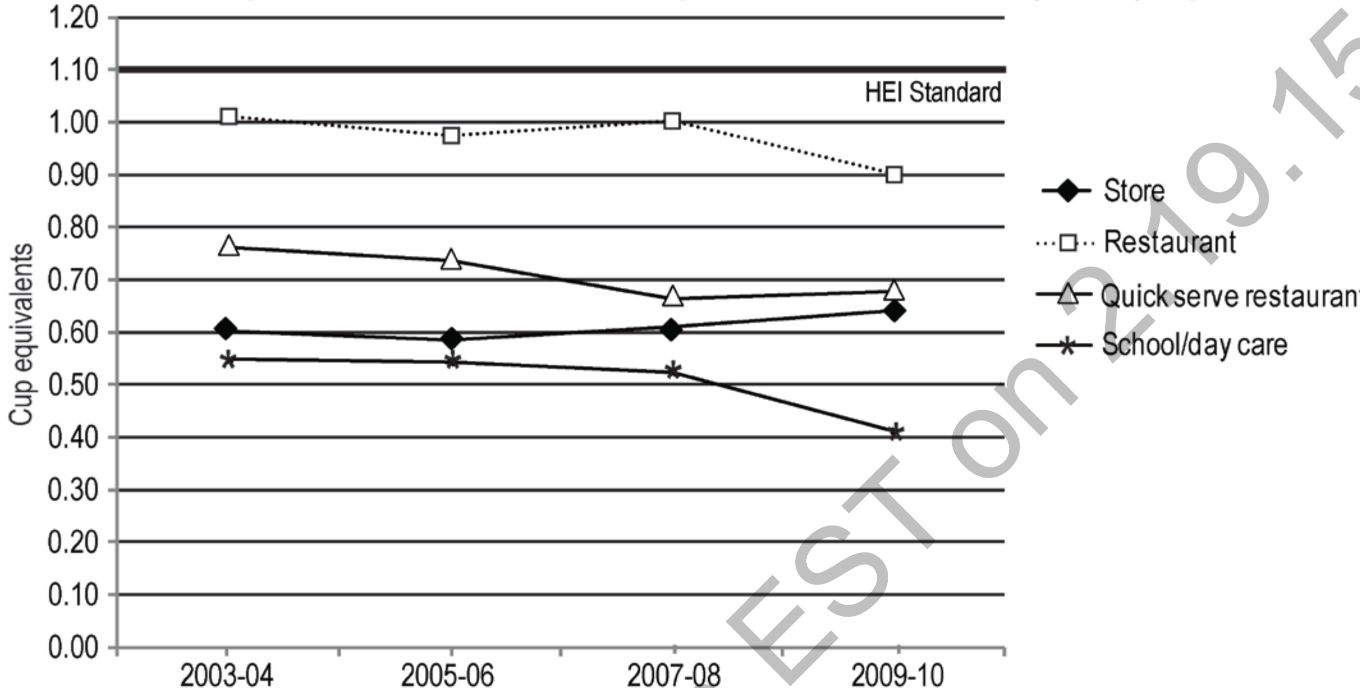
Darker shading indicates food eaten at home; lighter shading indicates food eaten away from home.
FS = Full Service (sit-down service); QS = Quick Service (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.42 Fruit group density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the fruit group.



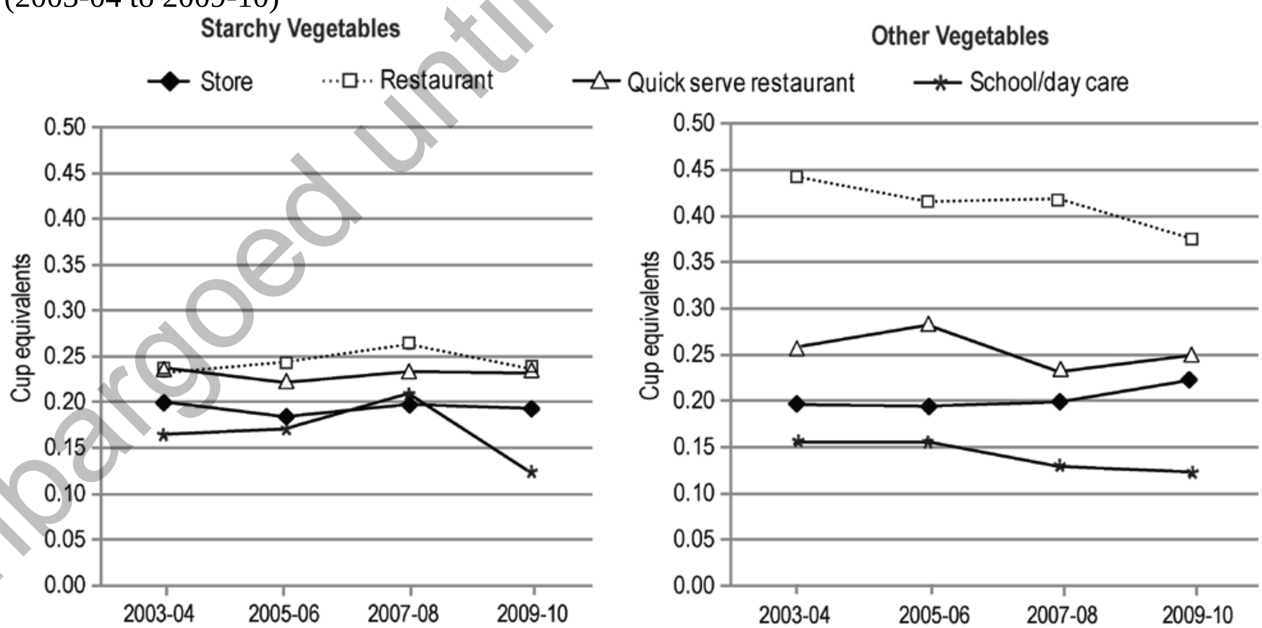
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.43 Vegetable density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the vegetable group



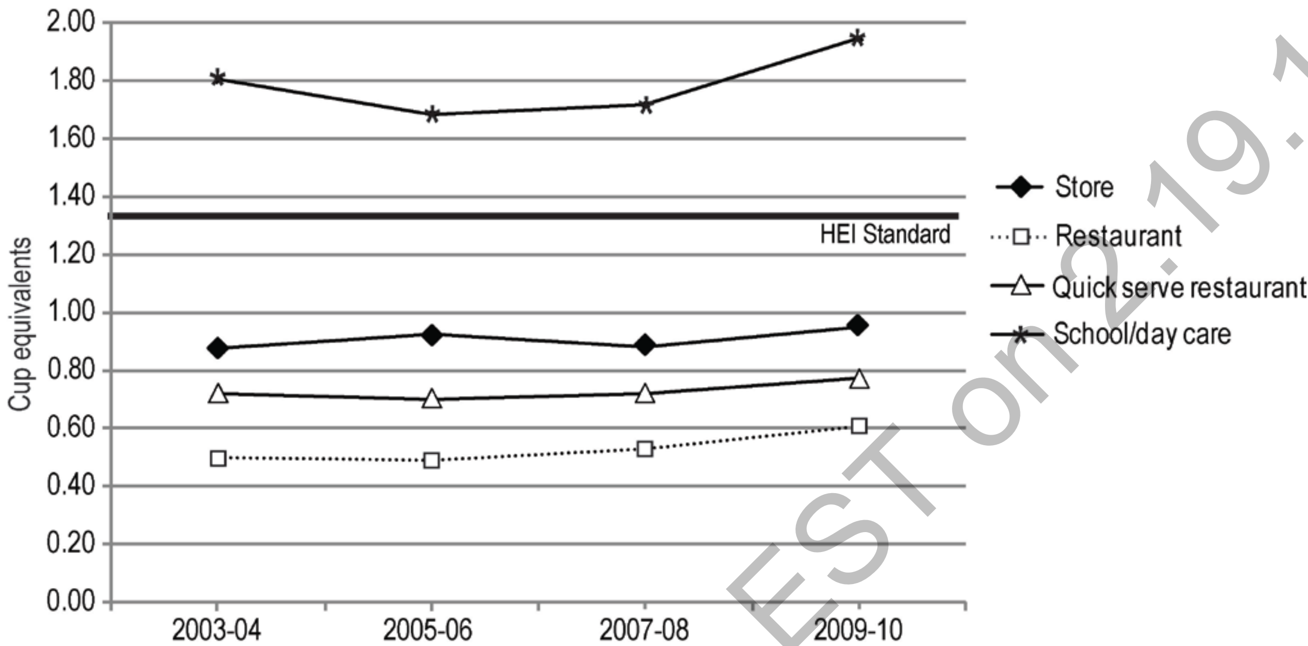
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.44 Vegetable subgroup density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10)



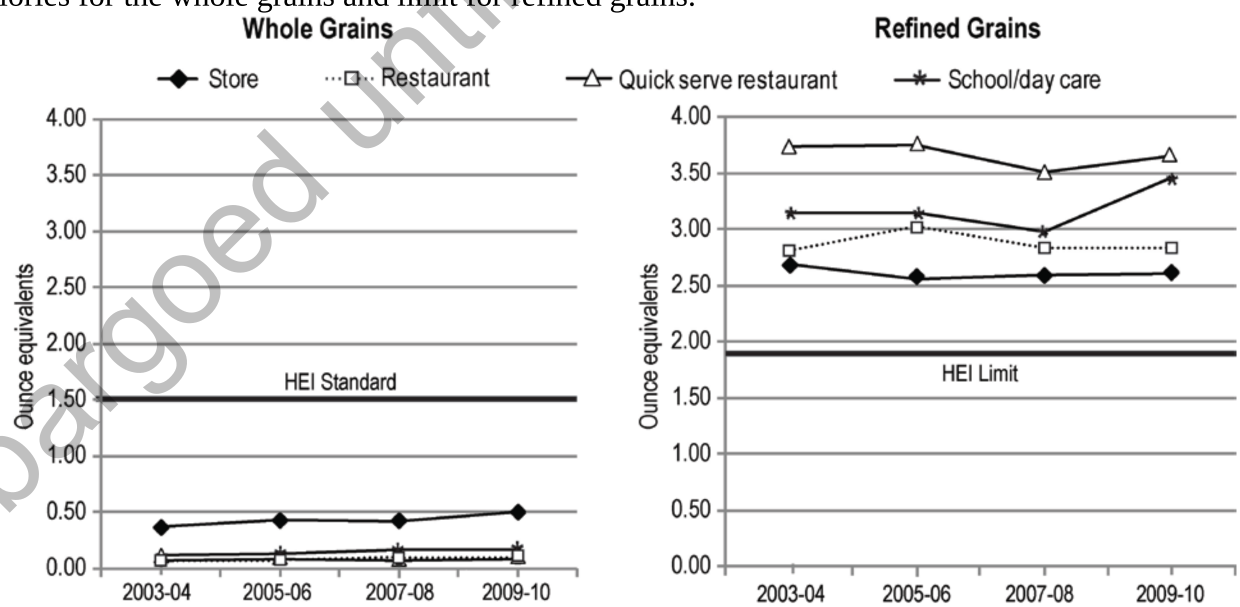
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.45 Dairy group density: cups per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the dairy group



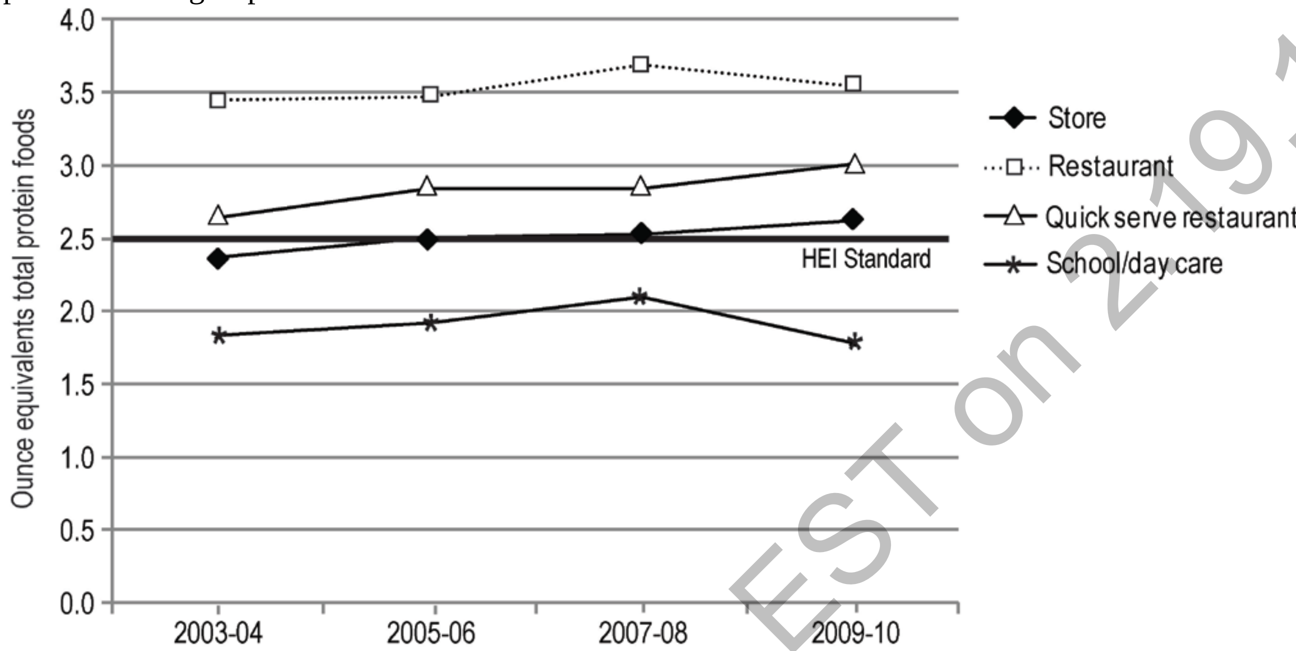
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.46 Grain group density (whole and refined) : ounce eqs per 1000 calories by where obtained over time (2003-04 to 2009-10) in comparison to the 2010 HEI standard per 1000 calories for the whole grains and limit for refined grains.



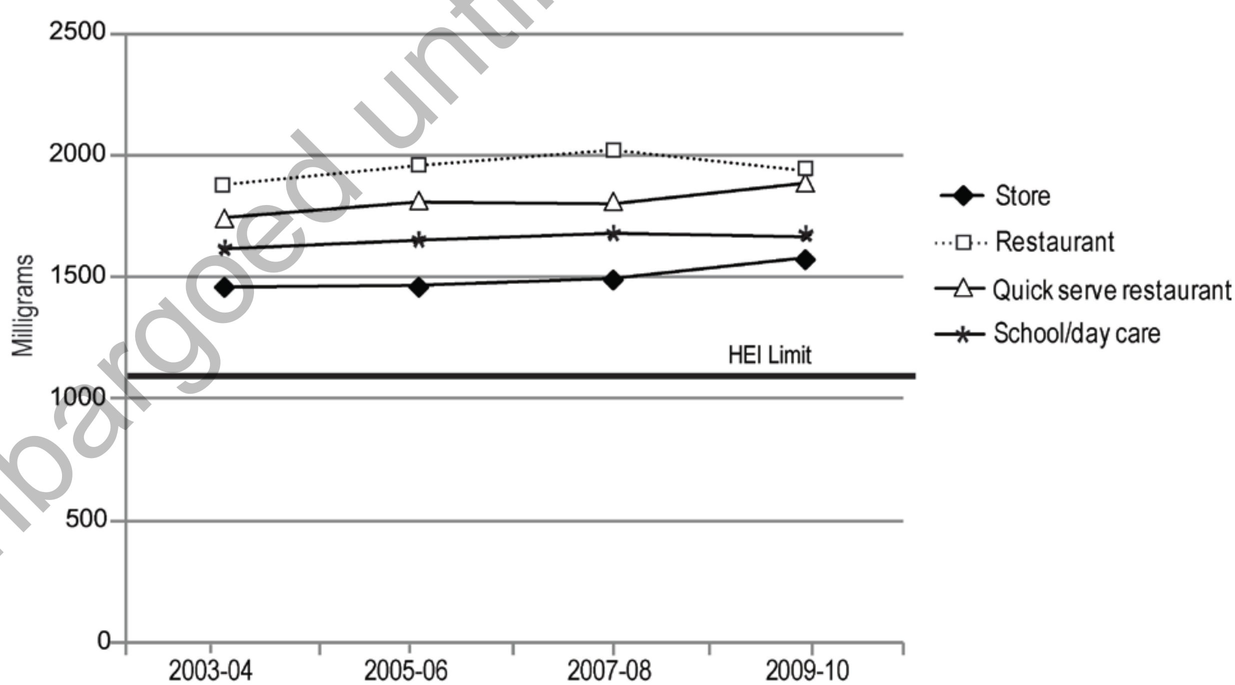
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.47 Protein Foods Group density: ounce eqs per 1000 calories by where obtained, over time (2001-04 vs. 2007-10) in comparison to the 2010 HEI standard per 1000 calories for the protein foods group.



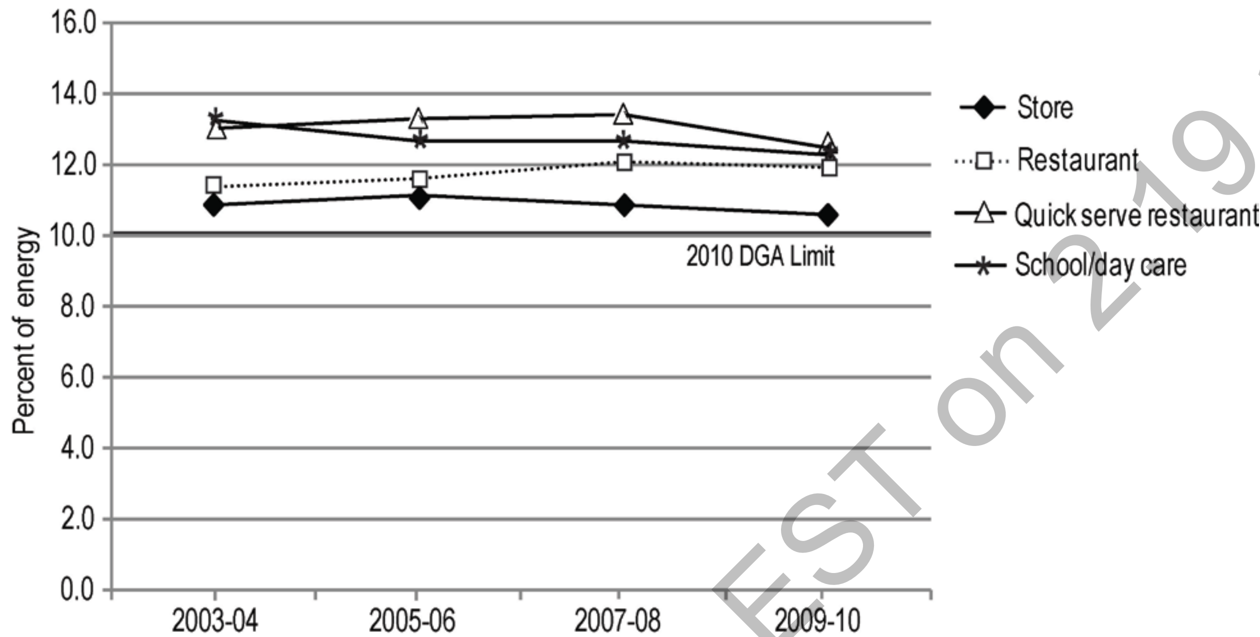
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.48 Sodium density: milligrams per 1000 calories by where obtained, over time (2003-04 to 2009-10) in comparison to the 2010 HEI limit per 1000 calories for sodium.



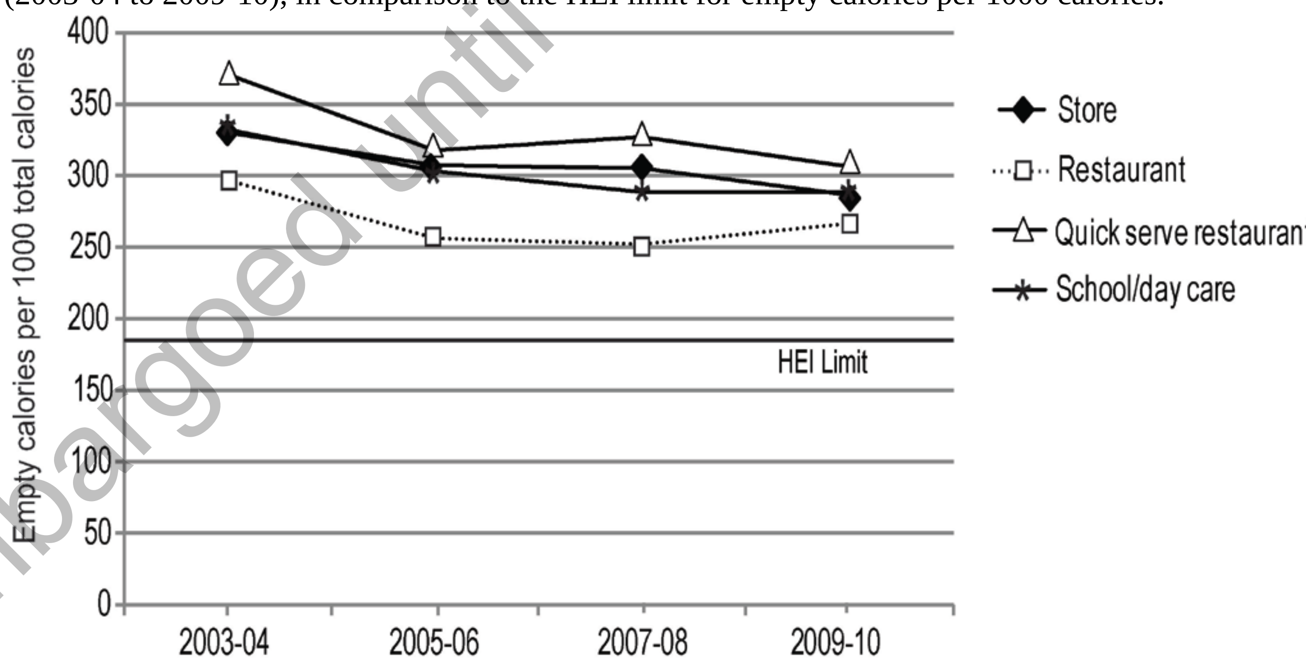
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.49 Saturated fat density: percent of energy by where obtained, over time (2003-04 to 2009-10), in comparison to the 2010 DGA limit for saturated fat as a percent of energy.



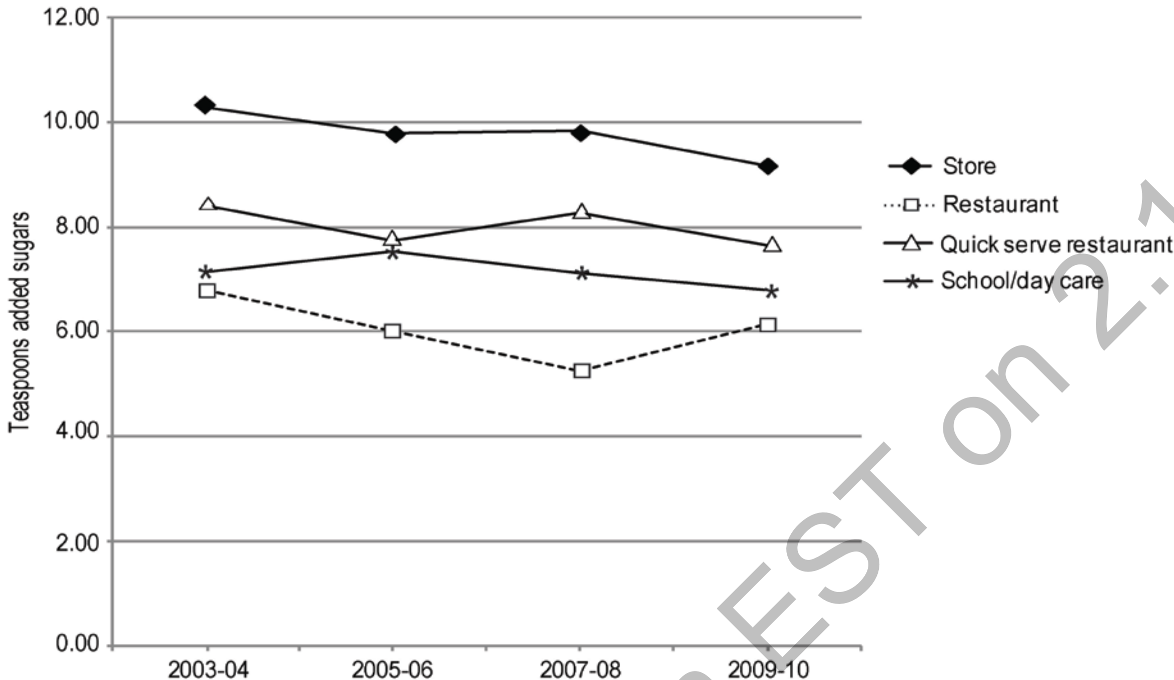
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.50 Empty calorie density: calories per 1000 calories by where obtained, over time (2003-04 to 2009-10), in comparison to the HEI limit for empty calories per 1000 calories.



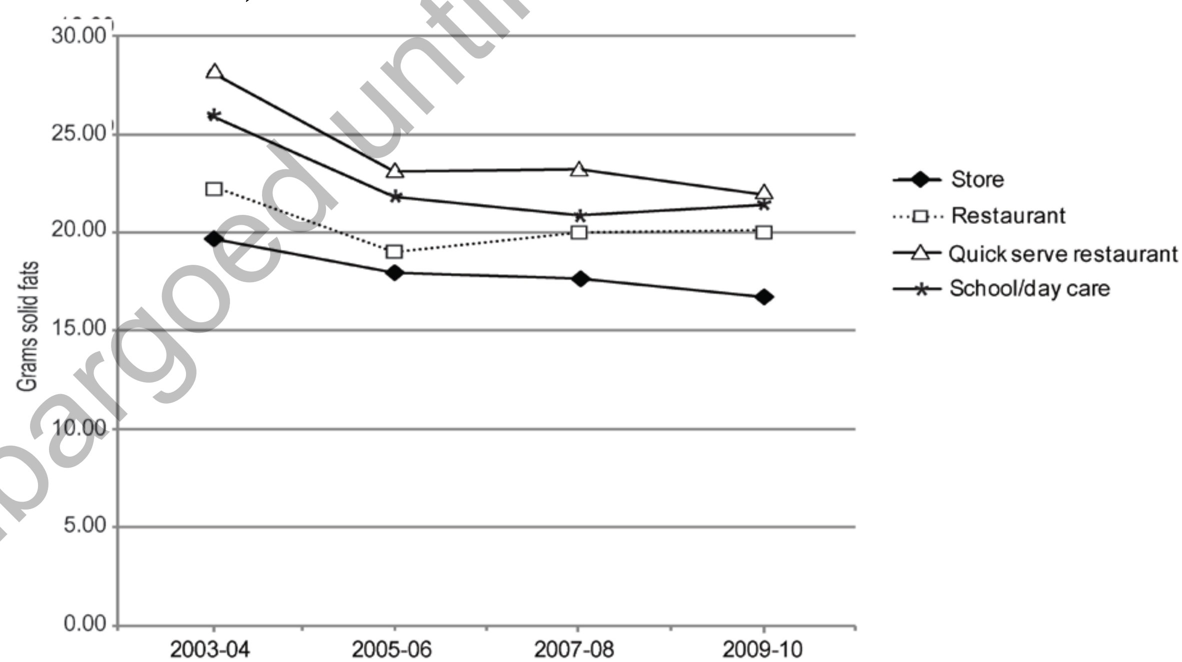
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.51 Added sugars density: Added sugars per 1000 calories by where obtained, over time (2003-04 to 2009-10)



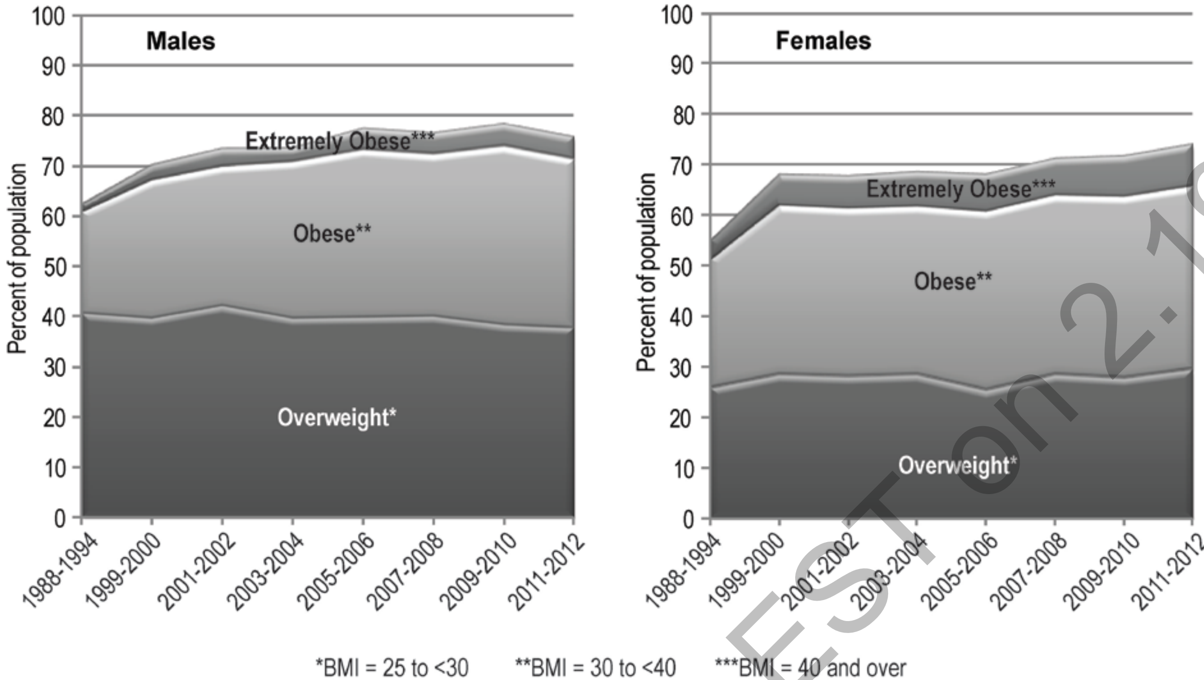
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.52 Solid fats density: Solid fats per 1000 calories by where obtained, over time (2003-04 to 2009-10)



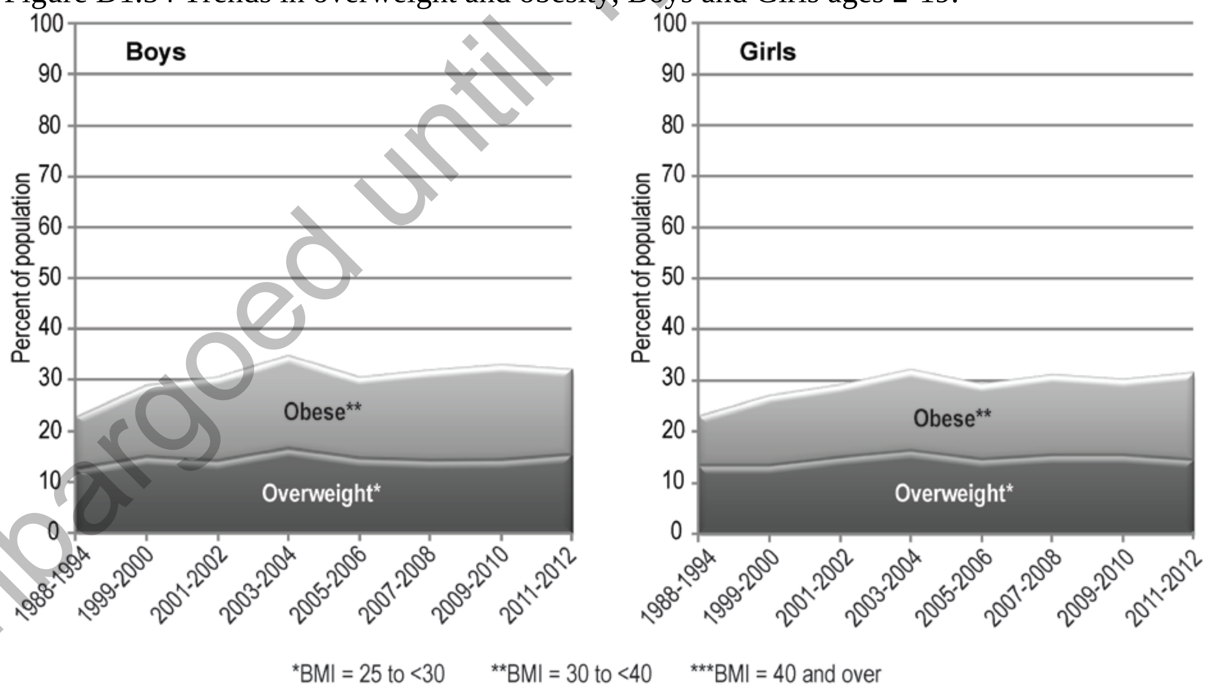
Restaurant = Full Service (sit-down service); Quick Serve = (fast food, food trucks, etc.)
Source: What We Eat in America, NHANES2003-2004, 2005-2006, 2007-2008, 2009-2010

Figure D1.53 Trends in overweight and obesity, Males and Females ages 20+.



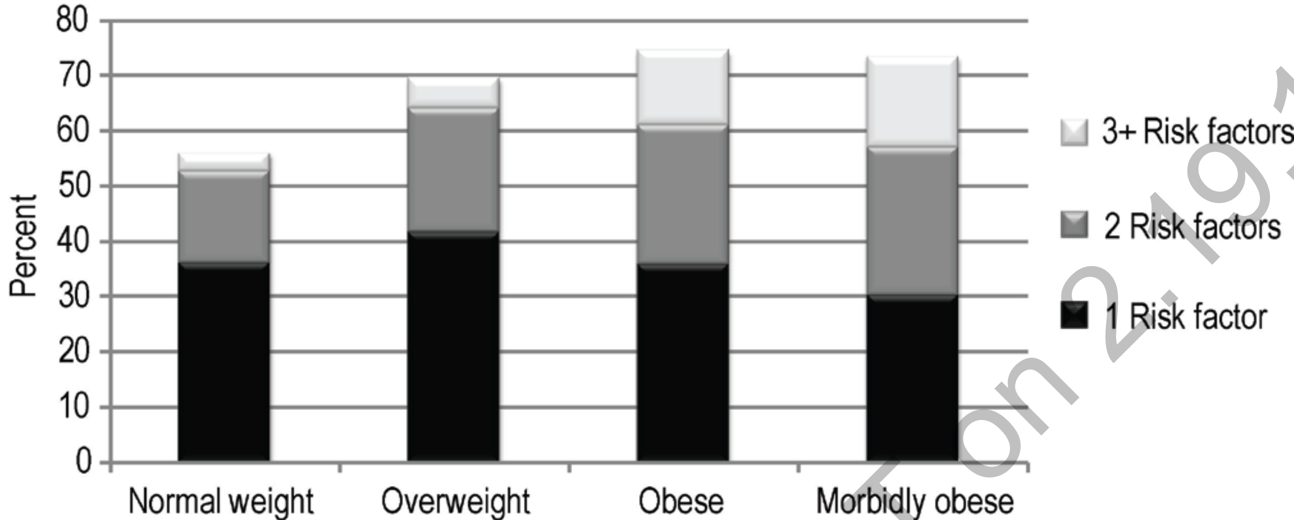
Source: Fryar, CD, Carroll, MD, Ogden, CL. Prevalence of Overweight and Obesity among Adults: United States, 1960–1962 Through 2011–2012. CDC/NCHS, the Health E-Stat, September 2014

Figure D1.54 Trends in overweight and obesity, Boys and Girls ages 2-19.



Source: Fryar, CD, Carroll, MD, Ogden, CL. Prevalence of Overweight and Obesity among Children and Adolescents: United States, 1963–1965 Through 2011–2012. CDC/NCHS, the Health E-Stat, September 2014

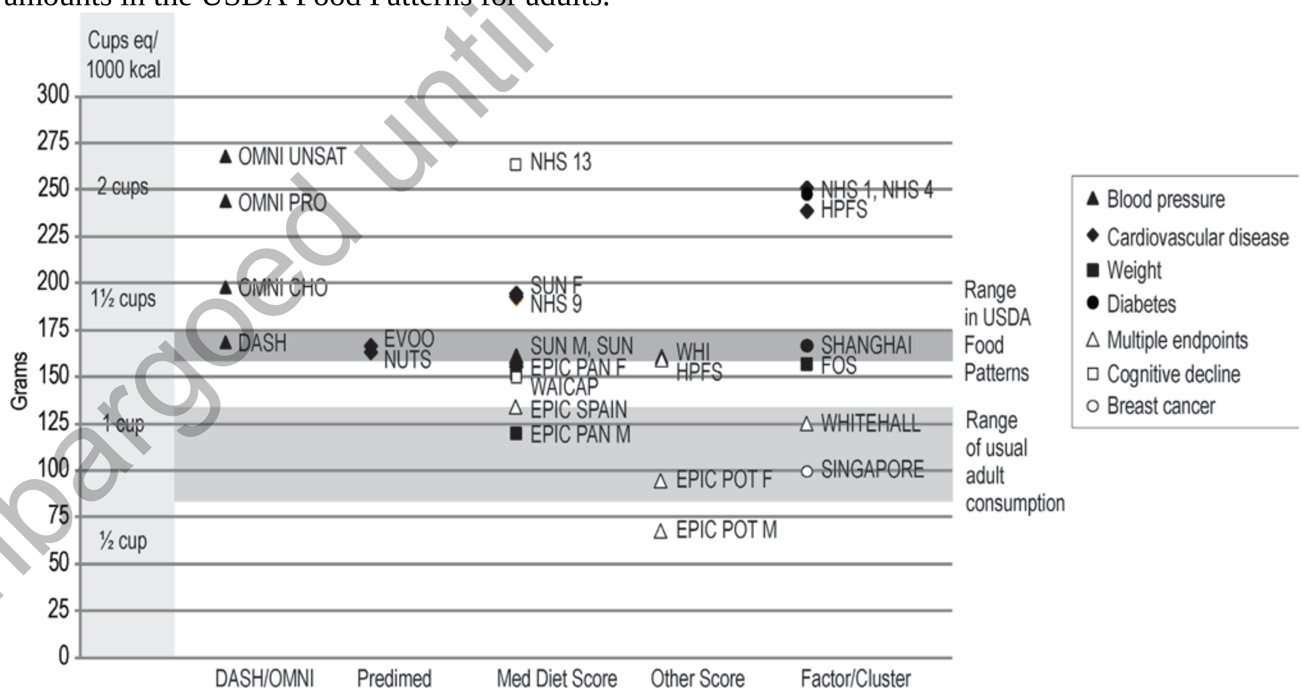
Figure D1.55 Prevalence and number of CVD risk factors by weight category, among adults 18 years and older, NHANES 2007-10.



Note: Risk factors included: total diabetes, total hypertension, total dislipidemia, and self reported smoking

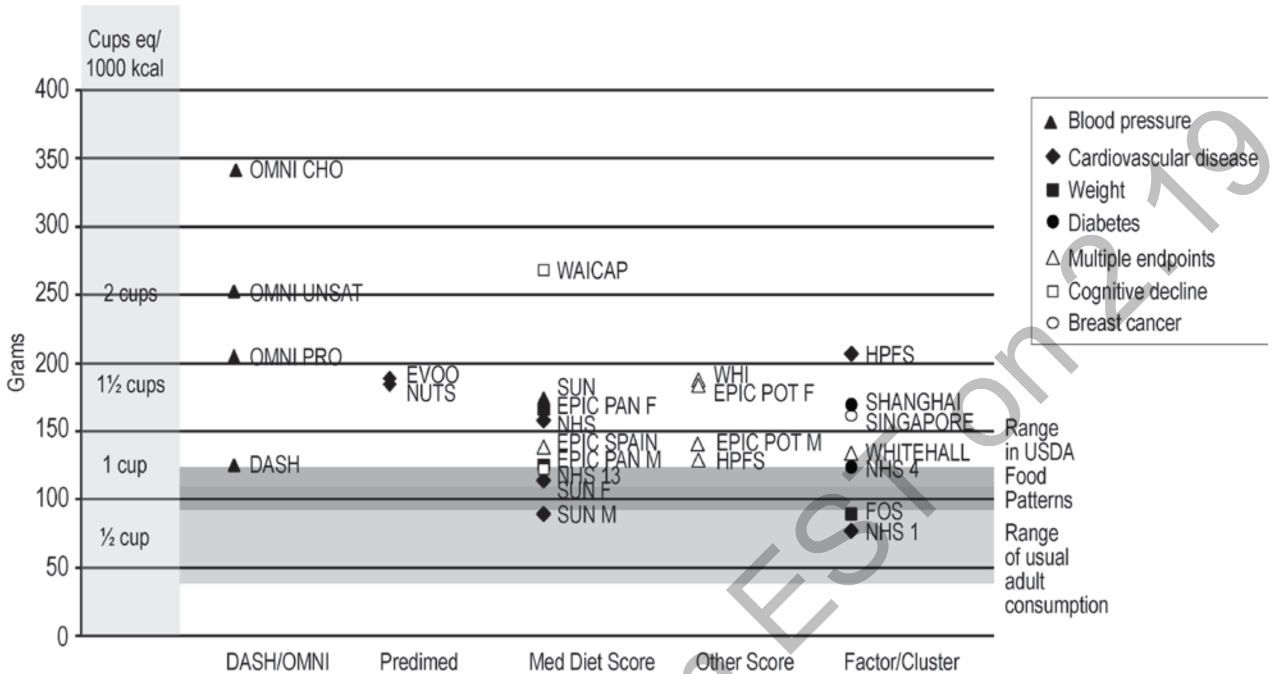
Source: Saydah S, Bullard KM, Cheng Y, Ali MK, Gregg EW, Geiss L, et al. Trends in cardiovascular disease risk factors by obesity level in adults in the United States, NHANES 1999-2010. Obesity (Silver Spring). 2014.

Figure D1.56 Vegetable intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual vegetable intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



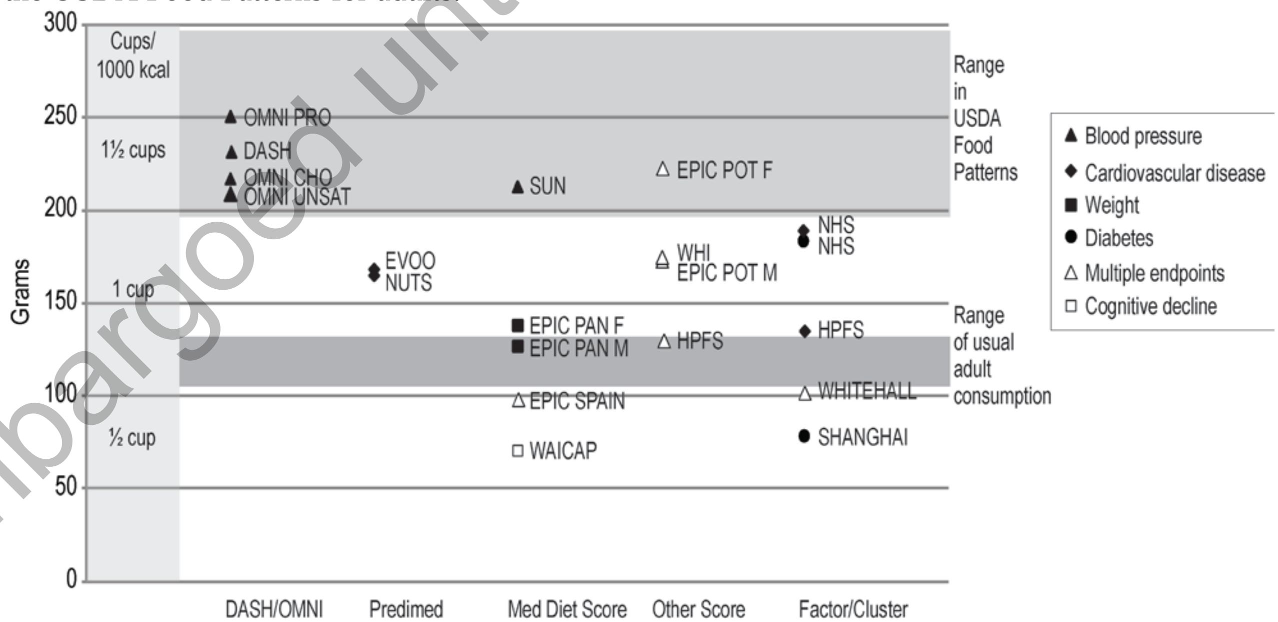
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31.

Figure D1.57 Fruit intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual fruit intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



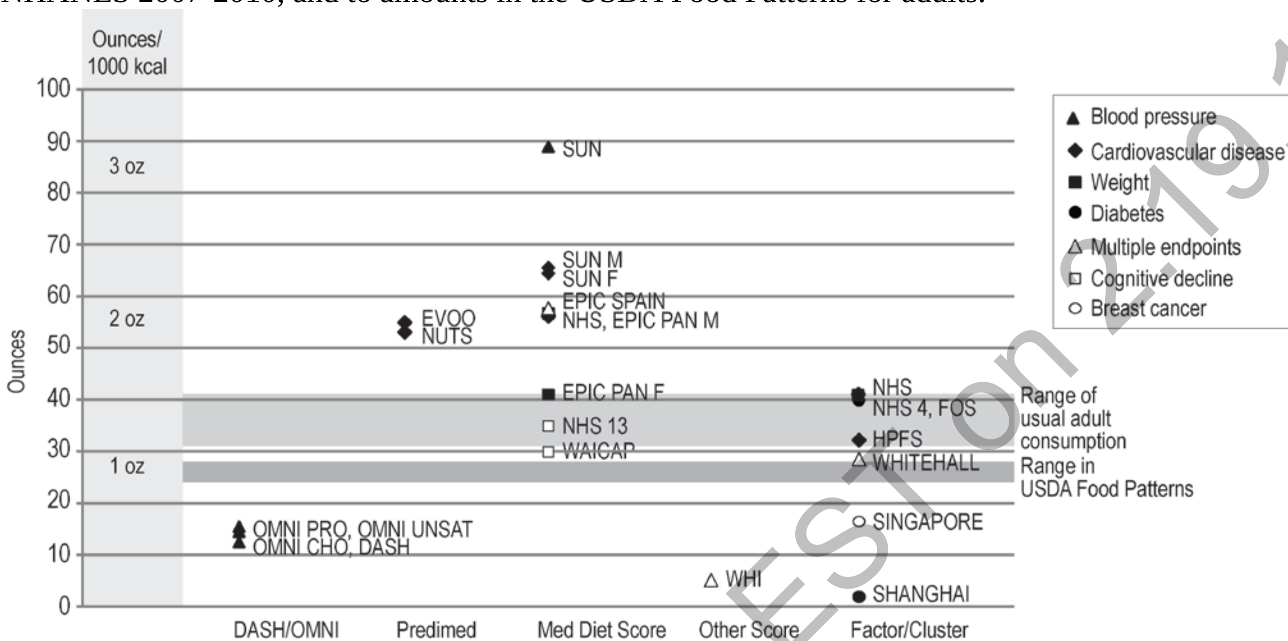
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31.

Figure D1.58 Dairy intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual dairy intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



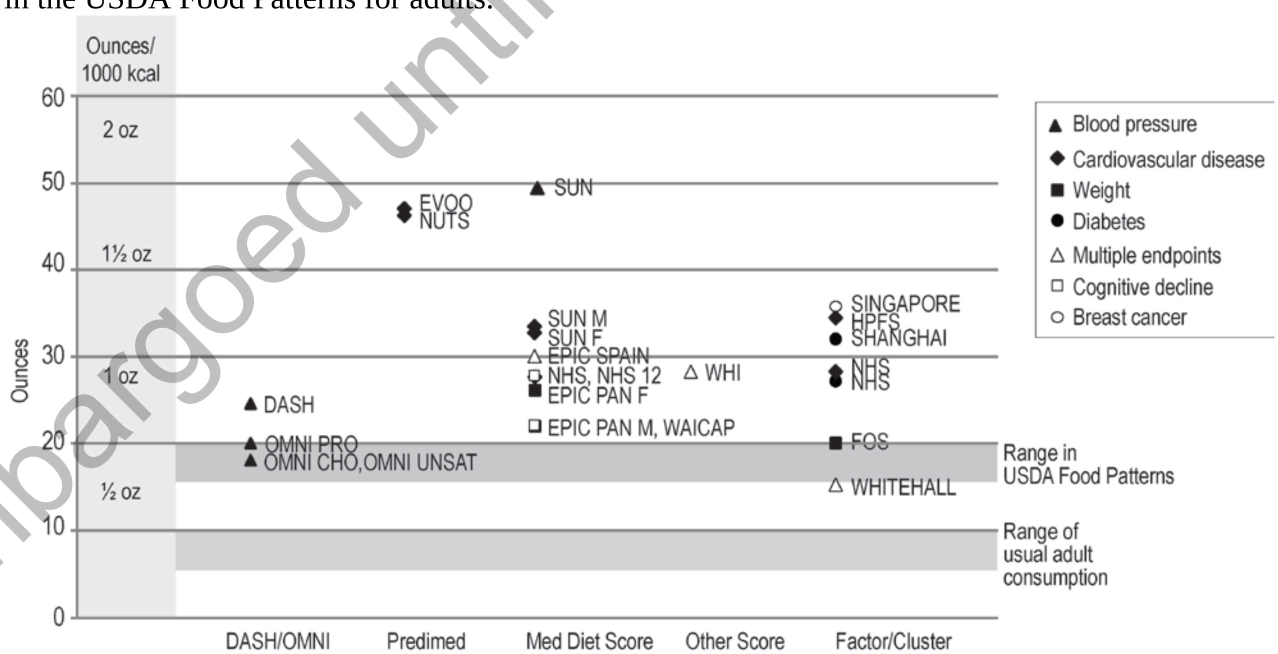
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31

Figure D1.59 Red and processed meat intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual red and processed meat intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



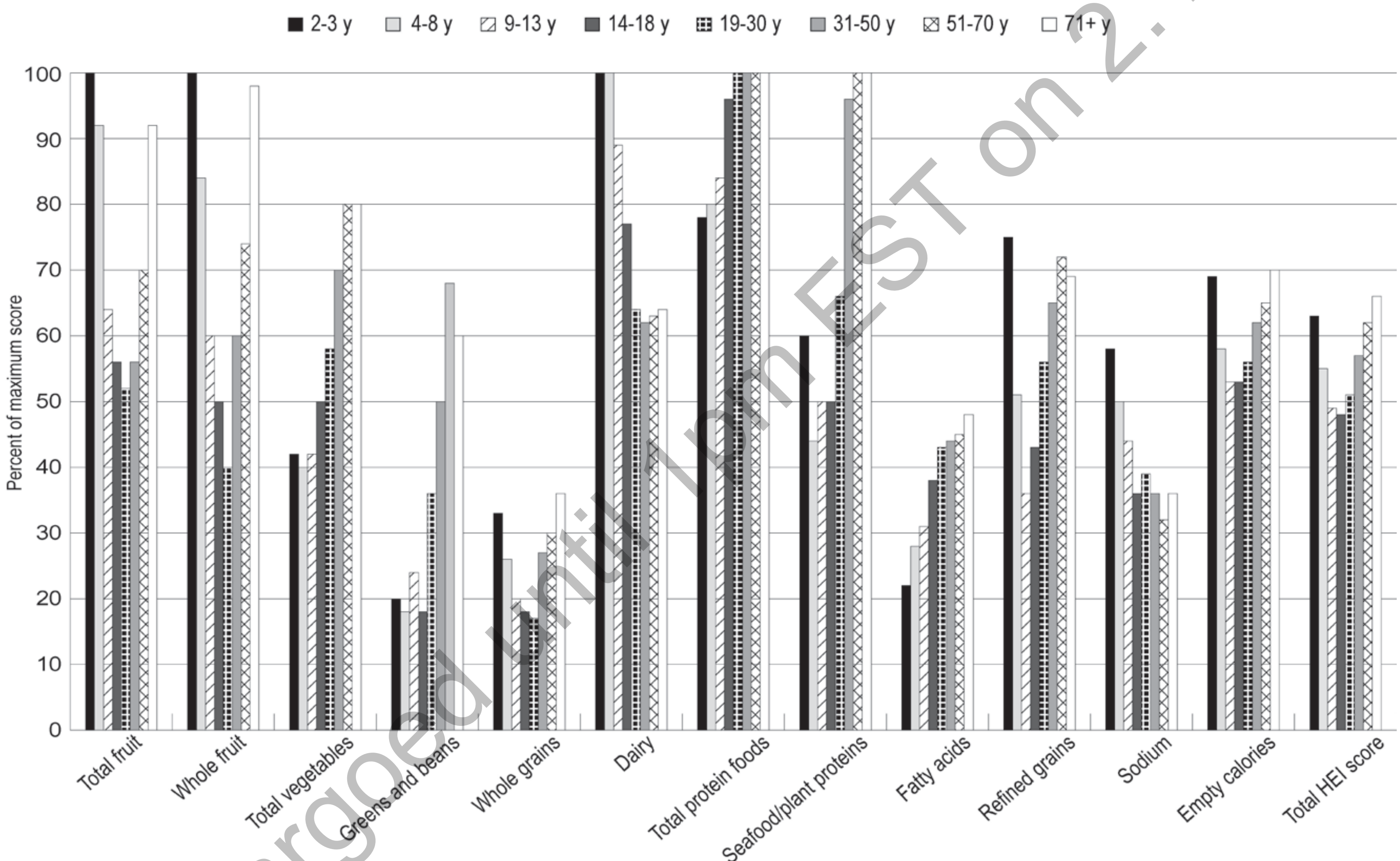
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31

Figure D1.60 Seafood intake (g/1000 calories) in dietary patterns identified as having health benefits, in comparison to usual seafood intake by adults, NHANES 2007-2010, and to amounts in the USDA Food Patterns for adults.



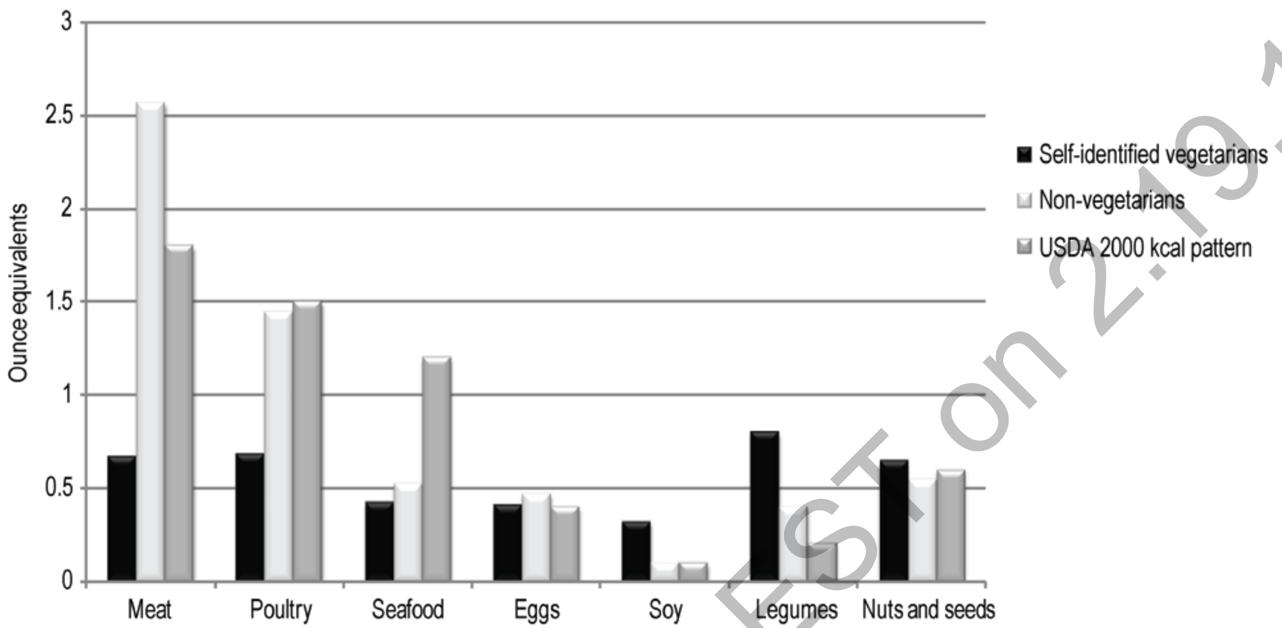
Source: USDA Food Patterns, What We Eat in America, NHANES 2007-2010, articles identified in table D1.31

Figure D1.61 Average HEI-2010 component scores for Americans by age group, 2009-10, as a percent of the total possible score for each component.



Source: HEI scores for Americans by age group, What We Eat in America, NHANES 2009-10 Appendix E2.x. Average Healthy Eating Index-2010 Scores for Americans ages 2 years and older

Figure D1.62 Intake from Protein Foods subgroups by self-identified vegetarians in comparison to non-vegetarian and to amounts in USDA Food Pattern at 2000 calories.



Source: Juan, WY, S. Yamini, P. Britten (2014) Food intake patterns of self-identified vegetarians among the U.S. population, 2007-2010. 38th Nutrient Data Bank Conference, May 2014 http://www.nutrientdataconf.org/PastConf/NDBC38/NNDC38_PosterAbstracts.pdf

Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes

INTRODUCTION

A healthy diet is a pillar of well-being throughout the lifespan. It promotes the achievement of healthy pregnancy outcomes; supports normal growth, development and aging; helps maintain healthful body weight; reduces chronic disease risks; and promotes overall health and well-being. Previous Dietary Guidelines Advisory Committees focused on examining specific foods, nutrients, and dietary components and their relationships to health outcomes. In its review, however, the 2010 DGAC noted that it is often not possible to separate the effects of individual nutrients and foods, and that the totality of diet—the combinations and quantities in which foods and nutrients are consumed—may have synergistic and cumulative effects on health and disease.[1] This approach has been adopted by others as well (e.g. American Heart Association, American College of Cardiology and the National Cancer Institute) and is being used by the 2015 DGAC. The 2010 Committee acknowledged the importance of dietary patterns and recommended additional research in this area. After the release of the *2010 Dietary Guidelines for Americans*, the USDA Nutrition Evidence Library (NEL) completed a systematic review project examining the relationships between dietary patterns and several health outcomes, including cardiovascular disease (CVD), body weight and type 2 diabetes.[2] Their report has been used by the 2015 DGAC.

As also noted in the *2010 Dietary Guidelines for Americans*, individuals can achieve a healthy diet in multiple ways and preferably with a wide variety of foods and beverages. Optimal nutrition can be attained with many dietary patterns and a single dietary pattern approach or prescription is unnecessary. Indeed, for long-term maintenance, a dietary pattern to support optimal nutrition and health should be based on the biological and medical needs as well as preferences of the individual.

Dietary patterns are defined as the quantities, proportions, variety or combinations of different foods and beverages in diets, and the frequency with which they are habitually consumed. Americans consume many habitual dietary patterns, rather than a "typical American pattern," which reflect their life experiences and wide-ranging personal, socio-cultural and other environmental influences. The nutritional quality of a dietary pattern can be determined by assessing the nutrient content of its constituent foods and beverages and comparing these characteristics to age- and sex-specific nutrient requirements and standards for nutrient adequacy, as shown in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** for the USDA Food Patterns, including the "Healthy U.S.-style Pattern," the "Healthy Mediterranean-style Pattern," and the "Healthy Vegetarian Pattern." Understanding the array of dietary patterns in a population and their nutrient quality allows a more complete characterization of individual eating behaviors and enables their examination in relationship with diverse health outcomes. For these reasons, the DGAC focused on considering the evidence for overall dietary patterns in addition to key foods and nutrients. A major goal was to describe the common characteristics of a healthy diet, which informed and is complementary to the quantitative description of dietary patterns provided in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***.

Dietary patterns can be characterized in three main ways, drawing from Dr. Susan Krebs-Smith's presentation to the DGAC during the second public meeting (available at www.DietaryGuidelines.gov). The first is by the use of an a priori index that is based on a set of dietary recommendations for a healthy dietary pattern as a result of scientific consensus or proposed by investigators using an evidence-based approach. An individual's index/score is derived by comparing and quantifying their adherence to the criterion food and/or nutrient component of the index and then summed up over all components. A population's average mean and individual component scores can be similarly determined. Examples of dietary quality scores include: the Healthy Eating Index (HEI)-2005 and 2010,[3] the Alternate HEI (AHEI) and updated AHEI-2010,[4] the Recommended Food Score (RFS),[5] the Dietary Approaches to Stop Hypertension (DASH) score,[6] the Mediterranean Diet Score (MDS),[7] and the Alternate Mediterranean Diet Score (aMed).[8]

The second method of dietary pattern assessment is through data-driven approaches, such as cluster analysis (which addresses the question, "Using the self-reported food and beverage intake data are there groups of people with distinct (non-overlapping) dietary patterns?") and factor analysis (which addresses the question, "Which components of the diet track together to explain variations in food or beverage intake across diet patterns?"). These data-driven approaches are outcome-independent. That is, the relationships between the dietary patterns and intermediate or longer-term health outcomes are examined once the patterns themselves are defined. Other data-driven approaches are outcome-dependent, such as reduced rank regression (which addresses the question, "What combination of foods explains the most variation in one or more intermediate health markers?").

The third method examines individuals' food and beverage intake preferences as they are commonly defined by foods included or eliminated. In cohort studies, this pattern is usually based upon qualitative self-reported behaviors rather than detailed questionnaires. Vegetarianism and its various forms (e.g., ovo-lacto vegetarianism) are examples of this type of dietary pattern.

The dietary patterns approach has a number of major strengths. The method captures the relationship between the overall diet and its constituent foods, beverages and nutrients in relationship to outcomes of interest and quality, thereby overcoming the collinearity among single foods and nutrients. In so doing, it considers the inherent interactions between foods and nutrients in promoting health or increasing disease risk. Because foods are consumed in combinations, it is difficult, if not impossible, to determine their separate effects on health. Relationships or effects attributed to a particular food or nutrient may be accurate or reflect those of other dietary components acting in synergy. The dietary pattern approach has advanced nutrition research by capturing overall food consumption behaviors and its quality in relationship to health.

Despite these considerable strengths, however, the approach has several limitations that are important to consider. First, the dietary assessment instruments used to define the dietary patterns (e.g., food frequency questionnaires [FFQ] and 24–hour or multi-day dietary recalls or records) are based upon self-report and may introduce levels of report bias that can attenuate diet-health relationships. The FFQ has been evaluated as a valid and reliable measure of usual food and nutrient intake. However, the extent to which data from FFQs are valid measures of dietary patterns is not well established. Second, dietary patterns are not uniformly defined by investigators and vary substantially from one study to the next even though studies may use the same nomenclature. This may hamper cross-study comparisons and limits reproducibility. Third, scoring algorithms used to evaluate dietary pattern adherence may differ and affect the results of studies examining specific health outcomes. Fourth, data-driven methods may not derive comparable patterns in different populations because these patterns may be population specific. Lastly, dietary patterns do not assess the frequency of meal and snack consumption, specific combinations of foods consumed together, and aspects of food purchase and preparation, all of which may influence the overall dietary pattern.

Another challenge to examining dietary patterns is that randomized dietary intervention studies have used different approaches for ensuring that subjects comply with the intervention diet when testing their relationships with health outcomes. For example, randomized controlled trials (RCTs), such as Prevencion con Dieta Mediterranean (PREDIMED), coached participants to follow a dietary pattern and provided them with key foods (e.g., olive oil or nuts) to facilitate adherence. In contrast, feeding studies (another form of intervention study), such as those conducted in the DASH and the Optimal Macronutrient Intake Trial for Heart Health (OmniHeart), provided all food to be consumed to each participant. These study designs across randomized trials and feeding studies provide strong evidence for the benefits and risks of particular dietary patterns because a prescribed intervention allows relatively precise definition of dietary exposures, and randomization helps ensure that any potential confounding variables are randomly distributed between study arms. However, some trials (i.e. DASH, OmniHeart) are necessarily restricted to testing a dietary pattern's effect on an intermediate outcome or a surrogate endpoint, such as blood lipids, because of the complexities involved in maintaining dietary compliance over long study duration. Additionally, the feeding trials fail to represent what happens in real world situations. Thus, well-conducted observational cohort studies provide an important evidentiary complement to RCTs because they enable the study of hard endpoints for disease in addition to intermediate outcomes and often provide a wider range of exposures for study.

Dietary patterns and their food and nutrient characteristics are at the core of the conceptual model that has guided the DGAC's work (see ***Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence***), and the relationship of dietary patterns to health outcomes is the centerpiece of this chapter. The Committee considered evidence about the relationship of diet with several health outcomes that are listed as major public health outcomes of concern in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***. Several of these outcomes—CVD, overweight and obesity, type 2 diabetes, congenital anomalies, and bone health—also were addressed by the 2010 DGAC. Others—cancers (lung, colon, prostate and breast) and neurological and psychological illness—while previously addressed, are considered here in more depth and represent an expanded list of health outcomes for which there is growing evidence of a diet-disease relationship. The 2015 Committee was not able to consider the relationship between dietary patterns during the peri- and prenatal period and pregnancy outcomes (e.g., birth weight, preterm birth, pregnancy complications) or other cancer outcomes, such as total cancer mortality or gynecological, pancreatic, and gastric-esophageal cancers due to time limitations and limited work done in these areas involving dietary patterns. However, it is important to note that recently the NIH-AARP Diet and Health Study (n = 492,823) conducted in the United States demonstrated that high adherence on several indices (the HEI-2010, the AHEI-2010, the aMED, and DASH) was associated with lower risk of overall CVD and cancer mortality.[9] The authors concluded that this finding provides further credence for using the dietary pattern approach, indicating that multiple dietary indices reflecting core tenets of a healthy diet may lower the risk of mortality outcomes.[9]

Over the course of the DGAC's review, when strong or moderate evidence related to dietary patterns and a particular health outcome was available, the Committee focused its discussion on dietary patterns and, as possible, highlighted the most consistent common food and nutrient characteristics identified in the dietary patterns literature. When only limited or insufficient evidence related to dietary patterns and a particular health outcome was available (as in the case of congenital anomalies and neurological and psychological illnesses), the Committee summarized these findings and also provided a brief summary of existing evidence on specific foods and/or nutrients and selected health outcomes.

In addition to its work on dietary patterns, the DGAC considered conducting an evidence review on the relationship between the role of the microbiome and various health outcomes. This novel area of research has generated considerable interest in the scientific community and the lay public. Investigators are examining the diversity of organisms (i.e., microbes) that inhabit different parts of the body such as the gut, mouth, skin, and vagina, and are attempting to understand how the microbial communities are influenced by diet, environment, host genetics and other microbes, as well as their association with various health outcomes. The DGAC conducted an exploratory search but did not find sufficient evidence to address this question in the 2015 report. However, the Committee considers the microbiome to be an emerging topic of potential importance to future DGACs.

LIST OF QUESTIONS

Dietary Patterns and Cardiovascular Disease

1. What is the relationship between dietary patterns and risk of cardiovascular disease?

Dietary Patterns and Body Weight

2. What is the relationship between dietary patterns and measures of body weight or obesity?

Dietary Patterns and Type 2 Diabetes

3. What is the relationship between dietary patterns and risk of type 2 diabetes?

Dietary Patterns and Cancer

4. What is the relationship between dietary patterns and risk of cancer?

Dietary Patterns and Congenital Anomalies

5. What is the relationship between dietary patterns and risk of congenital anomalies?

Dietary Patterns and Neurological and Psychological Illnesses

6. What is the relationship between dietary patterns and risk of neurological and psychological illnesses?

Dietary Patterns and Bone Health

7. What is the relationship between dietary patterns and bone health?

METHODOLOGY

For the first time, the 2015 DGAC included a chapter focusing solely on the relationship between dietary patterns and health outcomes. Although the 2010 DGAC considered some research on certain dietary patterns and specific health outcomes, notably body weight, they did not complete

NEL systematic reviews on this research. The 2015 DGAC began by acknowledging a desire to continue and expand on the total diet approach initiated by the 2010 DGAC. They then identified outcomes of public health concern on which to focus their reviews.

For the purposes of the 2015 DGAC, dietary patterns were defined as the quantities, proportions, variety or combinations of different foods and beverages in diets, and the frequency with which they are habitually consumed. Because the purpose of the Dietary Guidelines is to develop food-based recommendations to promote health and reduce risk of diet-related disease, one of the key aspects of the research that the DGAC considered was a description of the foods and beverages consumed by participants in the studies that the Committee reviewed. This was particularly important for the NEL systematic reviews, for which a description of foods and beverages was a key criterion for inclusion. Data on nutrients were not required for inclusion, but were considered when provided as part of the dietary pattern description.

Self-reported food and beverage intake was typically assessed using a qualitative or semi-quantitative food intake questionnaire (i.e., FFQ). However, some studies used other methods, such as 24-hour recalls. When reviewing the evidence, the Committee attempted to adhere to the language used by the study authors in describing food groupings. There was variability across the food groupings, and this was particularly apparent in the meat group; for example, "total meat" may have been defined as "meat, sausage, fish, and eggs," "red meat, processed meat, and poultry," or various other combinations of meat. Similarly, "vegetables" seemed to most often exclude potatoes, but some studies included potatoes, yet they rarely provided information on how the potatoes were consumed (e.g., fried versus baked). When reported in the studies, the Committee considered these definitions in their review.

Because of the variability in dietary patterns methodology and food groupings reported, the Committee focused on providing a qualitative description of healthy dietary patterns. Additionally, as most studies reported intake in relative terms (e.g., comparing the first and fifth quintiles or across tertiles), the Committee has presented its conclusions with relative terminology (e.g., "higher" and "lower" in a certain component). Quantitative information on dietary patterns is provided in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** as part of the Dietary Patterns Composition section.

A number of studies in the scientific literature describe diets based on macronutrient proportion or test only a specific food group or nutrient in the diet. For example, a low-carbohydrate diet fits this description and has been of public interest. The DGAC reviewed the body of evidence related to this type of diet as part of Question 2. Additionally, the Committee examined the results of exploratory searches on low-carbohydrate diets (defined as less than 45 percent of calories from carbohydrate) and all of the health outcomes considered in this chapter published since 2000. Overall, it appears that only limited evidence is available to address the relationship between low-carbohydrate diets and health, particularly evidence derived from U.S.-based populations. The most evidence available focuses on low-carbohydrate diets and body weight. The 2010 DGAC examined the relationship between macronutrient proportion and various body weight outcomes, concluding that:

> *"1) There is strong and consistent evidence that when calorie intake is controlled, macronutrient proportion of the diet is not related to losing weight; 2) A moderate body of evidence provides no data to suggest that any one macronutrient is more effective than any other for avoiding weight re-gain in weight reduced persons; 3) A moderate body of evidence demonstrates that diets with less than 45% of calories as carbohydrates are not more successful for long-term weight loss (12 months). There is also some evidence that they may be less safe. In shorter-term studies, low-calorie, high-protein diets may result in greater weight loss, but these differences are not sustained over time; and 4) A moderate amount of evidence demonstrates that intake of dietary patterns with less than 45% calories from carbohydrate or more than 35% calories from protein are not more effective than other diets for weight loss or weight maintenance, are difficult to maintain over the long term, and may be less safe."*

The published literature since that review does not provide sufficient evidence to change these conclusions. Thus, in summary, although studies that examine macronutrient proportion or that test only a specific food group or nutrient are important, they answer different questions related to diet and health than those proposed by the DGAC. In addition, these studies generally did not meet the DGAC's definition of a dietary pattern study unless a full description of the dietary pattern consumed was provided and appropriate methods were used to adjust for the confounding of foods and nutrients.

Questions 1, 2, and 3 were answered using existing reports, systematic reviews, and meta-analyses. All three of these questions were addressed in the NEL Dietary Patterns Systematic Review Project. This project was supported by USDA's Center for Nutrition Policy and Promotion and was informed by a Technical Expert Collaborative of experts in dietary patterns research.[2] Additionally, the DGAC reviewed reports from systematic reviews recently conducted by the National Heart, Lung, and Blood Institute (NHLBI) that included dietary patterns research. For Question 1, the DGAC used the NHLBI *Lifestyle Interventions to Reduce Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group*[10] and the associated American Heart Association (AHA)/ American College of Cardiology (ACC) *Guideline on Lifestyle Management to Reduce Cardiovascular Risk*.[11] For Question 2, the DGAC used the NHLBI *Managing Overweight and Obesity in Adults: Systematic Evidence Review from the Obesity Expert Panel*[12] and the associated AHA/ACC/The Obesity Society (TOS) *Guideline for the Management of Overweight and Obesity in Adults*.[13] For all three questions, in an attempt to capture new research published since the searches for these systematic reviews were completed, the Committee considered existing systematic reviews and meta-analyses published in peer-reviewed journals since 2008. The existing systematic reviews and meta-analyses considered by the DGAC had to meet the general inclusion criteria of the DGAC, and were required to consider dietary patterns and the outcomes of interest. A description of the process the DGAC used to answer existing report questions is provided in ***Part C: Methodology***. The DGAC followed this approach, including consideration of reference overlap, for all three questions. For more information on the existing reports, systematic reviews, and meta-analyses considered by the DGAC, the reader is encouraged to review the original sources, which are referenced within each evidence review.

Questions 4, 5, 6, and 7 were answered using NEL systematic reviews. A description of the NEL process is provided in ***Part C: Methodology***. All reviews were conducted in accordance with NEL methodology, and the DGAC made all substantive decisions required throughout the process to ensure that the most complete and relevant body of evidence was identified and evaluated to answer each question. All steps in the process were documented to ensure transparency and reproducibility. Specific information about individual systematic reviews can be found at www.NEL.gov, including the search strategy, inclusion and exclusion criteria, a complete list of included and excluded articles, and a detailed write-up describing the included studies and the body of evidence. A link for each question is provided following each evidence review.

Introductory sections were written for Questions 4, 5, 6, and 7 because the conclusion statements for these questions were graded limited or insufficient. The purpose of the introduction was to provide a brief description of the current evidence available related to foods and nutrients and the health outcome of interest. However, this evidence was not considered in developing the dietary pattern conclusion statements. During the course of the dietary pattern reviews, the DGAC chose to highlight particular components of the diet, which are discussed further in ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance***.

Question 1: What is the relationship between dietary patterns and risk of cardiovascular disease?

Source of evidence: Existing reports

Conclusion

The DGAC concurs with the conclusions of the NEL Dietary Patterns Systematic Review Project and AHA/ACC *Guideline on Lifestyle Management to Reduce Cardiovascular Risk* that strong and consistent evidence demonstrates that dietary patterns associated with decreased risk of CVD are characterized by higher consumption of vegetables, fruits, whole grains, low-fat dairy, and seafood, and lower consumption of red and processed meat, and lower intakes of refined grains, and sugar-sweetened foods and beverages relative to less healthy patterns. Regular consumption of nuts and legumes and moderate consumption of alcohol also are shown to be components of a beneficial dietary pattern in most studies. Randomized dietary intervention studies have demonstrated that healthy dietary patterns exert clinically meaningful impact on cardiovascular risk factors, including blood lipids and blood pressure. Additionally, research that includes specific nutrients in their description of dietary patterns indicate that patterns that are lower in saturated fat, cholesterol, and sodium and richer in fiber, potassium, and unsaturated fats are beneficial for reducing cardiovascular disease risk. **DGAC Grade: Strong**

Implications

Individuals are encouraged to consume dietary patterns that emphasize vegetables, fruits, whole grains, legumes, and nuts; include low-fat dairy products and seafood; limit sodium, saturated fat, refined grains, and sugar-sweetened foods and beverages; and are lower in red and processed meats. Multiple dietary patterns can achieve these food and nutrient patterns and are beneficial for cardiovascular health, and they should be tailored to individuals' biological needs and cultural as well as individual food preferences. The Committee recommends the development and implementation of programs and services at the individual and population levels that facilitate the improvement in eating behaviors consistent with the above dietary patterns.

Review of the Evidence

The DGAC examined research compiled in the NEL Dietary Patterns Systematic Review Project, which included 55 articles summarizing evidence from 52 prospective cohort studies and 7 RCTs, and the 2013 AHA/ACC Lifestyle Guideline and associated NHLBI Lifestyle Report, which included primarily RCTs. The Committee drew additional evidence and effect size estimates from six published systematic reviews/meta-analyses published since 2008 that included one or more studies not covered in the NEL or NHLBI Lifestyle reports.[14-19] In total, 142 articles were considered in these reports, of which 35 were included in two or more reviews. Little evidence on the contribution of dietary patterns to CVD risk factors in the pediatric populations was available, and that which was published was not systematically reviewed.

Most evidence examining hard disease endpoints comes from large, prospective cohort studies in adults using a priori scores to rank individuals with respect to adherence to dietary patterns of interest. Though the observational design allows the necessary duration of follow-up to observe CVD endpoints, comparison across studies was difficult because of different methods for deriving scores and different versions of scores measuring adherence to the same dietary pattern. In the Mediterranean dietary indices and the AHEI scores, moderate alcohol was included as a "positive" component (associated with potential benefits). Red and processed meats were "negative" (potentially detrimental) components in the Mediterranean scores, AHEI scores, and DASH. Certain scores also included sugars or sugar-sweetened beverages as negative components. Poultry was considered as a positive component in the original AHEI. Total high-fat dairy was a negative component in the Mediterranean diet scores, but dairy was a positive component when meeting recommended intakes for the HEI-2005, and low-fat dairy was positive in the DASH scores. As the NEL systematic review points out, several components of scores associated with decreased CVD risk recurred in multiple dietary patterns and were associated as part of scores and as individual components with reduced CVD risk. These included consumption of vegetables, fruits, whole grains, nuts, legumes, unsaturated fats, and fish.

The NHLBI Lifestyle Report summarized the evidence from two RCTs of the DASH dietary pattern and two trials testing DASH variations with differing levels of sodium or macronutrients. The diet provided to participants in standard DASH intervention trials was high in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, and nuts. It also was low in sweets, sugar-sweetened beverages, and reduced in (or lower in) red and processed meats. The DASH dietary pattern is high in fiber and potassium and low in sodium, saturated fat, total fat, and cholesterol. It is rich in potassium, magnesium, and calcium, as well as protein and fiber.

In contrast to the patterns described above, vegetarian diets were defined by what they excluded. Variations included: vegan (no meat, fish, eggs, or dairy); lacto-ovo vegetarian (includes eggs and dairy, but no fish or meat), and pesco-vegetarian (includes fish, but no meat) diets. The content of these diets varied substantially, though they tended to emphasize plant based foods, especially fruits and vegetables, legumes, nuts, and whole grains.

Dietary Patterns and Blood Pressure (BP)

DASH or DASH-style Dietary Patterns

The NEL systematic review and AHA/ACC Lifestyle Guideline conclude that strong and consistent evidence from RCTs demonstrates that compared to a dietary pattern that is relatively high in saturated fat and sodium and low in vegetables and fruits, the DASH-style dietary pattern reduced BP by approximately 6/3 mmHg (systolic blood pressure/diastolic blood pressure) across subgroups defined by sex, race, age, and hypertension status. The DASH trial provided all food to participants for 8 weeks. Fat intake was relatively low at 26 percent of energy (7 percent each monounsaturated and saturated, 10 percent polyunsaturated), compared to 36 percent in the control group. Carbohydrates accounted for 57 percent of energy and protein for 18 percent. Sodium was stable at 3,000 mg/day and body weight did not change. Variations of the DASH diet also lowered blood pressure: in the OmniHeart Trial, compared to the standard DASH, replacing 10 percent of calories from carbohydrate with either the same calorie content of protein or with unsaturated fat (8 percent MUFA and 2 percent PUFA) lowered systolic BP by 1 mmHg. Among adults with BP 140–159/90–95 mmHg, these substitutions lowered systolic BP by 3 mmHg relative to standard DASH.[2, 11]

Observational evidence summarized in the NEL report included one cohort showing that increased DASH score was associated with small, but decreased levels of systolic and diastolic

BP over time;[20] two others cohorts showed no relationship between DASH scores and risk of hypertension.[21, 22]

Mediterranean-Style Dietary Patterns

Several RCTs provide limited to moderate evidence on the benefits of a Mediterranean-style diet for reducing blood pressure. The AHA/ACC Lifestyle Guideline conclude that consuming a Mediterranean dietary pattern instead of a lower-fat dietary pattern had beneficial effects on blood pressure. The NHLBI Lifestyle Report reviewed two RCTs of free-living middle-aged or older adults (with type 2 diabetes or at least three CVD risk factors) in which a Mediterranean diet intervention reduced BP by 6–7/2–3 mmHg.[23, 24] The report also reviewed one observational study of healthy younger adults. Higher adherence to a Mediterranean-style diet, as measured through a Mediterranean score, was associated with a decrease in BP of 2–3/1–2 mmHg.[25]

Vegetarian Dietary Patterns

Evidence for the blood pressure benefits of vegetarian dietary patterns is more limited, but moderately consistent trends appear to exist. A recent meta-analysis of seven RCTs found that consumption of vegetarian diets was associated with a reduction in mean systolic blood pressure (-4.8 mm Hg; 95% CI = -6.6 to -3.1; $p<0.01$) and diastolic blood pressure (-2.2 mm Hg; 95% CI = -3.5 to -1.0) compared with the consumption of omnivorous diets.[19] The AHA/ACC Lifestyle Guideline did not find sufficient evidence to examine vegetarian dietary patterns, and the NEL systematic review summarized only three studies comparing blood pressure outcomes in lacto-ovo vegetarian diets versus non-vegetarian diets in which meat and fish were consumed. Of the two studies, one was a large prospective cohort that found no association with blood pressure,[26] and the other was a RCT among individuals with hypertension that demonstrated a decrease in systolic blood pressure, but not diastolic blood pressure.[27] The more recent EPIC-Oxford cohort found lower systolic, but not diastolic blood pressure compared to the findings of Crowe, 2013.[28]

Other Dietary Patterns

As summarized in the NEL systematic review, adherence to the *2005 Dietary Guidelines for Americans* was related to lower blood pressure in one study of healthy young adults. Zamora et al reported 20-year findings from the CARDIA study including 4,381 Black and White young adults.[29] Participants in the highest (vs. lowest) quartile of adherence to the 2005 Dietary Guidelines had significantly less increase in systolic and diastolic blood pressure over time.

Dietary Patterns and Blood Lipids

DASH or DASH-style Dietary Patterns

As reviewed in the NHLBI Lifestyle Report, RCTs of the DASH diet show favorable effects on low-density lipoprotein cholesterol (LDL-C) and total cholesterol: high-density lipoprotein cholesterol (total-C: HDL-C) ratio, and no effect on triglycerides (TG). Benefits were seen with a variety of different macronutrient compositions, though they were enhanced when some carbohydrates in the standard DASH pattern were replaced with protein or unsaturated fat. In the standard DASH, when food was supplied to adults with a total cholesterol level of less than 260 mg/dL and LDL-C less than 160 mg/dL, and body weight was kept stable, the DASH dietary pattern compared to the control diet decreased LDL-C by 11 mg/dL, decreased HDL-C by 4 mg/dL, and had no effect on TG. The OmniHeart trial tested the DASH dietary pattern with different macronutrient compositions among adults with average baseline LDL-C 130 mg/dL, HDL-C 50 mg/dL, and TG 100 mg/dL. Modifying the DASH diet by replacing 10 percent of calories from carbohydrate with 10 percent of calories from protein decreased LDL-C by 3 mg/dL, decreased HDL-C by 1 mg/dL, and decreased TG by 16 mg/dL compared to the DASH dietary pattern. Replacing 10 percent of calories from carbohydrate with 10 percent of calories from unsaturated fat (8 percent MUFA and 2 percent PUFA) decreased LDL-C similarly, increased HDL-C by 1 mg/dL, and decreased TG by 10 mg/dL compared to the DASH dietary pattern.[11]

Mediterranean-style Dietary Patterns

As with blood pressure, few trials have evaluated the effects of Mediterranean dietary patterns on blood lipids. According to the AHA/ACC Lifestyle Guideline, consuming a Mediterranean-style diet (compared to minimal or no dietary advice) resulted in no consistent effect on plasma LDL-C, HDL-C, and TG. In part, this was due to substantial differences in dietary interventions conducted among free-living middle aged or older adults with or without CVD or at high risk for CVD.[11] In the PREDIMED trial (reviewed in both the NHLBI Lifestyle and NEL reports), both treatment groups (Mediterranean diet +olive oil or +nuts) had favorable changes in HDL-C, total-C: HDL-C ratio and TG when compared to the control group, which received minimal advice to follow a lower-fat diet.[23] One of the prospective cohort studies reviewed by the NEL showed each one-point increase in alternate Mediterranean diet score assessed in adolescence and early adulthood was associated with a -6.19 (-10.44, -1.55) mg/dL lower total cholesterol in adulthood but no significant effects on HDL-C.[30] Of other observational cohorts reviewed, one reported adherence to a Mediterranean diet was associated with favorable changes in HDL-C and TG,[31] and another found no associations between adherence to a Mediterranean diet and blood lipids.[32]

Vegetarian Dietary Patterns

The NEL systematic review included three articles on vegetarian patterns that measured blood pressure or blood lipids.[26-28] One study reported decreased total-C[26] and another reported decreased non-HDL-C in vegetarian versus non-vegetarian participants.[28]

Other Dietary Patterns

Of note, adherence to the *2005 Dietary Guidelines for Americans* also was related to higher HDL-C levels in a cohort of Black and White young adults.[29]

Dietary Patterns and Cardiovascular Disease Outcomes

The NHLBI Lifestyle review did not include any trials examining the evidence of particular dietary patterns with CVD outcomes. Overall, the NEL systematic review found that individuals whose diets mirrored the dietary patterns of interest (typically compared with diets having lower scores) was associated with lower CVD incidence and mortality in 14 out of 17 studies. The studies were predominantly observational, but included some trial evidence, and they typically assessed dietary intakes through self-report. The effect sizes varied substantially, with the decrease in risk of CVD ranging from 22 to 59 percent for increased adherence to various Mediterranean-style dietary patterns and from 20 to 44 percent for increased adherence to a U.S. Dietary Guidelines-related pattern (e.g., HEI or AHEI and updates). The majority of studies that assessed coronary heart disease (CHD) incidence or mortality also reported a favorable association between adherence to a healthy dietary pattern and CHD risk. The lower CHD risk ranged from 29 to 61 percent for greater adherence to Mediterranean-style dietary patterns, from 24 to 31 percent for greater adherence to a U.S. Dietary Guidelines-related pattern, and from 14 to 27 percent for greater adherence to DASH. Similarly, the majority of studies assessing stroke incidence or mortality reported favorable associations, with the lower stroke risk ranging from 13 to 53 percent for greater adherence to a Mediterranean-style dietary pattern and from 14 to 60 percent for greater adherence to a U.S. Dietary Guidelines-related pattern.[2]

Mediterranean-style Dietary Patterns

To gather additional information on dietary patterns and CVD outcomes, the DGAC consulted two meta-analyses,[15, 18] which included many of the same observational prospective cohort studies as one another and as the NEL systematic review. These meta-analyses each reported summary estimates across studies as a 10 percent reduction in risk of CVD (fatal or nonfatal clinical CVD event) per 2-increment increase in adherence to the Mediterranean-style diet. The NEL report also included results from the largest Mediterranean diet trial, PREDIMED, which found that a Mediterranean diet (plus extra virgin olive oil or nuts) had favorable effects in high-risk participants compared to the control group who were advised to reduce dietary fat intake. An approximately 30 percent decrease in risk of major CVD events (a composite endpoint including myocardial infarction, stroke, and deaths) was observed and the trial was stopped early for meeting benefit requirements.[2, 33] According to food questionnaires measuring adherence to the assigned diet by the end of follow-up, the intervention groups had significantly increased consumption of fish and legumes and non-significant reductions in refined grains and red meat from baseline, in addition to increased intake of supplemental foods (olive oil or nuts depending on the intervention arm), compared to the control group.

DASH-style Dietary Patterns

A recent meta-analysis[17] of six prospective cohort studies with CVD endpoints assessed DASH-style diet through the Fung et al. method,[6] which assigns points based on population-specific quintiles of eight DASH dietary pattern components: fruits, vegetables, nuts and legumes, whole grains, low-fat dairy, sodium, red and processed meats, and sweetened beverages. This meta-analysis reported that greater adherence to a DASH-style diet significantly reduced CVD (Relative Risk [RR]=0.80; 95% CI = 0.74 to 0.86), CHD (RR=0.79; 95% CI = 0.71 to 0.88), and stroke (RR=0.81; 95% CI = 0.72 to 0.92). All of the studies meta-analyzed also were included the NEL's evidence base for the DASH-style diet.

Vegetarian Dietary Patterns

The NEL systematic review concluded that evidence for the effects of vegetarian dietary patterns on cardiovascular endpoints is limited. Most of this evidence was from prospective cohort studies; four out of six studies suggested that a vegetarian dietary pattern was associated with reduced incidence of ischemic heart disease (IHD) or CVD mortality. A meta-analysis of seven studies related to CVD mortality and vegetarian diet[14] (including two of the studies from the NEL systematic review) found that mortality from IHD was significantly lower in vegetarians than in non-vegetarians (RR=0.71; 95% CI = 0.56 to 0.87). The authors estimated a 16 percent lower mortality from circulatory diseases (RR=0.84; 95% CI = 0.54 to 1.14) and a 12 percent lower mortality from cerebrovascular disease (RR=0.88; 95% CI = 0.70 to 1.06) in vegetarians compared to non-vegetarians.

For additional details on this body of evidence, visit: References 2, 10, 11, 14-19 and ***Appendix E-2.26***

DIETARY PATTERNS AND BODY WEIGHT

Question 2: What is the relationship between dietary patterns and measures of body weight or obesity?

Source of evidence: Existing reports

Conclusion

The DGAC concurs with the 2013 AHA/ACC/TOS *Guideline for the Management of Overweight and Obesity* that strong evidence demonstrates that, preferably as part of a comprehensive lifestyle intervention carried out by multidisciplinary teams of professionals or nutrition professionals, overweight and obese adults can achieve weight loss through a variety of dietary patterns that achieve an energy deficit. Clinically meaningful weight losses that were achieved ranged from 4 to 12 kg at 6-month follow-up. Thereafter, slow weight regain is observed, with total weight loss at 1 year of 4 to 10 kg and at 2 years of 3 to 4 kg. However, some dietary patterns may be more beneficial in the long-term for cardiometabolic health.

DGAC Grade: Strong

The DGAC concurs with the NEL Dietary Patterns Systematic Review Project that moderate evidence indicates dietary patterns that are higher in vegetables, fruits, and whole grains; include seafood and legumes; are moderate in dairy products (particularly low and non-fat dairy) and alcohol; lower in meats (including red and processed meats), and low in sugar-sweetened foods and beverages, and refined grains are associated with favorable outcomes related to healthy body weight (including lower BMI, waist circumference, or percent body fat) or risk of obesity. Components of the dietary patterns associated with these favorable outcomes include higher intakes of unsaturated fats and lower intakes of saturated fats, cholesterol, and sodium. **DGAC Grade: Moderate**

Evidence for children is limited, but studies in the NEL Dietary Patterns Systematic Review Project and the systematic review focused on this age group by Ambrosini et al.[34] suggest that dietary patterns in childhood or adolescence that are higher in energy-dense and low-fiber foods, such as sweets, refined grains, and processed meats, as well as sugar-sweetened beverages, whole milk, fried potatoes, certain fats and oils, and fast foods increase the risk of obesity later on in life. **DGAC Grade: Limited**

Implications

To achieve and maintain a healthy body weight, individuals are encouraged to consume dietary patterns that are higher in vegetables, fruits, and whole grains; include seafood and legumes; are moderate in dairy products (with an emphasis on low- and non-fat dairy), and alcohol; and are lower in meats (including red and processed meats), sugar-sweetened foods and beverages, and refined grains. During childhood and adolescence, a time period critical for the prevention of obesity later in life, a dietary pattern similar to that associated with a healthy weight in adults should be encouraged.

Among overweight and obese individuals, an energy deficit is necessary to achieve weight loss. This can be achieved through a variety of evidence-based dietary patterns and should be approached with comprehensive lifestyle interventions. While it is possible to lose weight on his/her own, it is more successful if conducted by trained professionals or by referral to a nutrition professional for individual or group counseling (for more details refer to AHA/ACC/TOS *Guideline for the Management of Overweight and Obesity*[13] algorithm Box 11B). Strategies should be based on the individual's preferences and health status and consider the socio-cultural influences on lifestyle behaviors that relate to long-term behavior maintenance. These approaches are best complemented with population-based approaches, as mentioned in ***Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change*** and ***Part D. Chapter 4: Food Environment and Settings***, which will allow all factors influencing lifestyle behaviors to be addressed as defined in the socio-ecological model.

Review of the Evidence

The DGAC considered evidence from the 2013 AHA/ACC/TOS Obesity Guideline and associated NHLBI Obesity Report, which included only randomized trials,[12, 13] the NEL Dietary Patterns Systematic Review Project,[2] which included 38 studies predominately of prospective cohort design and a few randomized trials, and two systematic reviews/meta-analyses published since 2008.[34, 35] In total, 81 articles were considered in these reports. The published reviews provided evidence for the pediatric population (included 7 studies of which 2 overlapped with those in the NEL review) and further evidence for dietary patterns related to the Mediterranean-style diet and its effect on obesity and weight loss (all randomized trials of which 1 out of the 16 studies overlapped with the NEL review).

Dietary Patterns and the Management of Overweight and Obesity

In the NHLBI Obesity Report, the 12 randomized studies described in summary Table 3.1 of the report all confirm that to lose weight, a variety of dietary pattern approaches can be used and a reduction in caloric intake is required. The energy balance equation requires that for weight loss, one must consume less energy than one expends or expend more energy than one consumes. The report states that any one of the following methods can be used to reduce food and calorie intake: prescription of 1,200 to 1,500 kcal/day for women and 1,500 to 1,800 kcal/day for men (kcal levels are usually adjusted for the individual's body weight); prescription of a 500 kcal/day or 750 kcal/day energy deficit; or prescription of an evidence-based diet that restricts certain food types (such as high-carbohydrate foods, low-fiber foods, or high-fat foods) in order to create an energy deficit by reduced food intake.

For the different dietary approaches (provided either as part of a comprehensive lifestyle change intervention carried out by a multi-disciplinary team of trained professionals or within nutrition interventions conducted by nutrition professionals) that the authors of the report evaluated, it is evident that all prescribed diets that achieved an energy deficit were associated with weight loss. There was no apparent superiority of one approach when behavioral components were balanced in the treatment arms. Results indicated that average weight loss is maximal at 6 months with smaller losses maintained for up to 2 years, while treatment and follow-up taper. Weight loss achieved by dietary techniques aimed at reducing daily energy intake ranges from 4 to 12 kg at 6-month follow-up. Thereafter, slow weight regain is observed, with total weight loss at 1 year of 4 to 10 kg and at 2 years of 3 to 4 kg. The following dietary approaches are associated with weight loss if reduction in dietary energy intake is achieved:

- A diet from the European Association for the Study of Diabetes Guidelines, which focuses on targeting food groups, rather than formal prescribed energy restriction while still achieving an energy deficit.

- Higher protein (25 percent of total calories from protein, 30 percent of total calories from fat, 45 percent of total calories from carbohydrate) with provision of foods that realized energy deficit.
- Higher protein Zone™-type diet (5 meals/day, each with 40 percent of total calories from carbohydrate, 30 percent of total calories from protein, 30 percent of total calories from fat) without formal prescribed energy restriction but realized energy deficit.
- Lacto-ovo-vegetarian-style diet with prescribed energy restriction.
- Low-calorie diet with prescribed energy restriction.
- Low-carbohydrate (initially less than 20 g/day carbohydrate) diet without formal prescribed energy restriction but realized energy deficit.
- Low-fat (10 percent to 25 percent of total calories from fat) vegan-style diet without formal prescribed energy restriction but realized energy deficit.
- Low-fat (20 percent of total calories from fat) diet without formal prescribed energy restriction but realized energy deficit.
- Low-glycemic load diet, either with formal prescribed energy restriction or without formal prescribed energy restriction but with realized energy deficit.
- Lower fat (≤30 percent fat), high dairy (4 servings/day) diets with or without increased fiber and/or low-glycemic index/load foods (low-glycemic load) with prescribed energy restriction.
- Macronutrient-targeted diets (15 percent or 25 percent of total calories from protein; 20 percent or 40 percent of total calories from fat; 35 percent, 45 percent, 55 percent, or 65 percent of total calories from carbohydrate) with prescribed energy restriction.
- Mediterranean-style diet with prescribed energy restriction.
- Moderate protein (12 percent of total calories from protein, 58 percent of total calories from carbohydrate, 30 percent of total calories from fat) with provision of foods that realized energy deficit.
- Provision of high-glycemic load or low-glycemic load meals with prescribed energy restriction.
- The AHA-style Step 1 diet (with prescribed energy restriction of 1,500 to 1,800 kcal/day, <30 percent of total calories from fat, <10 percent of total calories from saturated fat).

Although these dietary patterns with an energy deficit will result in weight loss during a 6-months to 2-year period, long-term health implications with certain patterns may be detrimental to cardiometabolic health. These associations have been discussed in the dietary patterns and cardiovascular health section as well as the saturated fat and cardiovascular health section.

As presented in Table D2.1 at the end of the chapter, the results of the randomized studies considered in the AHA/ACC/TOS Guideline provide evidence for what works in terms of the components of a comprehensive lifestyle intervention or nutrition interventions that are needed to achieve weight loss with the variety of dietary approaches described above.

Dietary Patterns and their Association with Body Weight

A total of 14 studies met the inclusion criteria for the index/score question of the NEL systematic review and were categorized based on dietary pattern exposure. Two major categories were identified: (1) studies that examined exposure based on a Mediterranean-designated dietary pattern and (2) studies that examined exposure based on expert dietary guidelines recommendations. Taken together, there were six studies on Mediterranean-designated diet scores,[23, 31, 32, 36-38] five studies on dietary guidelines-based indices,[39-43] two studies on Mediterranean-designated scores and dietary guidelines indices,[44, 45] and one study that used a trial-based customized score.[46] Two of the studies were RCTs of positive quality[23, 46] and 12 were prospective cohort studies. The studies were carried out between 2006 and 2012.

The sample sizes for prospective cohort studies ranged from 732 to 373,803 participants, with follow-up times from 1.5 to 20 years. Ten out of 12 of the prospective cohort studies were conducted with generally healthy adults with a mean age of 25 to 63 years. Two studies were conducted with children and adolescents (one with girls).[39, 40] The two RCTs were conducted in adults with elevated chronic disease risk: one study with a Mediterranean-designated diet intervention on older adults at increased CVD risk with more than 90 percent overweight or obese[23] and one study using an a priori diet intervention on men with pre-existing metabolic syndrome.[46] The sample sizes for the RCTs were from 187 to 769 subjects and duration of follow-up ranged from 3 to 12 months.

Mediterranean-style Dietary Pattern

Four out of the six studies evaluating the Mediterranean style dietary pattern were conducted in Spain.[23, 32, 36, 37] Of the other two, one study was the European multicenter study that was part of the EPIC-Physical Activity, Nutrition, Alcohol Consumption, Cessation of Smoking, Eating out of Home, and Obesity (EPIC-PANACEA) study,[38] and one was conducted in the United States.[31]

<u>Dietary Patterns and Body Weight and Incidence of Overweight and/or Obesity</u>

The Prevencion con Dieta Mediterranean (PREDIMED) study tested the effects of a Mediterranean diet on the primary prevention of cardiovascular disease in a high-risk group of men and women. Subjects either had type 2 diabetes or three cardiovascular disease risk factors (such as hypertension or current smoking) and 90 percent were overweight or obese defined as BMI ≥ 25 kg/m^2. The PREDIMED trial randomly assigned participants to three interventions: (1) Mediterranean diet with extra virgin olive oil, (2) Mediterranean diet with mixed nuts, and (3) low-fat diet. At end of 3 months of a 4-year clinical trial, the authors found that the Mediterranean diet score increased in the two Mediterranean diet groups of the trial and remained unchanged in the low-fat group. However, no significant changes in body weight and adiposity occurred within or between groups from baseline to the 3 months. Beunza et al., 2010 reported on a prospective cohort study in Spain, the Seguimiento Universidad de Navarra (SUN) study.[36] Participants with the highest adherence to a Mediterranean dietary pattern, assessed using the Trichopoulou Mediterranean Diet Score (MDS) were found to have lower average yearly weight gain, -0.059 kg/y (95% CI = -0.111 to -0.008 kg/y; p for trend = 0.02), than participants in the lowest adherence group.[7] However, the MDS was not associated with incidence of overweight or obesity in participants who were normal weight at baseline. Mendez et al., 2006 reported on the EPIC-Spain prospective cohort study.[37] Adherence to a Mediterranean diet was assessed using a slight modification of the Trichopoulou MDS, with exposure categorized in tertiles of low (0-3), medium (4-5), and high (6-8) adherence. Participants with highest MDS adherence had reduced incidence of obesity when overweight at baseline; overweight women and men were 27 percent and 29 percent, respectively, less likely to become obese. High MDS adherence was not associated with incidence of overweight in subjects who were normal weight at baseline. The EPIC-PANACEA study examined the association between adherence to the relative Mediterranean dietary pattern (rMDS), prospective weight change, and the incidence of overweight or obesity. Participants with high rMED adherence gained less weight in 5 years than did participants with low rMED adherence (-0.16 kg; 95% CI = -0.24 to -0.07 kg) and had a 10 percent lower odds of becoming overweight or obese (OR = 0.90; 95% CI = 0.82 to 0.96). The contribution of each rMED scoring component also was assessed and it was found that the association between rMED and weight change was no longer significant when meat and meat products were not part of the score. Lastly, a meta-analysis of the odds ratio scores of all 10 European countries showed that a 2-point increase in rMED score was associated with 3 percent (95% CI = 1 to 5%) lower odds of becoming overweight or obese over 5 years.

<u>Dietary Patterns and Waist Circumference</u>

Rumawas et al., 2009 conducted a prospective cohort study using a subset of the Framingham Offspring and Spouse (FOS) study.[31] Dietary exposure was assessed in quintiles of low to high adherence to the Mediterranean style dietary pattern score (MSDPS). Participants with a higher MSDPS had significantly lower waist circumference (p for trend < 0.001). Tortosa et al., 2007 reported on the association of the Mediterranean dietary pattern and metabolic syndrome in the SUN study conducted in Spain.[32] Participants in the highest tertile of adherence to the MDS had lower waist circumference, -0.05 cm over 6 years (p for trend = 0.038), compared to the lowest tertile.

Although some mixed results from prospective studies may be due to differences in the length of follow up, definition of the Mediterranean dietary pattern and population included, the results of randomized studies indicate a significant reduction in body weight when calories are restricted. A high quality meta-analysis (AMSTAR rating of 11) on the association of a Mediterranean-style diet with body weight conducted by Esposito included 16 randomized studies of which one[32] overlapped with the NEL systematic review was included in the DGAC body of evidence for this question. The meta-analysis included studies conducted in the United States, Italy, Spain, France, Israel, Greece, Germany, and the Netherlands that lasted from 4 weeks to 24 months with a total of 3,436 participants. Using a random effects model, participants in the Mediterranean diet group had significant weight loss (mean difference between Mediterranean diet and control diet, -1.75 kg; 95% CI = -2.86 to -0.64) and reduction in BMI (mean difference, -0.57 kg/m^2; 95% CI = 0.93 to 0.21 kg/m^2) compared to those in the control arm. The effect of Mediterranean diet on body weight was greater in association with energy restriction (mean difference, -3.88 kg; 95% CI = -6.54 to -1.21 kg), increased physical activity (-4.01 kg; 95% CI = -5.79 to -2.23 kg), and follow up longer than 6 months (-2.69 kg; 95% CI = -3.99 to -1.38 kg). Across all 16 studies, the Mediterranean style dietary pattern did not cause weight gain.

Dietary Guidelines-Based Indices

Of the seven studies conducted on dietary guidelines-based indices, three studies were conducted in the United States with U.S.-based indices.[39, 41, 43] One study was conducted in Germany with an index developed in the United States,[40] and two studies were conducted in France (one used a French index,[42] and the other compared six different dietary scores).[44]

<u>Dietary Patterns and Body Weight and Incidence of Overweight and/or Obesity</u>

Gao et al., 2008 reported on a prospective cohort study of White, African American, Hispanic, and Chinese men and women in the Multi-Ethnic Study of Atherosclerosis (MESA) in the US. Two versions of the 2005 HEI were used: the original and a modified version that adjusted the food group components to incorporate levels of caloric need based on sex, age, and activity level.[41] For the overall population, there was an inverse association between quintiles of each HEI score and BMI (p<0.001). The risk of obesity in normal weight participants was inversely associated with HEI scores only for Whites (p<0.05). A comparison of the HEI-1995 and HEI-2005 scores indicated that beta-coefficients, as predictors of body weight and BMI, were higher for the HEI-2005 scores in Whites. Zamora et al., 2010 analyzed data from the prospective cohort study, Coronary Artery Risk Development in Young Adults (CARDIA), conducted in the United States, to examine the association between diets consistent with the 2005 Dietary Guidelines and subsequent weight gain in Black and White young adults.[43] The Diet Quality Index (DQI) included 10 components of the 2005 Dietary Guidelines relating to the consumption of total fat, saturated fat, cholesterol, added sugars, reduced-fat milk, fruit, vegetables, whole grains, nutrient-dense foods, and limited sodium and alcohol intake. They found, a 10-point increase in DQI score was associated with a 10 percent lower risk of gaining 10 kg in normal-weight Whites. However, the same magnitude increase in score was associated with a 15 percent higher risk in obese Blacks (p<0.001). Kesse-Guyot et al., 2009 conducted a prospective cohort study in France to examine the association between adherence to a dietary score based on the French 2001 nutritional guidelines (Programme National Nutrition Sante´ guidelines score (PNNS-GS) and changes in body weight, body fat distribution, and obesity risk.[42] The PNNS-GS includes 12 nutritional components: fruit and vegetables, starchy foods, whole grains, dairy products, meat, seafood, added fat, vegetable fat, sweets, water and soda, alcohol, and salt. The last PNNS-GS component is physical activity. In fully adjusted models, an increase of one PNNS-GS unit was associated with lower weight gain (p=0.004), and lower BMI gain (p=0.002). An increase of 1 PNNS-GS unit was associated with a lower probability of becoming overweight (including obese) (OR = 0.93; 95% CI = 0.88 to 0.99). Similarly, an increase of 1 PNNS-GS unit was associated with a lower probability of becoming obese (OR = 0.89; 95% CI = 0.80 to 0.99).

Two studies were conducted in children. Cheng et al., 2010 analyzed data from a prospective cohort study conducted in Germany, the Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) study, to examine whether the diet quality of healthy children before puberty was associated with body composition at onset of puberty.[40] Adherence to a diet pattern was assessed by the Revised Children's Diet Quality Index (RC-DQI) which was based on the Dietary Guidelines for Americans. In this study, a higher dietary quality was associated with a higher energy intake, and children with a lower diet quality had lower BMI and Fat Mass Index (FMI) Z-scores at baseline (p<0.01) but not at onset of puberty. Berz et al., 2011 reported on a prospective cohort study to assess the effects of the DASH eating pattern on BMI in adolescent females over a 10-year period.[39] Only seven out of the 10 original components of the DASH score were used; the three excluded were added sugars, discretionary fats and oils, and alcohol. Overall, girls in the highest vs. lowest quintile of DASH score had an adjusted mean BMI of 24.4 vs. 26.3 kg/m2 (p<0.05).

<u>Dietary Patterns and Waist Circumference</u>

Gao et al, found, for the overall population in the MESA study, an inverse association between quintiles of each HEI score and waist circumference (WC) (p<0.001).[41] The study by Kesse-Guyot conducted in France showed, in fully adjusted models, an increase of one PNNS-GS unit was associated with lower waist circumference gain (p=0.01) and lower waist-to-hip ratio gain (p=0.02).[42]

Other Indices

Jacobs et al., 2009 conducted an RCT in Norway, the Oslo Diet and Exercise Study, to examine the effect of changes in diet patterns on body weight and other outcomes among men who met the criteria for the metabolic syndrome (n=187 men).[46] Study participants were randomly assigned to: (1) the diet protocol, (2) the exercise protocol, (3) the diet + exercise protocol, or (4) the control protocol. The trial duration was 12 months. The authors created their own diet score to assess adherence to the intervention. The score was based on summing the participants ranking of intake (across tertiles) of 35 food groups that, based on the literature, had a beneficial neutral or detrimental effect on health. A higher score reflected greater adherence to the diet intervention. Over the course of the intervention, the diet score increased by 2 points (SD ±5.5) in both diet groups, with a decrease of an equivalent amount in the exercise and control groups. A 10-point change in the diet score during the intervention period was associated with a 3.5 kg decrease in weight, a 2.8 cm decrease in waist circumference and 1.3 percent decrease in percent body fat (all significant at $p<0.0001$).

Studies that Compared Various Dietary Indices

In a study by Lassale et al., subjects were participants in the SUpplementation en VItamines et Minereaux AntioXydants (SU.VI.MAX) study and diet quality was assessed using a Mediterranean Score (MDS, rMED, MSDPS), the Diet Quality Index-International (DQI-I), the 2005 Dietary Guidelines for Americans Adherence Index (DGAI), and the French Programme National Nutrition Sante-Guidelines Score (PNNS-GS).[44] Overall, better adherence to a Mediterranean diet (except for the MSDPS) or expert dietary guidelines was associated with lower weight gain in men who were normal weight at baseline (p for trend = <0.05). In addition, among the 1,569 non-obese men at baseline, the odds of becoming obese associated with one standard deviation increase in dietary score ranged from OR = 0.63 (95% CI = 0.51 to 0.78) for the DGAI to OR = 0.72 (95% CI = 0.59 to 0.88) for the MDS, only the MSDPS was non-significant. In women, no association between diet scores and weight gain or incidence of obesity was found. Woo et al., 2008 reported on a prospective cohort study in Hong Kong to examine adherence to a diet pattern using the MDS and the Diet Quality Index International (DQI-I).[45] They found that increased adherence to either the MDS or DQI-I was not associated with becoming overweight.

Dietary Patterns from Data-Driven Methods

In the NEL review, a total of 11 studies from prospective cohort studies were included that either used factor or cluster analyses to derive dietary patterns. Eight of the eleven studies were conducted in the United States, with additional studies from the United Kingdom, Iran, and Sweden. The sample sizes ranged from 206 to 51,670 participants with follow-up times from 3 to 20 years. The majority of the studies were conducted with generally healthy adult men and women,[47-52] five studies included women only,[53-57] and one was conducted in children to examine weight gain in adolescence over the period of follow-up.[56] Outcomes examined included change in body weight (3 studies), BMI (7 studies), and waist circumference (6 studies); one study examined both percent body fat and incidence of overweight/obesity.

Most of the studies found at least two generic food patterns: a “healthy/prudent” food pattern and an “unhealthy/western” pattern. Generally, healthy patterns were associated with more favorable body weight outcomes, while the opposite was seen for unhealthy patterns. However, not all studies reported significant associations. There was a potential difference in associations found by sex: of the three studies that analyzed men and women separately, men tended to have null results. However, data were insufficient to draw conclusions about population subgroups. Furthermore, because the patterns are data-driven, they represent what was consumed by the study population, and thus it is difficult to compare across the disparate patterns. The one study that analyzed the dietary patterns of pre-pubescent children transitioning into adolescence showed that patterns vary widely at this age and caution should be observed when analyzing these data because the diet of children changes rapidly, as does their weight.

The DGAC considered the systematic review by Ambrosini et al. that included seven articles, two of which overlapped with the NEL review.[34] Results demonstrated a positive association between a dietary pattern high in energy-dense, high fat, and low fiber foods and later obesity (4 of the 7 studies), while three studies demonstrated null associations. The seven longitudinal studies of children from the United Kingdom, United States, Australia, Norway, Finland, and Colombia had follow-up periods ranging from 2 to 21 years and had sample sizes from 427 to 6772 individuals. The studies determined dietary patterns using factor or cluster analysis (5) or reduced rank regression (2).

For additional details on this body of evidence, visit: References 2, 13, 34, 35 and ***Appendix E-2.27***

DIETARY PATTERNS AND TYPE 2 DIABETES

Question 3: What is the relationship between dietary patterns and risk of type 2 diabetes?

Source of evidence: Existing reports

Conclusion

Moderate evidence indicates that healthy dietary patterns higher in vegetables, fruits, and whole grains and lower in red and processed meats, high-fat dairy products, refined grains, and sweets/sugar-sweetened beverages reduce the risk of developing type 2 diabetes. **DGAC Grade: Moderate**

Evidence is lacking for the pediatric population.

Implications

To reduce the risk of developing type 2 diabetes, individuals are encouraged to consume dietary patterns that are rich in vegetables, fruits, and whole grains and lower in red and processed meats, high-fat dairy, refined grains, and sweets/sugar-sweetened beverages in addition to maintaining a healthy body weight. Diabetes can be prevented through the consumption of a variety of healthy dietary patterns that share these components and that are tailored to the biological needs and socio-cultural preferences of the individual and carried out preferably through counseling by a nutrition professional.

Review of the Evidence

The Committee considered two sources of evidence. The primary source was the NEL Dietary Patterns Systematic Review Project which included 37 studies predominantly of prospective cohorts design and some randomized trials (n=8).[2] This primary source was supplemented by a published meta-analysis[58] that included 15 cohort studies of which 13 overlapped with the NEL review.[58] The meta-analysis provided an estimate of the effect size of incident type 2 diabetes associated with a healthy and unhealthy dietary pattern.

Although the NEL rated the overall body of evidence for type 2 diabetes as limited, this was primarily a result of examining the different methods for defining dietary patterns (e.g. indices, data driven, and reduce rank regression) separately. As such, the NEL noted these methodological inconsistencies across studies but stated general support for the consumption of a dietary pattern rich in vegetables and fruits and low in high-fat dairy and meats. The DGAC concurred with this conclusion. However, the DGAC has elevated the grade of the entire body of evidence to moderate given that the NEL findings were corroborated by the results of a high quality meta-analysis (AMSTAR rating of 11) and the magnitude of the associations that showed when the results of 15 cohort studies are pooled, evidence indicated a 21 percent reduction in the risk of developing type 2 diabetes associated with dietary patterns characterized by high consumption of whole grains, vegetables, and fruit. Conversely, a 44 percent increased risk of developing type 2 diabetes was seen with an unhealthy dietary pattern characterized by higher consumption of red or processed meats, high-fat dairy, refined grains, and sweets.

Dietary Patterns and Incident Type 2 Diabetes

Dietary Approaches to Stop Hypertension (DASH)

One study used the DASH score in a cohort of 820 U.S. adults ages 40 to 69 years and with equal sex distribution and racial diversity.[59] Liese et al. found adherence to the DASH score was associated with markedly reduced odds of type 2 diabetes in Whites but not in the total population, or in the Blacks and Hispanics, which comprised the majority of this cohort.

Mediterranean-style Dietary Patterns

Three studies assessed Mediterranean-style dietary pattern adherence (Mediterranean Diet Score [MDS]) with sample sizes ranging from 5,000 to more than 20,000 in both Mediterranean and U.S. populations. One study conducted in Spain with the SUN cohort (n=13,380) found a favorable association between the MDS (the original MDS of Trichopoulou) and risk of type 2 diabetes. Overall, a 2-point increase in MDS was associated with a 35 percent reduction in risk of type 2 diabetes.[60] Another study, conducted in Greece with the EPIC-Greece cohort (n=22,295), also assessed the relationship between the MDS and type 2 diabetes. In this second Mediterranean population, adherence to the MDS also was favorably associated with decreased risk of diabetes.[61] Conversely, a study conducted in the United States, using the authors' MedDiet Score with the Multi-Ethnic Study of Atherosclerosis (MESA) cohort (n=5,390) found no association between their MedDiet Score and type 2 diabetes incidence in the total population, in men or women, or in specific racial/ethnic groups.[62]

Dietary Indices based on the Dietary Guidelines

Four studies used dietary guidelines-based indices such as the AHEI and the Diet Quality Index (DQI). The sample sizes of the studies ranged from 1,821 to 80,029. A study that assessed adherence to the AHEI in the United States found a favorable association between AHEI score and risk of incident type 2 diabetes in women in the Nurses' Health Study (n=80,029).[63] In the CARDIA study (n=4,381), also from the United States, the authors found no association between DQI-2005 score and type 2 diabetes incidence in the total population or in Blacks or Whites.[29] Studies from outside the United States included one conducted in Australia using a Total Diet score in the Blue Mountains Eye Study (BMES, n=1,821) and one from Germany using a German Food Pyramid Index with the EPIC-Potsdam cohort (n=23,531). Neither found an association between these scores and incident type 2 diabetes.[64, 65] Thus, evidence for an association only exists with the AHEI, which does contain slightly different components from the other indices, such as nuts and legumes, trans fat, EPA + DHA (n–3 FAs), PUFAs, alcohol, red and processed meat.

Data-Driven Approaches

Eleven studies used factor analysis and one study used cluster analysis. These analyses were all conducted using data from prospective cohort studies published between 2004 and 2012 and had sample sizes ranging from 690 to more than 75,000 individuals. Five studies were conducted in the United States and the rest from developed countries around the world. Each study identified one to four dietary patterns, with the most common comparison between "western"/"unhealthy" and "prudent"/"healthier" patterns; a total of 35 diverse dietary patterns were identified within the body of evidence. Many studies had null findings, particularly studies with duration of less than 7 years of follow up.[66-69] Patterns associated with lower risk of type 2 diabetes were characterized by higher intakes of vegetables, fruits, low-fat dairy products, and whole grains, and those associated with increased risk were characterized by higher intakes of red meat, sugar-sweetened foods and drinks, French fries, refined grains, and high-fat dairy products. However, the food groups identified varied substantially, even among patterns with the same name.

Three prospective cohort studies used reduced rank regression to examine the relationship between dietary patterns and type 2 diabetes.[70-72] Two of the studies were conducted in the

United States and one in the United Kingdom. The sample sizes were 880 for Liese (2009), 2,879 for Imamura (2009), and 6,699 for McNaughton (2008). The independent variables in these studies were dietary pattern scores, and biomarkers were used as response variables in two of the studies. Dietary patterns that included meat intake and incident type 2 diabetes were positively associated in the two studies that used biomarkers as response variables, though the definitions of meat differed.[70, 71] However, because so few studies were available and the methodology used and different populations considered varied so much, the information was insufficient to assess consistency or draw conclusions.

Other Dietary Patterns

The body of evidence examined included seven studies conducted between 2004 and 2013, consisting of six RCTs[73-79] and one prospective cohort study (PCS).[80] Two studies were conducted in the United States; one in the United States and Canada; one in Spain (2 PREDIMED articles); and one each in Greece, Italy, and Sweden. The sample sizes of the RCTs ranged from 82 to 1,224 participants and the PCS had a sample size of 41,387 participants. All eight studies were conducted in adults. RCT duration ranged from 6 weeks to a median of 4 years and the PCS duration was 2 years. The RCTs were primary prevention studies of at-risk participants. Baseline health status in the study participants included those with mild hypercholesterolemia, overweight or obesity, metabolic syndrome, abdominal obesity, and three or more CVD risk factors, including metabolic syndrome. The PCS participants were individuals in the Adventist Health Study who did not have type 2 diabetes.[80] Three studies looked at a Mediterranean-style diet,[75, 77-79] one study examined the Nordic diet (defined by the authors of the study as a diet rich in high-fiber plant foods, fruits, berries, vegetables, whole grains, rapeseed oil, nuts, fish and low-fat milk products, but low in salt, added sugars, and saturated fats),[73] and three studies looked at either the DASH diet or a variation of the DASH diet,[74, 76] or a vegetarian diet.[80]

Two of the seven studies examined the association between adherence to a dietary pattern and incidence of type 2 diabetes.[79, 80] Although the results of both studies showed a favorable association between either a Mediterranean-style or a vegetarian dietary pattern and incidence of type 2 diabetes the studies differed in design and dietary pattern used to assess diet exposure. The other studies examined the intermediate outcomes of impaired glucose tolerance and/or insulin resistance and are discussed in the next section.

Dietary Patterns and Intermediate Outcomes

Five studies examined adherence to a dietary pattern and intermediate outcomes related to glucose tolerance and/or insulin resistance: two RCTs[23, 46] and three prospective cohort studies.[29, 31, 64] It was difficult to assess food components across these studies, as numerous different scores were used and no compelling number of studies used any one score or index. Even so, favorable associations between dietary patterns and intermediate outcomes were found.

The two RCTs were conducted in populations in Europe that were at risk of diabetes. An early report from the PREDIMED trial showed that a Mediterranean diet decreased fasting blood glucose, fasting insulin, and HOMA-IR scores in a Spanish population at risk of CVD.[23] In the Oslo Diet and Exercise Study (ODES), increased adherence to the authors' a priori diet score resulted in decreased fasting insulin and insulin after a glucose challenge, but not fasting glucose, in Norwegian men with metabolic syndrome.[46] Results from prospective cohort studies were consistent in showing a favorable association between diet score and fasting glucose, fasting insulin or HOMA-IR,[29, 31] with the exception of one study that found the association with fasting glucose only in men.[64]

Data-Driven Approaches

Variations in populations studies, definition of outcomes, dietary assessment methodologies, and methods used to derive patterns resulted in a highly variable set of dietary patterns, thus making it difficult to draw conclusions from studies using data-driven approaches. For example, one study measured fasting blood glucose with a cutoff of 6.1 and greater mmol/L;[47] another study measured plasma glucose with a cutoff of 5.1 and greater mmol/L,[81] while a third study measured plasma glucose after an overnight fast and after a standard 75 g oral glucose tolerance test.[82] Three prospective cohort studies assessed the association between dietary patterns and plasma glucose levels. Two U.S. studies derived patterns using cluster analysis[47, 81] and one study conducted in Denmark used factor analysis.[82] Duffey et al. identified two diet clusters: "Prudent Diet" and "Western Diet";[47] Kimokoti et al. identified five clusters: "Heart Healthier," "Lighter Eating," "Wine and Moderate Eating," "Higher Fat," and "Empty Calories";[81] and Lau et al. derived two factors: "Modern" and "Traditional."[82]

For additional details on this body of evidence, visit: References 2, 58, and ***Appendix E-2.28***

DIETARY PATTERNS AND CANCER

Existing Evidence around Foods and Nutrients and Cancer

The role of dietary composition in cancer risk has been postulated since ancient times, yet scientific evidence for such relationships was sparse until nearly a century ago. Experimental models of cancer based upon chemical carcinogens, radiation, viral-transmission, and inherited genetic variations gradually emerged in first half of the 20th century and were soon found to be influenced by dietary and nutritional interventions. The establishment of population-based cancer registries around the globe in the years following World War II clearly indicated that the incidence and mortality of specific cancers and the patterns of cancers varied widely between countries. Soon, studies of migrant populations demonstrated that in parallel with acculturation, cancer risk evolved toward that observed in the adopted country, implicating a strong role for environmental influences, such as dietary patterns, in cancer risk. When coupled with national food consumption data, relationships between dietary patterns or components and cancer risk were hypothesized. The development of dietary assessment tools, such as FFQs, paved the way for large prospective epidemiologic cohort studies designed to examine more precisely the role of dietary patterns, foods, and specific nutrients in the risk of various cancers.[83] Additional diet assessment tools, such as food diaries, and single and multi-day 24-hr recalls enhanced the ability to undertake population studies and mechanism-based RCTs. These studies were made possible by USDA support of research to advance laboratory methods to define the nutrient content of foods in the U.S. food supply and establish a database that, when coupled with diet assessment tools, provides an estimated intake of energy, macronutrients, vitamins, minerals and other dietary variables. More recently, inclusion into the database of non-nutrient bioactive components primarily found in vegetables and fruits has enhanced the ability to define human intake of bioactive components that may affect health and disease.

In 1982, the American Institute for Cancer Research (AICR), a part of the World Cancer Research Fund (WCRF) global philanthropic network, was established. Together, the mission of WCRF/AICR is to fund research and disseminate evidence-based cancer prevention guidelines to the public. In 1997, the AICR/WCRF published the results of a comprehensive multi-year effort to systematically review the published scientific literature and develop dietary guidelines for cancer prevention.[84] With a rapid expansion of available data in the subsequent years, the process was repeated for the 2007 AICR/WCRF report.[85] This effort has been enhanced in subsequent years by the AICR/WCRF Continuous Update Project (CUP), in which data are reviewed and updated on a continuous, rolling basis for specific cancers, with several reports completed annually.[86] This effort is accomplished through a rigorous systematic review process in which scientific evidence is gathered, reviewed and judged by panels of experts in nutrition and cancer in order to generate nutrition and cancer prevention goals for policy makers, the general population, and individuals seeking to reduce cancer risk.[87] The most recent summary of the systematic review which documents important information about the relationship between specific foods, nutrients and other lifestyle behavior and cancer risk is found in Table D2.2.

As previously mentioned, the 2015 DGAC chose to determine whether an examination of dietary patterns, could inform the understanding of diet and cancer risk. As this scientific literature is relatively early in its development, we limited our search to the four most common malignancies affecting the American public—lung, breast, colon/rectal, and prostate—which account for the majority of the cancer burden in the United States. Although the published literature on dietary patterns and cancer risk is relatively young, the DGAC felt it was important to examine the evidence and conclusions, consider the implications for development of dietary guidelines, and indicate areas for future research.

Table D2.2. American Institute for Cancer Research / World Cancer Research Fund (AICR/WCRF) Summary of Strong Evidence on Diet, Nutrition, Physical Activity, and Cancer Prevention, updated 2014

↓↓ Convincing decreased risk. ↓ Probable decreased risk. ↑↑ Convincing increased risk. ↑ Probable increased risk. • Substantial effect on risk unlikely.	Mouth, Pharynx, Larynx (2007)	Nasopharynx (2007)	Esophagus (2007)	Lung (2007)	Stomach (2007)	Pancreas (2007)	Gall bladder (2007)	Liver (2007)	Colorectum (2011)	Breast Premenopause (2010)	Breast (Postmenopause (2010)	Ovary (2014)	Endometrium (2013)	Prostate (2014)	Kidney (2007)	Skin (2007)
Foods containing dietary fiber									↓↓							
Aflatoxins	↓		↓		↓			↑↑								
Non-starchy vegetables[1]					↓											
Allium vegetables									↓							
Garlic	↓		↓	↓	↓											
Fruits[2]									↑↑							
Red meat									↑↑							
Processed meat																
Cantonese-style salted fish		↑														
Diets high in calcium[3]									↓							
Salt, salted and salty foods					↑											
Glycemic load													↑			
Arsenic in drinking water				↑↑												↑
Mate			↑													
Alcoholic drinks[4]	↑↑		↑↑					↑	↑ | ↑↑	↑↑	↑↑				•	
Coffee					•								↓		•	
Beta-carotene[5]				↑↑										•		•
Physical activity[6]									↓↓		↓		↓			
Body fatness[7]			↑↑			↑↑	↑		↑↑	↓	↑↑	↑	↑↑	↑	↑↑	
Adult attained height						↑			↑↑	↑	↑↑	↑↑		↑		
Greater birth weight										↑						
Lactation										↓↓	↓↓					

1 Includes evidence on foods containing carotenoids for mouth, pharynx, larynx; foods containing beta-carotene for esophagus; foods containing vitamin C for esophagus.
2 Includes evidence on foods containing carotenoids for mouth, pharynx, larynx, and lung; foods containing beta-carotene for esophagus; food containing vitamin C for esophagus.
3 Evidence is from milk and studies using supplements for colorectum.
4 Convincing increased risk for men and probably increased risk for women for colorectum. Evidence applies to adverse effect for kidney.
5 Evidence derived from studies using supplements for lung.
6 Convincing increased risk for colon not rectum.
7 Probable increased risk for advanced not non-advanced prostate cancer.

AICR/WCRF Evidence Stratification[87]

Convincing: The evidence for a convincing grade is strong enough to support a causal relationship. This relationship is robust enough that it is unlikely to be modified of research in the foreseeable future. A grade of "convincing" requires evidence from more than one study type, data from at least two cohort studies, no unexplained heterogeneity between study types with regard to the presence or absence of an association, good quality studies where random or systematic errors are unlikely, presence of a dose-response relationship, and strong and plausible experimental evidence relating typical human exposures to relevant cancer outcomes.

Probable: The criteria for determining a probable diet and cancer relationship include: evidence from at least two cohort studies or at least five case-control studies, no substantial unexplained heterogeneity between or within study types in the presence or absence of an association or direction of effect, good quality studies where the likelihood of random or systematic error is low, and evidence for biologic plausibility.

Limited—suggestive: This grade is assigned when the evidence is too limited to permit a probable or convincing judgment, but there is evidence of a direction of effect. The evidence may have methodological flaws, or there may be a limited number of studies. A grade of "limited-suggestive" requires the following: evidence from at least two cohort studies or five case-control studies, there is some evidence for biologic plausibility, and the direction of the effect is generally consistent, although there may be some unexplained heterogeneity.

Limited—no conclusion: This grade describes diet and cancer relationships where the evidence was ample for review by the panel, but it was too limited to receive one of the other grades. The available studies may be of good quality, but limited in number or yielding inconsistent results.

Substantial effect on risk unlikely: This grade is assigned when the evidence is strong that a particular nutrient, food, dietary pattern, or physical activity is unlikely to have a substantial causal relationship to a cancer outcome. Data must be strong enough that modification in the foreseeable future is unlikely.

Question 4: What is the relationship between dietary patterns and risk of cancer?

Source of evidence: NEL systematic review

Conclusions

Colon/Rectal Cancer: Moderate evidence indicates an inverse association between dietary patterns that are higher in vegetables, fruits, legumes, whole grains, lean meats/seafood, and low-fat dairy and moderate in alcohol; and low in red and/or processed meats, saturated fat, and sodas/sweets relative to other dietary patterns and the risk of colon/rectal cancer. Conversely, diets that are higher in red/processed meats, French fries/potatoes, and sources of sugars (i.e., sodas, sweets, and dessert foods) are associated with a greater colon/rectal cancer risk. **DGAC Grade: Moderate**

Breast Cancer: Moderate evidence indicates that dietary patterns rich in vegetables, fruit, and whole grains, and lower in animal products and refined carbohydrate, are associated with reduced risk of post-menopausal breast cancer. The data regarding this dietary pattern and pre-menopausal breast cancer risk point in the same direction, but the evidence is limited due to fewer studies. **DGAC Grade: Moderate for postmenopausal breast cancer risk; Limited for premenopausal breast cancer risk**

Lung Cancer: Limited evidence from a small number of studies suggests a lower risk of lung cancer associated with dietary patterns containing more frequent servings of vegetables, fruits, seafood, grains/cereals, and legumes, and lean versus higher fat meats and lower fat or non-fat dairy products. Despite reported modest significant reductions in risk, definitive conclusions cannot be established at this time due to the small number of articles, as well as wide variation in study design, dietary assessment, and case ascertainment. **DGAC Grade: Limited**

Prostate Cancer: No conclusion can be drawn regarding the relationship between dietary patterns and the risk of prostate cancer. This is due to limited evidence from a small number of studies with wide variation in study design, dietary assessment methodology and prostate cancer outcome ascertainment. **DGAC Grade: Grade not assignable**

Implications

The data accumulating regarding the impact of dietary patterns on risk of certain types of cancers supports the concept that a healthy dietary pattern may significantly reduce the overall burden of cancer in the United States. Emerging studies on dietary patterns support the findings of expert reviews regarding individual foods and nutrients. Effective strategies to initiate early in life and maintain a healthy dietary pattern and body weight, coupled with regular physical activity, will significantly reduce the cancer burden in America.

Review of the Evidence

Dietary Patterns and Colorectal Cancer

This systematic review included 21 articles from prospective cohort studies and one article from an RCT published since 2000 that examined the relationship between dietary patterns and risk of colorectal cancer.[88-109] The articles used diverse methodology to assess dietary patterns. Nine articles used indices/scores to assess dietary patterns, 10 articles used data-driven methods, and three used other approaches.

The dietary patterns examined in this systematic review were defined in various ways, making comparisons between articles difficult. However, despite general heterogeneity in this body of evidence, some protective dietary patterns emerged, particularly in articles where patterns were defined by index or score; articles using data-driven methods were less consistent. Patterns emphasizing vegetables, fruits, fish/seafood, legumes, low-fat dairy, and whole grains were generally associated with reduced risk of colorectal cancer. Patterns higher in red/processed meats, potatoes/French fries, and sodas/sweets/added sugars were generally associated with increased risk of colorectal cancer.

The relationship between dietary patterns and colorectal cancer risk often varied by sex and tumor location. Results based on analysis by sex were mixed, while analysis in tumor subgroups seemed to indicate that dietary patterns may be more strongly associated with tumor development in distal regions of the colon/rectum. Although most cohort studies make extensive efforts to include participants across a wide range of race/ethnic groups and across the socio-economic continuum, there still may be some groups for which the association between dietary patterns and colorectal cancer risk cannot be reliably assessed and therefore conclusions cannot be drawn.

Dietary Patterns and Breast Cancer

This systematic review included 25 prospective cohort studies and one RCT published since 2000 that examined the relationship between dietary patterns and risk of breast cancer.[94, 101, 104, 110-131] The studies used multiple approaches to assess dietary patterns and cancer risk. Eight studies used indices/scores to assess dietary patterns, 13 studies used factor or principal components analysis, two used reduced rank regression, two made comparisons on the basis of animal product consumption, and one conducted an RCT of a low-fat dietary pattern.

This moderate body of evidence encompassed a large diversity in methods to assess or determine dietary patterns, making comparison across studies challenging. Despite this variability, 17 of the included studies found statistically significant relationships between dietary patterns and breast cancer risk, particularly among certain groups of women. Because a variety of different methodologies were employed to derive dietary patterns, and the patterns, while similar in many respects, were composed of different combinations of foods and beverages, it was difficult to determine which patterns had the greatest impact on breast cancer risk reduction.

The relationship between dietary patterns and breast cancer risk may be more consistent among postmenopausal women, but additional research is needed to explore the relationships for both pre- and post- menopausal cancer. Certain histopathologic and molecular phenotypes of breast cancer may be affected more by certain dietary patterns, but this has not yet been explored sufficiently. For example, limited studies to date suggest that estrogen or progesterone receptor status of breast cancers may define subgroups with unique dietary risk profiles, but no conclusions can be drawn at this time. More research is needed to explore other factors that may influence the relationship between dietary patterns during various stages of life and breast cancer risk, such as anthropometrics, BMI (including weight change over adulthood), physical activity, sedentary behavior, and reproductive history, including ages of menarche, age of menopause, parity, and breast feeding.

Dietary Patterns and Lung Cancer

This systematic review included three prospective cohort studies and one nested case-cohort study published since 2000 that examined the relationship between dietary patterns and risk of lung cancer.[101, 104, 132, 133] The studies used different methods to assess dietary patterns. Two studies used an index/score to measure adherence to a dietary pattern, one study derived dietary patterns using principal components analysis, and another based dietary patterns on participant reports of animal product intake. With only four relevant studies that used different approaches for assessing or determining dietary patterns, the evidence available to examine the relationship between dietary patterns and risk of lung cancer is limited.

Dietary Patterns and Prostate Cancer

This systematic review included seven prospective cohort studies (from six different cohorts) published since 2000 that examined the relationship between dietary patterns and risk of prostate cancer.[101, 134-139] The studies used different methods to assess dietary patterns. Three studies used index/scores to assess dietary patterns, two studies used factor analysis, one study used principle components analysis, and one made comparisons on the basis of animal product consumption.

Most of the seven studies included in this systematic review did not detect clear or consistent relationships between dietary patterns and risk of prostate cancer, though one found that adherence to the Dietary Guidelines (assessed using the HEI-2005 and AHEI-2010) was associated with a lower risk of prostate cancer, particularly among men who had a prostate-specific antigen screening in the past 3 years. Because these studies used a range of different approaches for assessing dietary patterns in populations with variable cancer screening patterns, had heterogeneous prostate cancer outcome ascertainment, and were typically limited to dietary exposure late in life, the results were inconclusive regarding risk for clinically significant prostate cancer.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3344

DIETARY PATTERNS AND CONGENITAL ANOMALIES

Existing Evidence around Foods and Nutrients and Congenital Anomalies

It is well established that adequate folate status is critical for the prevention of neural tube defects, specifically anencephaly and spina bifida, as well as other birth defects.[140] Folate is often described by its source, with "folate" referring to naturally occurring folate from food sources, and "folic acid" referring to the synthetic form used in dietary supplements and food fortification. After mandatory fortification of enriched cereal products with folic acid in 1998, serum folate concentrations in the U.S. population more than doubled, and rates of neural tube defects decreased by 20 to 30 percent.[141, 142]

Despite this decrease, nearly one fifth of females ages 14 to 30 years do not meet the estimated average requirement for folate, the level deemed to be adequate for one half of healthy females in the age group.[143] The current U.S. Preventive Services Task Force recommends that women capable of becoming pregnant should take 400 to 800 micrograms of folic acid daily from fortified food or supplements in addition to a healthy diet rich in food sources of folate and folic acid to reduce risk of neural tube and other birth defects.[144] Women with a history of a pregnancy affected by a neural tube defect or who are at high risk of neural tube defects require 4 mg of synthetic folic acid supplements daily under the supervision of a physician.[145] Given the emphasis on a healthy diet, the DGAC was interested in understanding which dietary patterns, if any, were associated with a decreased risk of congenital anomalies among women of reproductive age.

Question 5: What is the relationship between dietary patterns and risk of congenital anomalies?

Source of evidence: NEL systematic review

Conclusion

Limited evidence suggests that healthy maternal dietary patterns during the preconception period that are higher in vegetables, fruits, and grains, and lower in red and processed meats, and low in sweets were associated with lower risk of developing of neural tube defects, particularly among women who do not take folic acid supplements. Whereas some dietary patterns were associated with lower risk of developing anencephaly, others were associated with lower risk of developing spina bifida.

Evidence is insufficient to determine an association between maternal dietary patterns and congenital heart defects or cleft lip/palate.

All studies were consistent in demonstrating that folic acid supplementation periconceptionally was associated with a decreased risk of having a child with a birth defect (e.g. neural tube defects, congenital heart defects, and cleft lip/palate). **DGAC Grade: Neural Tube Defects – Limited; Congenital Heart Defects – Grade not assignable; Cleft Lip/Palate – Grade not assignable**

Implications

Women of reproductive age should consume folic acid in the form of a supplement or through fortified foods in the range recommended by the U.S. Preventive Services Task Force (400 to 800 micrograms) in addition to consuming a diet rich in vegetables, fruits, and grains; lower in red and processed meats; and low in sweets.

Review of the Evidence

This series of systematic reviews included five case-control studies (using data from three cohorts) published since 1980 that examined the relationship between maternal dietary patterns and congenital anomalies in infants.[146-150] Three articles examined neural tube defects,[146, 147, 149] two articles examined congenital heart defects,[147, 150] and two articles examined orofacial clefts.[146, 148]

Although all five case-control studies reported significant associations between dietary patterns and risk of congenital anomalies in women not taking folic acid supplementation, the variability of dietary patterns methodology used and composition of dietary patterns identified made it difficult to draw conclusions. All studies were consistent in finding that folate delivered periconceptionally in food or as a supplement as a key nutrient was associated with lower risk of developing congenital anomalies. It should be noted that some of the included studies were conducted in countries with mandatory folate fortification, while others were from countries that prohibit such fortification.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3356

DIETARY PATTERNS AND NEUROLOGICAL AND PSYCHOLOGICAL ILLNESSES

Existing Evidence around Foods and Nutrients and Neurological and Psychological Illnesses

Neuropsychological development and function is increasingly recognized as a high national priority for health promotion and chronic disease prevention. Two major components of neuropsychological function are *cognition*, the ability to reason, and *mood*, balanced and appropriate to enable optimal cognition.

Nutrition for optimal neurodevelopment in very young children has long been a subject of research. The 2010 DGAC concluded that moderate evidence supported a positive relationship between maternal dietary intakes of n-3 from seafood and improved cognitive ability in infants.[151] The rising numbers of U.S. older adults and the potential human and financial cost of age-related cognitive impairments, such as Alzheimer's disease and other dementias, also have helped drive national interest in chronic mental disease.[152, 153] Separately, depression affected 8 percent of Americans for at least two weeks annually from 2007-2010, and of these, 80 percent report functional impairment.[154] Many preclinical and human studies have established relationships between traditional nutrients (e.g., omega-3 fatty acids) and central nervous system composition and function. Studies appearing in the last few years reflect the increasing research interest in the links between diet and neurological health.

The hypothesis that nutrition can reduce and/or play a role in the treatment of these mental diseases and their related burdens has been studied in relation to several nutrients and foods, including the B vitamins, vitamin E, and selenium.[155, 156] The omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are among the most studied nutrients for neural health, in part because DHA is a major component of the brain, specifically gray matter and its synapses, and the specialized light detecting cells of the retina. DHA, in particular, supports the amplitude and signaling speed of neural response. EPA has emerged as a nutrient with antidepressive properties and continued studies to define its role in prevention and therapy are underway. Sufficiently strong medical evidence has been obtained for EPA and DHA such that supplements are now considered as complementary therapy for major depressive disorder by the American Psychiatric Association[157] and more recent data from a meta-analysis has found them effective.[158] Before 2010, the number of published dietary pattern studies was small. However, a more substantial literature on dietary patterns and neuropsychological health has been published since 2010. The DGAC was therefore able to consider prevention of adult neuropsychological ill health for the first time.

Question 6: What is the relationship between dietary patterns and risk of neurological and psychological illnesses?

Source of evidence: NEL systematic review

Conclusion

Limited evidence suggests that a dietary pattern containing an array of vegetables, fruits, nuts, legumes and seafood consumed during adulthood is associated with lower risk of age-related cognitive impairment, dementia, and/or Alzheimer's disease. Although the number of studies available on dietary patterns and neurodegenerative disease risk is expanding, this body of evidence, which is made up of high-quality observational studies, has appeared only in recent years and is rapidly developing. It employs a wide range of methodology in study design, definition and measurement ascertainment of cognitive outcomes, and dietary pattern assessment. **DGAC Grade: Limited**

Limited evidence suggests that dietary patterns emphasizing seafood, vegetables, fruits, nuts, and legumes are associated with lower risk of depression in men and non-perinatal women. However, the body of evidence is primarily composed of observational studies and employs a range of methodology in study design, definition, and measurement of dietary patterns and ascertainment of depression/depressive signs and symptoms. Studies on dietary patterns in other populations, such as women in the post-partum period, children and adolescents, as well as those in various ethnic and cultural groups, are too limited to draw conclusions. **DGAC Grade: Adults – Limited; Children, adolescents, and women in the post-partum period – Grade not assignable**

Implications

Dietary patterns emphasizing vegetables, fruits, seafood, legumes and nuts similar to those that achieve chronic disease risk reduction are consistent with maintaining neurocognitive health, including cognitive ability in healthy aging, and balanced mood.

Review of the Evidence

Dietary Patterns and Cognitive Impairment, Dementia, and Alzheimer's Disease

This systematic review includes 30 articles (two articles analyzed data taken from RCTs and 28 articles used data from prospective cohort studies) published since 1980 (with all but two published since 2008) that examined the relationship between dietary patterns and age-related cognitive impairment, dementia, and/or Alzheimer's disease.[159-188] Twenty of the articles included in this review assessed the relationship between dietary patterns and cognitive impairment, 10 articles examined cognitive impairment or dementia, and eight articles looked at Alzheimer's disease.

The articles used several different methods to assess dietary patterns. Two articles analyzed data from RCTs that tested or described dietary patterns, 23 articles used indices/scores to assess dietary patterns quality or adherence, three articles used data-driven methods, and three used reduced rank regression. Most (18 of 28) articles found an association between dietary patterns and age-related cognitive impairment, dementia, and/or Alzheimer's disease. Despite some heterogeneity in this body of evidence, some common elements of dietary patterns were associated with measures of cognitive impairment, dementia, and/or Alzheimer's disease:

- Patterns higher in vegetables, fruits, nuts, legumes, and seafood were generally associated with reduced risk of age-related cognitive impairment, dementia, and/or Alzheimer's disease.
- Patterns higher in red and/or processed meats were generally associated with greater age-related cognitive impairment. Relatively few studies reported on refined sugar and added salt, and patterns including these nutrients tended to report greater cognitive impairment.

Although some studies included participants from a range of race/ethnic and socioeconomic groups, the results are most applicable to the general healthy aging population. In addition, dietary patterns were derived using dietary intake measured at baseline only, and therefore, may not reflect patterns consumed throughout relevant periods of life before enrollment in the study, or changes in intake that may have occurred over the duration of the study. Similarly, several studies measured cognitive function only at a single time point (follow-up), and therefore, could not assess change in cognitive function over time. Finally, though these studies controlled for a number of confounders, not all apparently relevant potential confounders were adjusted for (e.g., existing or family history of cognitive decline, dementia, or Alzheimer's disease; baseline health status; changes in dietary intake over time) and, as with all association studies, residual confounding is possible.

Dietary Patterns and Depression

This systematic review includes nineteen articles (17 from prospective cohort studies, and 2 using data from RCTs) published since 1980 (all of which were published since 2008) that assessed the relationship between dietary patterns and depression.[175, 182, 189-205]

The articles used several different methods to assess dietary patterns. Two studies tested the effects of dietary patterns as part of an RCT, six articles used indices/scores to assess dietary patterns, 10 articles used data-driven methods, and one used reduced rank regression. Despite methodological and outcome heterogeneity in this body of evidence, some protective dietary patterns emerged:

- Patterns emphasizing seafood, vegetables, fruits, and nuts, were generally associated with reduced risk of depression.
- Patterns emphasizing red and processed meats and refined sugar were generally associated with increased risk of depression.

This body of evidence did have several limitations. There was considerable variability in how the outcome of depression was assessed, with some studies using various depression scales, some using physician diagnosis/hospital discharge records, and others using proxies such as use of depression medication. Although most studies make extensive efforts to include participants across a wide range of race/ethnic groups and across the socio-economic continuum, there still may be some subgroups for which the association between dietary patterns and depression risk cannot be reliably assessed and therefore conclusions cannot be drawn for them. Research is needed to determine whether dietary patterns are associated with risk of depression in particularly vulnerable subgroups, specifically children, adolescents, young adults, and women during the post-partum period. Additional limitations within this body of evidence make it difficult to draw stronger conclusions, including assessment of dietary patterns and depression outcomes at a single point in time, potential for residual confounding despite adjustment for a number of factors, and few studies conducted in U.S.-based populations.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3352

DIETARY PATTERNS AND BONE HEALTH

Existing Evidence around Foods and Nutrients and Bone Health

Low bone mineral density and osteoporosis are common in the United States, particularly in older adults, and its contribution to disability and cost to the health care system continues to rise in parallel to longer life expectancy. As described in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***, more than half of women ages 60 to 69 years have low bone mass and approximately 12 percent meet established criteria for osteoporosis. The prevalence of osteoporosis increases with age; about one-quarter of women ages 70 to 79 years and about one-third of women older than age 80 years have osteoporosis. Low bone mass is less common in older men but is increasingly recognized. Among U.S. men ages 60 to 69 years, about a third have low bone mass and this increases to about 40 percent and slightly more than 50 percent for men ages 70 to 79 years and 80 years and older, respectively.

Poor bone health and osteoporotic fractures are a major cause of morbidity and mortality in the elderly and account for significant health care costs. Understanding the extent to which dietary factors can help improve bone health and reduce the incidence of fractures across all segments of the population, particularly in the elderly, is important for the health and well-being of the nation.

The most critical nutrients for healthy bone are calcium, vitamin D, and phosphorous. As part of their 2011 report on Calcium and Vitamin D, the Institute of Medicine extensively reviewed the available data and updated the Dietary Reference Intakes (DRIs) for calcium and vitamin D for men and women across life stages.[206] The new reference values were based upon a strong body of evidence regarding bone growth and maintenance. At the time of the report, these bone health outcomes (in particular bone mass [bone mineral content]) were the only indicators on which there was sufficient scientific evidence to define DRIs; a thorough review of other outcomes (bone mineral density, risk of fractures, and osteoporosis) provided mixed and inconclusive results, and thus did not inform the DRIs. ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** of this DGAC report concluded that calcium and vitamin D were shortfall nutrients of public health concern. The estimated low levels of intake in various age and sex groups place many at risk for suboptimal bone health. The DGAC asked additional questions regarding bone health that went beyond those relating to the role of specific and well-known nutrients on bone remodeling. Specifically, the DGAC considered the influence of dietary patterns and their relationship to bone health and specific bone health outcomes across the lifespan, including bone density and fractures. This approach enabled the DGAC to consider the relationship between the total diet and its component foods and nutrients, acting in combination, on bone health outcomes. This section reviews this evidence and forms the basis for the DGAC recommendation for action at individual and population level as well as its research recommendations.

Question 7: What is the relationship between dietary patterns and bone health?

Source of evidence: NEL systematic review

Conclusion

Limited evidence suggests that a dietary pattern higher in vegetables, fruits, grains, nuts, and dairy products, and lower in meats and saturated fat, is associated with more favorable bone health outcomes in adults, including decreased risk of fracture and osteoporosis, as well as improved bone mineral density. Although a growing number of studies are examining the relationship between dietary patterns and bone health in adults, the number of high-quality studies is modest and those available employ a wide range of methodologies in study design, dietary assessment techniques, and varying bone health outcomes.

Definitive conclusions regarding the relationship between dietary patterns and bone health outcomes (bone mineral density and bone mineral content) in children and adolescents cannot be drawn due to the limited evidence from a small number of studies with wide variation in study design, dietary assessment methodology, and bone health outcomes. **DGAC Grade: Adults – Limited; Children and Adolescents - Grade not assignable**

Implications

Only limited evidence is available on the relationships between *dietary patterns* and bone health outcomes in adults and other age groups. Although there is strong evidence on the roles of vitamin D and calcium in bone health across the age spectrum, further research is needed on dietary patterns that are most beneficial.

Review of the Evidence

This systematic review included two articles that used data from RCTs and 11 articles from prospective cohort studies published since 2000 that examined the relationship between dietary patterns and bone health.[207-219]

The articles employ diverse methodologies to assess dietary patterns. Four articles used an index or score, six articles used factor analysis/principal components analysis, two articles used reduced rank regression, and two articles tested dietary patterns in an intervention study where bone health or fractures were either secondary or tertiary trial outcomes. Seven studies assessed risk of fracture, six studies assessed bone mineral density, bone mineral content, or bone mass, and one study examined risk of osteoporosis. The dietary patterns examined in this systematic review were defined in various ways, making comparisons between articles difficult. However, despite heterogeneity in this body of evidence, some common characteristics of dietary patterns associated with better or adverse bone health outcomes emerged, particularly in articles where patterns were defined by index or score. Articles using data-driven methods were less consistent. The following overall conclusions can be drawn:

- Patterns emphasizing vegetables, fruits, legumes, nuts, dairy, and cereals/grains/pasta/rice, and unsaturated fats were generally associated with more favorable bone health outcomes.
- Patterns higher in meats and saturated fats were generally associated with increased risk of adverse bone health outcomes.
- Results were far less consistent for added sugars, alcohol, and sodium in relation to bone health.

Although many cohort studies make extensive efforts to include participants across a wide range of race/ethnic groups and across the socio-economic continuum, there still may be some groups for which the association between dietary patterns and bone health cannot yet be determined (i.e., children, adolescents).

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3360

CHAPTER SUMMARY

The dietary patterns approach captures the relationship between the overall diet and its constituent foods, beverages, and nutrients in relationship to outcomes of interest. Numerous dietary patterns were identified, with the most common ones defined using indices or scores such as the HEI-2010, the AHEI-2010, or various Mediterranean-style dietary patterns, the DASH pattern, vegetarian patterns, and data-driven approaches.

The Committee's examination of the association between dietary patterns and various health outcomes revealed remarkable consistency in the findings and implications that are noteworthy. When looking at the dietary pattern conclusion statements across the various health outcomes, certain characteristics of the diet were consistently identified (see Table D2.3). Common characteristics of dietary patterns associated with positive health outcomes include higher intake of vegetables, fruits, whole grains, low- or non-fat dairy, seafood, legumes, and nuts; moderate intake of alcohol (among adults); lower consumption of red and processed meat, and low intake of sugar-sweetened foods and drinks, and refined grains. Vegetables and fruits are the only characteristics of the diet that were consistently identified in every conclusion statement across the health outcomes. Whole grains were identified slightly less consistently compared to vegetables and fruits, but were identified in every conclusion with moderate to strong evidence. For studies with limited evidence, grains were not as consistently defined and/or they were not identified as a key characteristic. Low- or non-fat dairy, seafood, legumes, nuts, and alcohol were identified as beneficial characteristics of the diet for some, but not all, outcomes. For conclusions with moderate to strong evidence, higher intake of red and processed meats was identified as detrimental compared to lower intake. Higher consumption of sugar-sweetened foods and beverages as well as refined grains were identified as detrimental in almost all conclusion statements with moderate to strong evidence.

Table D2.3. Description of the dietary patterns highlighted in the DGAC's Conclusion Statements that are associated with benefit related to the health outcome of interest. **(Note: The reader is directed to the full Conclusion Statement above for more information on the relationship between dietary patterns and the health outcome. In some cases, dietary components were associated with increased health risk and this is noted in the table.)**

Health Outcome	DGAC Grade[a]	Description of the Dietary Pattern Associated with Beneficial Health Outcomes
Cardiovascular disease	Strong	Dietary patterns characterized by higher consumption of *vegetables, fruits, whole grains, low-fat dairy,* and *seafood*, and lower consumption of *red and processed meat*, and lower intakes of *refined grains,* and *sugar-sweetened foods and beverages* relative to less healthy patterns; regular consumption of *nuts* and *legumes;* moderate consumption of *alcohol*; lower in *saturated fat, cholesterol,* and *sodium* and richer in *fiber, potassium,* and *unsaturated fats*.
Measures of body weight or obesity	Moderate Limited	Dietary patterns that are higher in *vegetables, fruits,* and *whole grains*; include *seafood* and *legumes*; are moderate in *dairy products (particularly low and non-fat dairy)* and *alcohol*; lower in *meats (including red and processed meats),* and low in *sugar-sweetened foods and beverages*, and *refined grains*; higher intakes of *unsaturated fats* and lower intakes of *saturated fats, cholesterol*, and *sodium*. Dietary patterns in childhood or adolescence that are higher in energy-dense and low-fiber foods, such as *sweets, refined grains,* and *processed meats*, as well as *sugar-sweetened beverages, whole milk, fried potatoes, certain fats and oils,* and *fast foods* are associated with an increased risk.
Type 2 diabetes	Moderate	Dietary patterns higher in *vegetables, fruits,* and *whole grains* and lower in *red* and *processed meats, high-fat dairy products, refined grains,* and *sweets/sugar-sweetened beverages.*
Cancer	Moderate Moderate (post) / Limited (pre) Limited Not assignable	Colon/Rectal Cancer: Dietary patterns that are higher in *vegetables, fruits, legumes, whole grains, lean meats/seafood,* and *low-fat dairy* and moderate in *alcohol*; and low in *red and/or processed meats, saturated fat,* and *sodas/sweets*. (Conversely, diets that are higher in *red/processed meats*, *French fries/potatoes*, and *sources of sugars (i.e., sodas, sweets, and dessert foods)* are associated with a greater risk.) Breast Cancer: Dietary patterns rich in *vegetables, fruit,* and *whole grains,* and lower in *animal products* and *refined carbohydrate*. Lung Cancer: Dietary patterns containing more frequent servings of *vegetables, fruits, seafood, grains/cereals,* and *legumes*, and *lean versus higher fat meats* and *lower fat or non-fat dairy products*. Prostate Cancer: N/A
Congenital anomalies	Limited – Neural tube defects Not assignable	Neural tube defects: Dietary patterns during the preconception period that are higher in *vegetables, fruits,* and *grains*, and lower in *red and processed meats*, and low in *sweets.* Congenital heart defects or cleft lip/palate: N/A
Neurological and psychological illnesses	Limited Limited	Age-related cognitive impairment, dementia, and/or Alzheimer's disease: Dietary patterns containing an array of *vegetables, fruits, nuts, legumes* and *seafood.* Depression: Dietary patterns emphasizing *seafood, vegetables, fruits, nuts,* and *legumes.*
Bone health	Limited Not assignable	Adults: Dietary patterns higher in *vegetables, fruits, grains, nuts,* and *dairy products,* and lower in *meats* and *saturated fat*. Children: N/A

[a] The DGAC Grade presented represents the grade the Committee provided for the conclusion statement with the dietary pattern components described. Some health outcomes had more than one graded conclusion. Only the conclusion statements that describe dietary pattern components are presented here. Post = Post-menopausal; Pre = Pre-menopausal

As alcohol is a unique aspect of the diet, the DGAC considered evidence from several sources to inform recommendations. As noted above, moderate alcohol intake among adults was identified as a component of a healthy dietary pattern associated with some health outcomes, which reaffirms conclusions related to moderate alcohol consumption by the 2010 DGAC. The Committee also concurs with the conclusions reached by the 2010 DGAC on the relationship between alcohol intake and unintentional injury and lactation.[1] However, as noted in Table D2.1, evidence also suggests that alcoholic drinks are associated with increased risk for certain cancers, including pre- and post-menopausal breast cancer. After consideration of this collective evidence, the Committee concurs with the 2010 DGAC that if alcohol is consumed, it should be consumed in moderation, and only by adults. However, it is not recommended that anyone begin drinking or drink more frequently on the basis of potential health benefits because moderate alcohol intake also is associated with increased risk of violence, drowning, and injuries from falls and motor vehicle crashes. Women should be aware of a moderately increased risk of breast cancer even with moderate alcohol intake. There are many circumstances in which people should not drink alcohol:

- Individuals who cannot restrict their drinking to moderate levels.
- Anyone younger than the legal drinking age.
- Women who are pregnant or who may be pregnant.
- Individuals taking prescription or over-the-counter medications that can interact with alcohol.
- Individuals with certain specific medical conditions (e.g., liver disease, hypertriglyceridemia, pancreatitis).
- Individuals who plan to drive, operate machinery, or take part in other activities that require attention, skill, or coordination or in situations where impaired judgment could cause injury or death (e.g., swimming).

Finally, because of the substantial evidence clearly demonstrating the health benefits of breastfeeding, occasionally consuming an alcoholic drink does not warrant stopping breastfeeding. However, women who are breastfeeding should be very cautious about drinking alcohol, if they choose to drink at all.[§§]

The common characteristics of a healthy dietary pattern found in the conclusion statements across the outcomes examined implies that following a dietary pattern associated with reduced risk of CVD, overweight, and obesity will have positive health benefits beyond these categories

[§§] If the infant's breastfeeding behavior is well established, consistent, and predictable (no earlier than at 3 months of age), a mother may consume a single alcoholic drink if she then waits at least 4 hours before breastfeeding. Alternatively, she may express breast milk before consuming the drink and feed the expressed milk to her infant later.

of health outcomes. Thus, the U.S. population should be encouraged and guided to consume dietary patterns that are rich in vegetables, fruits, whole grains, seafood, legumes, and nuts; moderate in low- and non-fat dairy products and alcohol (among adults); lower in red and processed meat; and low in sugar-sweetened foods and beverages and refined grains. These dietary patterns can be achieved in many ways and should be tailored to the individual's biological and medical needs as well as socio-cultural preferences. As described in the DGAC's conceptual model, a multi-level process at individual and population levels is required to help achieve a healthy diet and other lifestyle behaviors so as to achieve chronic disease risk reduction and overall well-being. The Committee recommends the development and implementation of programs and services that facilitate the improvement in eating behaviors consistent with healthy dietary patterns in various settings, including preventive services in our healthcare and public health systems as well as those that reach populations in other settings of influence such as preschool and school settings and workplaces.

The dietary pattern characteristics being recommended by the 2015 DGAC reaffirms the dietary pattern characteristics recommended by the 2010 DGAC, despite the fact that different approaches were employed. Additionally, this dietary pattern aligns with recommendations from other groups, including AICR and AHA/ACC. The majority of evidence considered focuses on dietary patterns consumed in adulthood on health risks, primarily risks of chronic disease development and, in the case of pregnancy, birth defects. Very little evidence considered here was directed to dietary patterns in children, and risk reduction studies evaluating children's diets and risk of overweight and obesity provided limited evidence. No conclusions on chronic disease apply directly to evidence developed in children. Recommendations based on adult studies have implications for children based on general nutritional principles but caution is warranted, considering the fact that children with developing bodies and neurocognitive capabilities present unique nutritional issues.

NEEDS FOR FUTURE RESEARCH

1. Conduct additional dietary patterns research for other health outcomes to strengthen the evidence beyond CVD and body weight in populations of various ethnic backgrounds and life course stages in order for future DGACs to draw stronger conclusions.

   Rationale: The NEL systematic reviews demonstrated that considerable CVD research related to dietary patterns is available. However, it also is important to note, that unlike CVD, some of the other health outcomes are more heterogeneous and thus may require greater specificity in the examination of diet and disease risk. There is a clear need for all studies examining the relationship between dietary patterns and health outcomes to include the full age spectrum and to take a life course perspective (including pregnancy); insufficient research is being devoted to children and how diseases may evolve over time. An increased emphasis should be placed on understanding how the diets of all those in the U.S. population from various ethnic backgrounds may be associated with health outcomes, thereby broadening knowledge beyond Hispanics and African Americans to include the diversity that exists in the United States today. This may require our national nutrition monitoring programs to over-sample individuals from other national origins to conduct subgroup analysis.

2. Improve the understanding of how to more precisely characterize dietary patterns by their food constituents and the implications of the food constituents on nutrient adequacy through the use of Food Pattern Modeling. More precise characterization, particularly of protein foods, is needed.

   Rationale: Researchers are characterizing dietary patterns very differently and yet sometimes use similar nomenclatures. This makes it difficult to compare results across studies and as demonstrated in the NEL systematic reviews, can impair the grading of the body of evidence as strong. The reason why researchers are not replicating others findings in different populations may be a function of publication bias. It is important for editors of scientific journals and peer reviewers to appreciate the replication of findings first and then value a research group's methodological nuance that may improve the examination of the association between dietary patterns and health outcomes. Perhaps what should be stressed is a harmonization of research methods across various cohorts or randomized trials, similar to what is being done at the National Cancer Institute's Dietary Patterns Methods Project[9, 220] led by Drs. Krebs-Smith and Reedy. The use of Food Pattern Modeling as demonstrated in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** allows questions about the adequacy of the dietary patterns given specific food constituents to be addressed and how modifications of the patterns by altering the foods for specific population groups or to meet specific nutrient targets can be achieved.

3. Examine the long-term cardio-metabolic effects of the various dietary patterns identified in the *AHA/ACC/TOS Guidelines for the Management of Overweight and Obesity in Adults* that are capable of resulting in short-term weight loss (see Question 2, above).

   Rationale: Although the research to date demonstrates that to lose weight, a variety of dietary pattern approaches can be used if a reduction in caloric intake is achieved, the long-term effects of these diets on cardio-metabolic health are not well known. Emerging research is exploring health effects of variations of the low-carbohydrate, higher protein/fat dietary pattern. In some approaches (such as Atkins), the dietary pattern which emphasizes animal products, may achieve a macronutrient composition that is higher in saturated fat. Others may emphasize plant-based proteins and fats and may achieve a lower saturated fat content and may be higher in polyunsaturated fats and dietary fiber. Research is needed to determine the impact of these alternative approaches, and perhaps others, on CVD risk profiles as well as other health outcomes. As mentioned in the review of the literature associated with saturated fat and cardiovascular disease in ***Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance***, substituting one macronutrient for another may result in unintended consequences. Careful consideration to the types of foods that are used in these diets and in particular the type of fat and amount of added sugars should be taken into account.

REFERENCES

1. Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010, to the Secretary of Agriculture and the Secretary of Health and Human Services: U.S. Department of Agriculture, Agricultural Research Service, Washington D.C.; 2010. Available from: http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm.
2. Nutrition Evidence Library. A series of systematic reviews on the relationship between dietary patterns and health outcomes. Alexandria, VA: U.S. Department of Agriculture, Center for Nutrition Policy and Promotion; 2014. Available from: http://www.nel.gov/vault/2440/web/files/DietaryPatterns/DPRptFullFinal.pdf.
3. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, et al. Update of the Healthy Eating Index: HEI-2010. J Acad Nutr Diet. 2013;113(4):569-80. PMID: 23415502. http://www.ncbi.nlm.nih.gov/pubmed/23415502.
4. Chiuve SE, Fung TT, Rimm EB, Hu FB, McCullough ML, Wang M, et al. Alternative dietary indices both strongly predict risk of chronic disease. J Nutr. 2012;142(6):1009-18. PMID: 22513989. http://www.ncbi.nlm.nih.gov/pubmed/22513989.
5. Kant AK, Schatzkin A, Graubard BI, Schairer C. A prospective study of diet quality and mortality in women. JAMA. 2000;283(16):2109-15. PMID: 10791502. http://www.ncbi.nlm.nih.gov/pubmed/10791502.
6. Fung TT, Chiuve SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. Arch Intern Med. 2008;168(7):713-20. PMID: 18413553. http://www.ncbi.nlm.nih.gov/pubmed/18413553.
7. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348(26):2599-608. PMID: 12826634. http://www.ncbi.nlm.nih.gov/pubmed/12826634.
8. Fung TT, Rexrode KM, Mantzoros CS, Manson JE, Willett WC, Hu FB. Mediterranean diet and incidence of and mortality from coronary heart disease and stroke in women. Circulation. 2009;119(8):1093-100. PMID: 19221219. http://www.ncbi.nlm.nih.gov/pubmed/19221219.
9. Reedy J, Krebs-Smith SM, Miller PE, Liese AD, Kahle LL, Park Y, et al. Higher diet quality is associated with decreased risk of all-cause, cardiovascular disease, and cancer mortality among older adults. J Nutr. 2014;144(6):881-9. PMID: 24572039. http://www.ncbi.nlm.nih.gov/pubmed/24572039.

10. National Heart Lung and Blood Institute. Lifestyle Interventions to Reduce Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health; 2013. Available from: http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/lifestyle/index.htm.
11. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63(25 Pt B):2960-84. PMID: 24239922. http://www.ncbi.nlm.nih.gov/pubmed/24239922.
12. National Heart Lung and Blood Institute. Managing overweight and obesity in adults: Systematic evidence review from the Obesity Expert Panel, 2013. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health; 2013. Available from: http://www.nhlbi.nih.gov/guidelines/obesity/ser/index.htm.
13. Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. J Am Coll Cardiol. 2014;63(25 Pt B):2985-3023. PMID: 24239920. http://www.ncbi.nlm.nih.gov/pubmed/24239920.
14. Huang T, Yang B, Zheng J, Li G, Wahlqvist ML, Li D. Cardiovascular disease mortality and cancer incidence in vegetarians: a meta-analysis and systematic review. Ann Nutr Metab. 2012;60(4):233-40. PMID: 22677895. http://www.ncbi.nlm.nih.gov/pubmed/22677895.
15. Martinez-Gonzalez MA, Bes-Rastrollo M. Dietary patterns, Mediterranean diet, and cardiovascular disease. Curr Opin Lipidol. 2014;25(1):20-6. PMID: 24370845. http://www.ncbi.nlm.nih.gov/pubmed/24370845.
16. Rees K, Hartley L, Flowers N, Clarke A, Hooper L, Thorogood M, et al. 'Mediterranean' dietary pattern for the primary prevention of cardiovascular disease. Cochrane Database Syst Rev. 2013;8:CD009825. PMID: 23939686. http://www.ncbi.nlm.nih.gov/pubmed/23939686.
17. Salehi-Abargouei A, Maghsoudi Z, Shirani F, Azadbakht L. Effects of Dietary Approaches to Stop Hypertension (DASH)-style diet on fatal or nonfatal cardiovascular diseases--incidence: a systematic review and meta-analysis on observational prospective studies. Nutrition. 2013;29(4):611-8. PMID: 23466047. http://www.ncbi.nlm.nih.gov/pubmed/23466047.
18. Sofi F, Macchi C, Abbate R, Gensini GF, Casini A. Mediterranean diet and health status: an updated meta-analysis and a proposal for a literature-based adherence score. Public Health Nutr. 2013:1-14. PMID: 24476641. http://www.ncbi.nlm.nih.gov/pubmed/24476641.
19. Yokoyama Y, Nishimura K, Barnard ND, Takegami M, Watanabe M, Sekikawa A, et al. Vegetarian diets and blood pressure: a meta-analysis. JAMA Intern Med. 2014;174(4):577-87. PMID: 24566947. http://www.ncbi.nlm.nih.gov/pubmed/24566947.
20. Dauchet L, Kesse-Guyot E, Czernichow S, Bertrais S, Estaquio C, Péneau S, et al. Dietary patterns and blood pressure change over 5-y follow-up in the SU.VI.MAX cohort. Am J Clin Nutr. 2007;85(6):1650-6. PMID: 17556705. http://www.ncbi.nlm.nih.gov/pubmed/17556705.

21. Camões M, Oliveira A, Pereira M, Severo M, Lopes C. Role of physical activity and diet in incidence of hypertension: a population-based study in Portuguese adults. Eur J Clin Nutr. 2010;64(12):1441-9. PMID: 20808327. http://www.ncbi.nlm.nih.gov/pubmed/20808327.
22. Folsom AR, Parker ED, Harnack LJ. Degree of concordance with DASH diet guidelines and incidence of hypertension and fatal cardiovascular disease. Am J Hypertens. 2007;20(3):225-32. PMID: 17324731. http://www.ncbi.nlm.nih.gov/pubmed/17324731.
23. Estruch R, Martínez-González MA, Corella D, Salas-Salvadó J, Ruiz-Gutiérrez V, Covas MI, et al. Effects of a Mediterranean-style diet on cardiovascular risk factors: a randomized trial. Ann Intern Med. 2006;145(1):1-11. PMID: 16818923. http://www.ncbi.nlm.nih.gov/pubmed/16818923.
24. Jula A, Marniemi J, Huupponen R, Virtanen A, Rastas M, Rönnemaa T. Effects of diet and simvastatin on serum lipids, insulin, and antioxidants in hypercholesterolemic men: a randomized controlled trial. JAMA. 2002;287(5):598-605. PMID: 11829698. http://www.ncbi.nlm.nih.gov/pubmed/11829698.
25. Núñez-Córdoba JM, Valencia-Serrano F, Toledo E, Alonso A, Martínez-González MA. The Mediterranean diet and incidence of hypertension: the Seguimiento Universidad de Navarra (SUN) Study. Am J Epidemiol. 2009;169(3):339-46. PMID: 19037007. http://www.ncbi.nlm.nih.gov/pubmed/19037007.
26. Burr ML, Butland BK. Heart disease in British vegetarians. Am J Clin Nutr. 1988;48(3 Suppl):830-2. PMID: 3414590. http://www.ncbi.nlm.nih.gov/pubmed/3414590.
27. Margetts BM, Beilin LJ, Armstrong BK, Vandongen R. A randomized control trial of a vegetarian diet in the treatment of mild hypertension. Clin Exp Pharmacol Physiol. 1985;12(3):263-6. PMID: 3896594. http://www.ncbi.nlm.nih.gov/pubmed/3896594.
28. Crowe FL, Appleby PN, Travis RC, Key TJ. Risk of hospitalization or death from ischemic heart disease among British vegetarians and nonvegetarians: results from the EPIC-Oxford cohort study. Am J Clin Nutr. 2013;97(3):597-603. PMID: 23364007. http://www.ncbi.nlm.nih.gov/pubmed/23364007.
29. Zamora D, Gordon-Larsen P, He K, Jacobs DR, Shikany JM, Popkin BM. Are the 2005 Dietary Guidelines for Americans Associated With reduced risk of type 2 diabetes and cardiometabolic risk factors? Twenty-year findings from the CARDIA study. Diabetes Care. 2011;34(5):1183-5. PMID: 21478463. http://www.ncbi.nlm.nih.gov/pubmed/21478463.
30. van de Laar RJ, Stehouwer CD, van Bussel BC, Prins MH, Twisk JW, Ferreira I. Adherence to a Mediterranean dietary pattern in early life is associated with lower arterial stiffness in adulthood: the Amsterdam Growth and Health Longitudinal Study. J Intern Med. 2013;273(1):79-93. PMID: 22809371. http://www.ncbi.nlm.nih.gov/pubmed/22809371.
31. Rumawas ME, Meigs JB, Dwyer JT, McKeown NM, Jacques PF. Mediterranean-style dietary pattern, reduced risk of metabolic syndrome traits, and incidence in the Framingham Offspring Cohort. Am J Clin Nutr. 2009;90(6):1608-14. PMID: 19828705. http://www.ncbi.nlm.nih.gov/pubmed/19828705.
32. Tortosa A, Bes-Rastrollo M, Sanchez-Villegas A, Basterra-Gortari FJ, Nuñez-Cordoba JM, Martinez-Gonzalez MA. Mediterranean diet inversely associated with the incidence of metabolic syndrome: the SUN prospective cohort. Diabetes Care. 2007;30(11):2957-9. PMID: 17712023. http://www.ncbi.nlm.nih.gov/pubmed/17712023.

33. Estruch R, Ros E, Martínez-González MA. Mediterranean diet for primary prevention of cardiovascular disease. N Engl J Med. 2013;369(7):676-7. PMID: 23944307. http://www.ncbi.nlm.nih.gov/pubmed/23944307.

34. Ambrosini GL. Childhood dietary patterns and later obesity: a review of the evidence. Proc Nutr Soc. 2014;73(1):137-46. PMID: 24280165. http://www.ncbi.nlm.nih.gov/pubmed/24280165.

35. Esposito K, Kastorini CM, Panagiotakos DB, Giugliano D. Mediterranean diet and weight loss: meta-analysis of randomized controlled trials. Metab Syndr Relat Disord. 2011;9(1):1-12. PMID: 20973675. http://www.ncbi.nlm.nih.gov/pubmed/20973675.

36. Beunza JJ, Toledo E, Hu FB, Bes-Rastrollo M, Serrano-Martínez M, Sánchez-Villegas A, et al. Adherence to the Mediterranean diet, long-term weight change, and incident overweight or obesity: the Seguimiento Universidad de Navarra (SUN) cohort. Am J Clin Nutr. 2010;92(6):1484-93. PMID: 20962161. http://www.ncbi.nlm.nih.gov/pubmed/20962161.

37. Mendez MA, Popkin BM, Jakszyn P, Berenguer A, Tormo MJ, Sanchéz MJ, et al. Adherence to a Mediterranean diet is associated with reduced 3-year incidence of obesity. J Nutr. 2006;136(11):2934-8. PMID: 17056825. http://www.ncbi.nlm.nih.gov/pubmed/17056825.

38. Romaguera D, Norat T, Vergnaud AC, Mouw T, May AM, Agudo A, et al. Mediterranean dietary patterns and prospective weight change in participants of the EPIC-PANACEA project. Am J Clin Nutr. 2010;92(4):912-21. PMID: 20810975. http://www.ncbi.nlm.nih.gov/pubmed/20810975.

39. Berz JP, Singer MR, Guo X, Daniels SR, Moore LL. Use of a DASH food group score to predict excess weight gain in adolescent girls in the National Growth and Health Study. Arch Pediatr Adolesc Med. 2011;165(6):540-6. PMID: 21646587. http://www.ncbi.nlm.nih.gov/pubmed/21646587.

40. Cheng G, Gerlach S, Libuda L, Kranz S, Günther AL, Karaolis-Danckert N, et al. Diet quality in childhood is prospectively associated with the timing of puberty but not with body composition at puberty onset. J Nutr. 2010;140(1):95-102. PMID: 19923386. http://www.ncbi.nlm.nih.gov/pubmed/19923386.

41. Gao SK, Beresford SA, Frank LL, Schreiner PJ, Burke GL, Fitzpatrick AL. Modifications to the Healthy Eating Index and its ability to predict obesity: the Multi-Ethnic Study of Atherosclerosis. Am J Clin Nutr. 2008;88(1):64-9. PMID: 18614725. http://www.ncbi.nlm.nih.gov/pubmed/18614725.

42. Kesse-Guyot E, Castetbon K, Estaquio C, Czernichow S, Galan P, Hercberg S. Association between the French nutritional guideline-based score and 6-year anthropometric changes in a French middle-aged adult cohort. Am J Epidemiol. 2009;170(6):757-65. PMID: 19656810. http://www.ncbi.nlm.nih.gov/pubmed/19656810.

43. Zamora D, Gordon-Larsen P, Jacobs DR, Popkin BM. Diet quality and weight gain among black and white young adults: the Coronary Artery Risk Development in Young Adults (CARDIA) Study (1985-2005). Am J Clin Nutr. 2010;92(4):784-93. PMID: 20685947. http://www.ncbi.nlm.nih.gov/pubmed/20685947.

44. Lassale C, Fezeu L, Andreeva VA, Hercberg S, Kengne AP, Czernichow S, et al. Association between dietary scores and 13-year weight change and obesity risk in a French prospective cohort. Int J Obes (Lond). 2012;36(11):1455-62. PMID: 22249228. http://dx.doi.org/10.1038/ijo.2011.264.

45. Woo J, Cheung B, Ho S, Sham A, Lam TH. Influence of dietary pattern on the development of overweight in a Chinese population. Eur J Clin Nutr. 2008;62(4):480-7. PMID: 17327865. http://www.ncbi.nlm.nih.gov/pubmed/17327865.

46. Jacobs DR, Sluik D, Rokling-Andersen MH, Anderssen SA, Drevon CA. Association of 1-y changes in diet pattern with cardiovascular disease risk factors and adipokines: results from the 1-y randomized Oslo Diet and Exercise Study. Am J Clin Nutr. 2009;89(2):509-17. PMID: 19116328. http://www.ncbi.nlm.nih.gov/pubmed/19116328.

47. Duffey KJ, Steffen LM, Van Horn L, Jacobs DR, Popkin BM. Dietary patterns matter: diet beverages and cardiometabolic risks in the longitudinal Coronary Artery Risk Development in Young Adults (CARDIA) Study. Am J Clin Nutr. 2012;95(4):909-15. PMID: 22378729. http://www.ncbi.nlm.nih.gov/pubmed/22378729.

48. Hosseini-Esfahani F, Djazaieri SA, Mirmiran P, Mehrabi Y, Azizi F. Which food patterns are predictors of obesity in Tehranian adults? J Nutr Educ Behav. 2012;44(6):564-73. PMID: 21652267. http://www.ncbi.nlm.nih.gov/pubmed/21652267.

49. McNaughton SA, Mishra GD, Stephen AM, Wadsworth ME. Dietary patterns throughout adult life are associated with body mass index, waist circumference, blood pressure, and red cell folate. J Nutr. 2007;137(1):99-105. PMID: 17182808. http://www.ncbi.nlm.nih.gov/pubmed/17182808.

50. Newby PK, Muller D, Hallfrisch J, Qiao N, Andres R, Tucker KL. Dietary patterns and changes in body mass index and waist circumference in adults. Am J Clin Nutr. 2003;77(6):1417-25. PMID: 12791618. http://www.ncbi.nlm.nih.gov/pubmed/12791618.

51. Newby PK, Muller D, Hallfrisch J, Andres R, Tucker KL. Food patterns measured by factor analysis and anthropometric changes in adults. Am J Clin Nutr. 2004;80(2):504-13. PMID: 15277177. http://www.ncbi.nlm.nih.gov/pubmed/15277177.

52. Togo P, Osler M, Sørensen TI, Heitmann BL. A longitudinal study of food intake patterns and obesity in adult Danish men and women. Int J Obes Relat Metab Disord. 2004;28(4):583-93. PMID: 14770197. http://www.ncbi.nlm.nih.gov/pubmed/14770197.

53. Boggs DA, Palmer JR, Spiegelman D, Stampfer MJ, Adams-Campbell LL, Rosenberg L. Dietary patterns and 14-y weight gain in African American women. Am J Clin Nutr. 2011;94(1):86-94. PMID: 21593501. http://www.ncbi.nlm.nih.gov/pubmed/21593501.

54. Newby PK, Weismayer C, Akesson A, Tucker KL, Wolk A. Longitudinal changes in food patterns predict changes in weight and body mass index and the effects are greatest in obese women. J Nutr. 2006;136(10):2580-7. PMID: 16988130. http://www.ncbi.nlm.nih.gov/pubmed/16988130.

55. Quatromoni PA, Copenhafer DL, D'Agostino RB, Millen BE. Dietary patterns predict the development of overweight in women: The Framingham Nutrition Studies. J Am Diet Assoc. 2002;102(9):1239-46. PMID: 12792620. http://www.ncbi.nlm.nih.gov/pubmed/12792620.

56. Ritchie LD, Spector P, Stevens MJ, Schmidt MM, Schreiber GB, Striegel-Moore RH, et al. Dietary patterns in adolescence are related to adiposity in young adulthood in black and white females. J Nutr. 2007;137(2):399-406. PMID: 17237318. http://www.ncbi.nlm.nih.gov/pubmed/17237318.

57. Schulze MB, Fung TT, Manson JE, Willett WC, Hu FB. Dietary patterns and changes in body weight in women. Obesity (Silver Spring). 2006;14(8):1444-53. PMID: 16988088. http://www.ncbi.nlm.nih.gov/pubmed/16988088.

58. Alhazmi A, Stojanovski E, McEvoy M, Garg ML. The association between dietary patterns and type 2 diabetes: a systematic review and meta-analysis of cohort studies. J Hum Nutr Diet. 2014;27(3):251-60. PMID: 24102939. http://www.ncbi.nlm.nih.gov/pubmed/24102939.

59. Liese AD, Nichols M, Sun X, D'Agostino RB, Haffner SM. Adherence to the DASH Diet is inversely associated with incidence of type 2 diabetes: the insulin resistance atherosclerosis study. Diabetes Care. 2009;32(8):1434-6. PMID: 19487638. http://www.ncbi.nlm.nih.gov/pubmed/19487638.

60. Martínez-González MA, de la Fuente-Arrillaga C, Nunez-Cordoba JM, Basterra-Gortari FJ, Beunza JJ, Vazquez Z, et al. Adherence to Mediterranean diet and risk of developing diabetes: prospective cohort study. BMJ. 2008;336(7657):1348-51. PMID: 18511765. http://www.ncbi.nlm.nih.gov/pubmed/18511765.

61. Rossi M, Turati F, Lagiou P, Trichopoulos D, Augustin LS, La Vecchia C, et al. Mediterranean diet and glycaemic load in relation to incidence of type 2 diabetes: results from the Greek cohort of the population-based European Prospective Investigation into Cancer and Nutrition (EPIC). Diabetologia. 2013;56(11):2405-13. PMID: 23975324. http://www.ncbi.nlm.nih.gov/pubmed/23975324.

62. Abiemo EE, Alonso A, Nettleton JA, Steffen LM, Bertoni AG, Jain A, et al. Relationships of the Mediterranean dietary pattern with insulin resistance and diabetes incidence in the Multi-Ethnic Study of Atherosclerosis (MESA). Br J Nutr. 2013;109(8):1490-7. PMID: 22932232. http://www.ncbi.nlm.nih.gov/pubmed/22932232.

63. Fung TT, McCullough M, van Dam RM, Hu FB. A prospective study of overall diet quality and risk of type 2 diabetes in women. Diabetes Care. 2007;30(7):1753-7. PMID: 17429059. http://www.ncbi.nlm.nih.gov/pubmed/17429059.

64. Gopinath B, Rochtchina E, Flood VM, Mitchell P. Diet quality is prospectively associated with incident impaired fasting glucose in older adults. Diabet Med. 2013;30(5):557-62. PMID: 23301551. http://www.ncbi.nlm.nih.gov/pubmed/23301551.

65. von Ruesten A, Illner AK, Buijsse B, Heidemann C, Boeing H. Adherence to recommendations of the German food pyramid and risk of chronic diseases: results from the EPIC-Potsdam study. Eur J Clin Nutr. 2010;64(11):1251-9. PMID: 20717136. http://www.ncbi.nlm.nih.gov/pubmed/20717136.

66. Malik VS, Fung TT, van Dam RM, Rimm EB, Rosner B, Hu FB. Dietary patterns during adolescence and risk of type 2 diabetes in middle-aged women. Diabetes Care. 2012;35(1):12-8. PMID: 22074723. http://www.ncbi.nlm.nih.gov/pubmed/22074723.

67. Hodge AM, English DR, O'Dea K, Giles GG. Dietary patterns and diabetes incidence in the Melbourne Collaborative Cohort Study. Am J Epidemiol. 2007;165(6):603-10. PMID: 17220476. http://www.ncbi.nlm.nih.gov/pubmed/17220476.

68. Nanri A, Shimazu T, Takachi R, Ishihara J, Mizoue T, Noda M, et al. Dietary patterns and type 2 diabetes in Japanese men and women: the Japan Public Health Center-based Prospective Study. Eur J Clin Nutr. 2013;67(1):18-24. PMID: 23093343. http://www.ncbi.nlm.nih.gov/pubmed/23093343.

69. Nettleton JA, Steffen LM, Ni H, Liu K, Jacobs DR. Dietary patterns and risk of incident type 2 diabetes in the Multi-Ethnic Study of Atherosclerosis (MESA). Diabetes Care. 2008;31(9):1777-82. PMID: 18544792. http://www.ncbi.nlm.nih.gov/pubmed/18544792.

70. Liese AD, Weis KE, Schulz M, Tooze JA. Food intake patterns associated with incident type 2 diabetes: the Insulin Resistance Atherosclerosis Study. Diabetes Care. 2009;32(2):263-8. PMID: 19033409. http://www.ncbi.nlm.nih.gov/pubmed/19033409.
71. McNaughton SA, Mishra GD, Brunner EJ. Dietary patterns, insulin resistance, and incidence of type 2 diabetes in the Whitehall II Study. Diabetes Care. 2008;31(7):1343-8. PMID: 18390803. http://www.ncbi.nlm.nih.gov/pubmed/18390803.
72. Imamura F, Lichtenstein AH, Dallal GE, Meigs JB, Jacques PF. Generalizability of dietary patterns associated with incidence of type 2 diabetes mellitus. Am J Clin Nutr. 2009;90(4):1075-83. PMID: 19710193. http://www.ncbi.nlm.nih.gov/pubmed/19710193.
73. Adamsson V, Reumark A, Fredriksson IB, Hammarström E, Vessby B, Johansson G, et al. Effects of a healthy Nordic diet on cardiovascular risk factors in hypercholesterolaemic subjects: a randomized controlled trial (NORDIET). J Intern Med. 2011;269(2):150-9. PMID: 20964740. http://www.ncbi.nlm.nih.gov/pubmed/20964740.
74. Blumenthal JA, Babyak MA, Sherwood A, Craighead L, Lin PH, Johnson J, et al. Effects of the dietary approaches to stop hypertension diet alone and in combination with exercise and caloric restriction on insulin sensitivity and lipids. Hypertension. 2010;55(5):1199-205. PMID: 20212264. http://www.ncbi.nlm.nih.gov/pubmed/20212264.
75. Esposito K, Marfella R, Ciotola M, Di Palo C, Giugliano F, Giugliano G, et al. Effect of a mediterranean-style diet on endothelial dysfunction and markers of vascular inflammation in the metabolic syndrome: a randomized trial. JAMA. 2004;292(12):1440-6. PMID: 15383514. http://www.ncbi.nlm.nih.gov/pubmed/15383514.
76. Gadgil MD, Appel LJ, Yeung E, Anderson CA, Sacks FM, Miller ER. The effects of carbohydrate, unsaturated fat, and protein intake on measures of insulin sensitivity: results from the OmniHeart trial. Diabetes Care. 2013;36(5):1132-7. PMID: 23223345. http://www.ncbi.nlm.nih.gov/pubmed/23223345.
77. Rallidis LS, Lekakis J, Kolomvotsou A, Zampelas A, Vamvakou G, Efstathiou S, et al. Close adherence to a Mediterranean diet improves endothelial function in subjects with abdominal obesity. Am J Clin Nutr. 2009;90(2):263-8. PMID: 19515732. http://www.ncbi.nlm.nih.gov/pubmed/19515732.
78. Salas-Salvadó J, Fernández-Ballart J, Ros E, Martínez-González MA, Fitó M, Estruch R, et al. Effect of a Mediterranean diet supplemented with nuts on metabolic syndrome status: one-year results of the PREDIMED randomized trial. Arch Intern Med. 2008;168(22):2449-58. PMID: 19064829. http://www.ncbi.nlm.nih.gov/pubmed/19064829.
79. Salas-Salvadó J, Bulló M, Babio N, Martínez-González M, Ibarrola-Jurado N, Basora J, et al. Reduction in the incidence of type 2 diabetes with the Mediterranean diet: results of the PREDIMED-Reus nutrition intervention randomized trial. Diabetes Care. 2011;34(1):14-9. PMID: 20929998. http://www.ncbi.nlm.nih.gov/pubmed/20929998.
80. Tonstad S, Stewart K, Oda K, Batech M, Herring RP, Fraser GE. Vegetarian diets and incidence of diabetes in the Adventist Health Study-2. Nutr Metab Cardiovasc Dis. 2013;23(4):292-9. PMID: 21983060. http://www.ncbi.nlm.nih.gov/pubmed/21983060.
81. Kimokoti RW, Gona P, Zhu L, Newby PK, Millen BE, Brown LS, et al. Dietary patterns of women are associated with incident abdominal obesity but not metabolic syndrome. J Nutr. 2012;142(9):1720-7. PMID: 22833658. http://www.ncbi.nlm.nih.gov/pubmed/22833658.

82. Lau C, Toft U, Tetens I, Carstensen B, Jørgensen T, Pedersen O, et al. Dietary patterns predict changes in two-hour post-oral glucose tolerance test plasma glucose concentrations in middle-aged adults. J Nutr. 2009;139(3):588-93. PMID: 19158222. http://www.ncbi.nlm.nih.gov/pubmed/19158222.
83. Willett W. Nutritional Epidemiology. New York: Oxford University Press; 1988.
84. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, and the Prevention of Cancer: a Global Perspective. Washington D.C.: AICR; 1997.
85. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington D.C.: AICR; 2007.
86. World Cancer Research Fund/American Institute for Cancer Research. Continuous Update Project (CUP) 2014 [cited 2014 November 6]. Available from: http://www.dietandcancerreport.org/cup/.
87. World Cancer Research Fund/American Institute for Cancer Research. Chapter 3: Judging the Evidence. Food Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington D.C.: AICR; 2007. p. 48-62.
88. Agnoli C, Grioni S, Sieri S, Palli D, Masala G, Sacerdote C, et al. Italian Mediterranean Index and risk of colorectal cancer in the Italian section of the EPIC cohort. Int J Cancer. 2013;132(6):1404-11. PMID: 22821300. http://www.ncbi.nlm.nih.gov/pubmed/22821300.
89. Bamia C, Lagiou P, Buckland G, Grioni S, Agnoli C, Taylor AJ, et al. Mediterranean diet and colorectal cancer risk: results from a European cohort. Eur J Epidemiol. 2013;28(4):317-28. PMID: 23579425. http://www.ncbi.nlm.nih.gov/pubmed/23579425.
90. Beresford SA, Johnson KC, Ritenbaugh C, Lasser NL, Snetselaar LG, Black HR, et al. Low-fat dietary pattern and risk of colorectal cancer: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA. 2006;295(6):643-54. PMID: 16467233. http://www.ncbi.nlm.nih.gov/pubmed/16467233.
91. Butler LM, Wang R, Koh WP, Yu MC. Prospective study of dietary patterns and colorectal cancer among Singapore Chinese. Br J Cancer. 2008;99(9):1511-6. PMID: 18813309. http://www.ncbi.nlm.nih.gov/pubmed/18813309.
92. Dixon LB, Balder HF, Virtanen MJ, Rashidkhani B, Mannisto S, Krogh V, et al. Dietary patterns associated with colon and rectal cancer: results from the Dietary Patterns and Cancer (DIETSCAN) Project. Am J Clin Nutr. 2004;80(4):1003-11. PMID: 15447912. http://www.ncbi.nlm.nih.gov/pubmed/15447912.
93. Doubeni CA, Major JM, Laiyemo AO, Schootman M, Zauber AG, Hollenbeck AR, et al. Contribution of behavioral risk factors and obesity to socioeconomic differences in colorectal cancer incidence. J Natl Cancer Inst. 2012;104(18):1353-62. PMID: 22952311. http://www.ncbi.nlm.nih.gov/pubmed/22952311.
94. Engeset D, Dyachenko A, Ciampi A, Lund E. Dietary patterns and risk of cancer of various sites in the Norwegian European Prospective Investigation into Cancer and Nutrition cohort: the Norwegian Women and Cancer study. Eur J Cancer Prev. 2009;18(1):69-75. PMID: 19077568. http://www.ncbi.nlm.nih.gov/pubmed/19077568.
95. Flood A, Rastogi T, Wirfalt E, Mitrou PN, Reedy J, Subar AF, et al. Dietary patterns as identified by factor analysis and colorectal cancer among middle-aged Americans. Am J Clin Nutr. 2008;88(1):176-84. PMID: 18614739. http://www.ncbi.nlm.nih.gov/pubmed/18614739.

96. Fung T, Hu FB, Fuchs C, Giovannucci E, Hunter DJ, Stampfer MJ, et al. Major dietary patterns and the risk of colorectal cancer in women. Arch Intern Med. 2003;163(3):309-14. PMID: 12578511. http://www.ncbi.nlm.nih.gov/pubmed/12578511.
97. Fung TT, Hu FB, Wu K, Chiuve SE, Fuchs CS, Giovannucci E. The Mediterranean and Dietary Approaches to Stop Hypertension (DASH) diets and colorectal cancer. Am J Clin Nutr. 2010;92(6):1429-35. PMID: 21097651. http://www.ncbi.nlm.nih.gov/pubmed/21097651.
98. Fung TT, Hu FB, Schulze M, Pollak M, Wu T, Fuchs CS, et al. A dietary pattern that is associated with C-peptide and risk of colorectal cancer in women. Cancer Causes Control. 2012;23(6):959-65. PMID: 22535146. http://www.ncbi.nlm.nih.gov/pubmed/22535146.
99. Jarvandi S, Davidson NO, Schootman M. Increased risk of colorectal cancer in type 2 diabetes is independent of diet quality. PLoS One. 2013;8(9):e74616. PMID: 24069323. http://www.ncbi.nlm.nih.gov/pubmed/24069323.
100. Kesse E, Clavel-Chapelon F, Boutron-Ruault MC. Dietary patterns and risk of colorectal tumors: a cohort of French women of the National Education System (E3N). Am J Epidemiol. 2006;164(11):1085-93. PMID: 16990408. http://www.ncbi.nlm.nih.gov/pubmed/16990408.
101. Key TJ, Appleby PN, Spencer EA, Travis RC, Roddam AW, Allen NE. Cancer incidence in vegetarians: results from the European Prospective Investigation into Cancer and Nutrition (EPIC-Oxford). Am J Clin Nutr. 2009;89(5):1620S-6S. PMID: 19279082. http://www.ncbi.nlm.nih.gov/pubmed/19279082.
102. Kim MK, Sasaki S, Otani T, Tsugane S, Japan Public Health Center-based Prospective Study G. Dietary patterns and subsequent colorectal cancer risk by subsite: a prospective cohort study. Int J Cancer. 2005;115(5):790-8. PMID: 15704172. http://www.ncbi.nlm.nih.gov/pubmed/15704172.
103. Kyro C, Skeie G, Loft S, Overvad K, Christensen J, Tjonneland A, et al. Adherence to a healthy Nordic food index is associated with a lower incidence of colorectal cancer in women: the Diet, Cancer and Health cohort study. Br J Nutr. 2013;109(5):920-7. PMID: 22874538. http://www.ncbi.nlm.nih.gov/pubmed/22874538.
104. Mai V, Kant AK, Flood A, Lacey JV, Jr., Schairer C, Schatzkin A. Diet quality and subsequent cancer incidence and mortality in a prospective cohort of women. Int J Epidemiol. 2005;34(1):54-60. PMID: 15649959. http://www.ncbi.nlm.nih.gov/pubmed/15649959.
105. Miller PE, Cross AJ, Subar AF, Krebs-Smith SM, Park Y, Powell-Wiley T, et al. Comparison of 4 established DASH diet indexes: examining associations of index scores and colorectal cancer. Am J Clin Nutr. 2013;98(3):794-803. PMID: 23864539. http://www.ncbi.nlm.nih.gov/pubmed/23864539.
106. Reedy J, Mitrou PN, Krebs-Smith SM, Wirfalt E, Flood A, Kipnis V, et al. Index-based dietary patterns and risk of colorectal cancer: the NIH-AARP Diet and Health Study. Am J Epidemiol. 2008;168(1):38-48. PMID: 18525082. http://www.ncbi.nlm.nih.gov/pubmed/18525082.
107. Wirfalt E, Midthune D, Reedy J, Mitrou P, Flood A, Subar AF, et al. Associations between food patterns defined by cluster analysis and colorectal cancer incidence in the NIH-AARP diet and health study. Eur J Clin Nutr. 2009;63(6):707-17. PMID: 18685556. http://www.ncbi.nlm.nih.gov/pubmed/18685556.

108. Wu K, Hu FB, Fuchs C, Rimm EB, Willett WC, Giovannucci E. Dietary patterns and risk of colon cancer and adenoma in a cohort of men (United States). Cancer Causes Control. 2004;15(9):853-62. PMID: 15577287. http://www.ncbi.nlm.nih.gov/pubmed/15577287.

109. Terry P, Hu FB, Hansen H, Wolk A. Prospective study of major dietary patterns and colorectal cancer risk in women. Am J Epidemiol. 2001;154(12):1143-9. PMID: 11744520. http://www.ncbi.nlm.nih.gov/pubmed/11744520.

110. Adebamowo CA, Hu FB, Cho E, Spiegelman D, Holmes MD, Willett WC. Dietary patterns and the risk of breast cancer. Ann Epidemiol. 2005;15(10):789-95. PMID: 16257363. http://www.ncbi.nlm.nih.gov/pubmed/16257363.

111. Agurs-Collins T, Rosenberg L, Makambi K, Palmer JR, Adams-Campbell L. Dietary patterns and breast cancer risk in women participating in the Black Women's Health Study. Am J Clin Nutr. 2009;90(3):621-8. PMID: 19587089. http://www.ncbi.nlm.nih.gov/pubmed/19587089.

112. Baglietto L, Krishnan K, Severi G, Hodge A, Brinkman M, English DR, et al. Dietary patterns and risk of breast cancer. Br J Cancer. 2011;104(3):524-31. PMID: 21157446. http://www.ncbi.nlm.nih.gov/pubmed/21157446.

113. Buckland G, Travier N, Cottet V, Gonzalez CA, Lujan-Barroso L, Agudo A, et al. Adherence to the mediterranean diet and risk of breast cancer in the European prospective investigation into cancer and nutrition cohort study. Int J Cancer. 2013;132(12):2918-27. PMID: 23180513. http://www.ncbi.nlm.nih.gov/pubmed/23180513.

114. Butler LM, Wu AH, Wang R, Koh WP, Yuan JM, Yu MC. A vegetable-fruit-soy dietary pattern protects against breast cancer among postmenopausal Singapore Chinese women. Am J Clin Nutr. 2010;91(4):1013-9. PMID: 20181808. http://www.ncbi.nlm.nih.gov/pubmed/20181808.

115. Cade JE, Taylor EF, Burley VJ, Greenwood DC. Common dietary patterns and risk of breast cancer: analysis from the United Kingdom Women's Cohort Study. Nutr Cancer. 2010;62(3):300-6. PMID: 20358467. http://www.ncbi.nlm.nih.gov/pubmed/20358467.

116. Cade JE, Taylor EF, Burley VJ, Greenwood DC. Does the Mediterranean dietary pattern or the Healthy Diet Index influence the risk of breast cancer in a large British cohort of women? Eur J Clin Nutr. 2011;65(8):920-8. PMID: 21587285. http://www.ncbi.nlm.nih.gov/pubmed/21587285.

117. Cottet V, Touvier M, Fournier A, Touillaud MS, Lafay L, Clavel-Chapelon F, et al. Postmenopausal breast cancer risk and dietary patterns in the E3N-EPIC prospective cohort study. Am J Epidemiol. 2009;170(10):1257-67. PMID: 19828509. http://www.ncbi.nlm.nih.gov/pubmed/19828509.

118. Couto E, Sandin S, Lof M, Ursin G, Adami HO, Weiderpass E. Mediterranean dietary pattern and risk of breast cancer. PLoS One. 2013;8(2):e55374. PMID: 23390532. http://www.ncbi.nlm.nih.gov/pubmed/23390532.

119. Fung TT, Hu FB, Holmes MD, Rosner BA, Hunter DJ, Colditz GA, et al. Dietary patterns and the risk of postmenopausal breast cancer. Int J Cancer. 2005;116(1):116-21. PMID: 15756679. http://www.ncbi.nlm.nih.gov/pubmed/15756679.

120. Fung TT, Hu FB, McCullough ML, Newby PK, Willett WC, Holmes MD. Diet quality is associated with the risk of estrogen receptor-negative breast cancer in postmenopausal women. J Nutr. 2006;136(2):466-72. PMID: 16424129. http://www.ncbi.nlm.nih.gov/pubmed/16424129.

121. Fung TT, Hu FB, Hankinson SE, Willett WC, Holmes MD. Low-carbohydrate diets, dietary approaches to stop hypertension-style diets, and the risk of postmenopausal breast cancer. Am J Epidemiol. 2011;174(6):652-60. PMID: 21832271. http://www.ncbi.nlm.nih.gov/pubmed/21832271.

122. Fung TT, Schulze MB, Hu FB, Hankinson SE, Holmes MD. A dietary pattern derived to correlate with estrogens and risk of postmenopausal breast cancer. Breast Cancer Res Treat. 2012;132(3):1157-62. PMID: 22218885. http://www.ncbi.nlm.nih.gov/pubmed/22218885.

123. Link LB, Canchola AJ, Bernstein L, Clarke CA, Stram DO, Ursin G, et al. Dietary patterns and breast cancer risk in the California Teachers Study cohort. Am J Clin Nutr. 2013;98(6):1524-32. PMID: 24108781. http://www.ncbi.nlm.nih.gov/pubmed/24108781.

124. Mannisto S, Dixon LB, Balder HF, Virtanen MJ, Krogh V, Khani BR, et al. Dietary patterns and breast cancer risk: results from three cohort studies in the DIETSCAN project. Cancer Causes Control. 2005;16(6):725-33. PMID: 16049811. http://www.ncbi.nlm.nih.gov/pubmed/16049811.

125. Prentice RL, Caan B, Chlebowski RT, Patterson R, Kuller LH, Ockene JK, et al. Low-fat dietary pattern and risk of invasive breast cancer: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA. 2006;295(6):629-42. PMID: 16467232. http://www.ncbi.nlm.nih.gov/pubmed/16467232.

126. Sant M, Allemani C, Sieri S, Krogh V, Menard S, Tagliabue E, et al. Salad vegetables dietary pattern protects against HER-2-positive breast cancer: a prospective Italian study. Int J Cancer. 2007;121(4):911-4. PMID: 17455245. http://www.ncbi.nlm.nih.gov/pubmed/17455245.

127. Schulz M, Hoffmann K, Weikert C, Nothlings U, Schulze MB, Boeing H. Identification of a dietary pattern characterized by high-fat food choices associated with increased risk of breast cancer: the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam Study. Br J Nutr. 2008;100(5):942-6. PMID: 18377685. http://www.ncbi.nlm.nih.gov/pubmed/18377685.

128. Sieri S, Krogh V, Pala V, Muti P, Micheli A, Evangelista A, et al. Dietary patterns and risk of breast cancer in the ORDET cohort. Cancer Epidemiol Biomarkers Prev. 2004;13(4):567-72. PMID: 15066921. http://www.ncbi.nlm.nih.gov/pubmed/15066921.

129. Terry P, Suzuki R, Hu FB, Wolk A. A prospective study of major dietary patterns and the risk of breast cancer. Cancer Epidemiol Biomarkers Prev. 2001;10(12):1281-5. PMID: 11751446. http://www.ncbi.nlm.nih.gov/pubmed/11751446.

130. Trichopoulou A, Bamia C, Lagiou P, Trichopoulos D. Conformity to traditional Mediterranean diet and breast cancer risk in the Greek EPIC (European Prospective Investigation into Cancer and Nutrition) cohort. Am J Clin Nutr. 2010;92(3):620-5. PMID: 20631204. http://www.ncbi.nlm.nih.gov/pubmed/20631204.

131. Velie EM, Schairer C, Flood A, He JP, Khattree R, Schatzkin A. Empirically derived dietary patterns and risk of postmenopausal breast cancer in a large prospective cohort study. Am J Clin Nutr. 2005;82(6):1308-19. PMID: 16332665. http://www.ncbi.nlm.nih.gov/pubmed/16332665.

132. Balder HF, Goldbohm RA, van den Brandt PA. Dietary patterns associated with male lung cancer risk in the Netherlands Cohort Study. Cancer Epidemiol Biomarkers Prev. 2005;14(2):483-90. PMID: 15734976. http://www.ncbi.nlm.nih.gov/pubmed/15734976.

133. Gnagnarella P, Maisonneuve P, Bellomi M, Rampinelli C, Bertolotti R, Spaggiari L, et al. Red meat, Mediterranean diet and lung cancer risk among heavy smokers in the COSMOS screening study. Ann Oncol. 2013;24(10):2606-11. PMID: 23956193. http://www.ncbi.nlm.nih.gov/pubmed/23956193.
134. Ax E, Garmo H, Grundmark B, Bill-Axelson A, Holmberg L, Becker W, et al. Dietary patterns and prostate cancer risk: report from the population based ULSAM cohort study of swedish men. Nutr Cancer. 2014;66(1):77-87. PMID: 24325263. http://www.ncbi.nlm.nih.gov/pubmed/24325263.
135. Bosire C, Stampfer MJ, Subar AF, Park Y, Kirkpatrick SI, Chiuve SE, et al. Index-based dietary patterns and the risk of prostate cancer in the NIH-AARP diet and health study. Am J Epidemiol. 2013;177(6):504-13. PMID: 23408548. http://www.ncbi.nlm.nih.gov/pubmed/23408548.
136. Kenfield SA, DuPre N, Richman EL, Stampfer MJ, Chan JM, Giovannucci EL. Mediterranean diet and prostate cancer risk and mortality in the health professionals follow-up study. Eur Urol. 2014;65(5):887-94. PMID: 23962747. http://www.ncbi.nlm.nih.gov/pubmed/23962747.
137. Muller DC, Severi G, Baglietto L, Krishnan K, English DR, Hopper JL, et al. Dietary patterns and prostate cancer risk. Cancer Epidemiol Biomarkers Prev. 2009;18(11):3126-9. PMID: 19861522. http://www.ncbi.nlm.nih.gov/pubmed/19861522.
138. Tseng M, Breslow RA, DeVellis RF, Ziegler RG. Dietary patterns and prostate cancer risk in the National Health and Nutrition Examination Survey Epidemiological Follow-up Study cohort. Cancer Epidemiol Biomarkers Prev. 2004;13(1):71-7. PMID: 14744736. http://www.ncbi.nlm.nih.gov/pubmed/14744736.
139. Wu K, Hu FB, Willett WC, Giovannucci E. Dietary patterns and risk of prostate cancer in U.S. men. Cancer Epidemiol Biomarkers Prev. 2006;15(1):167-71. PMID: 16434606. http://www.ncbi.nlm.nih.gov/pubmed/16434606.
140. Wolff T, Witkop CT, Miller T, Syed SB. Folic Acid Supplementation for the Prevention of Neural Tube Defects: An Update of the Evidence for the U.S. Preventive Services Task Force. Evidence Synthesis No. 70. AHRQ Publication No. 09-051132-EF-1. Rockville, Maryland: Agency for Healthcare Research and Quality.2009 May.
141. Pitkin RM. Folate and neural tube defects. Am J Clin Nutr. 2007;85(1):285S-8S. PMID: 17209211. http://www.ncbi.nlm.nih.gov/pubmed/17209211.
142. Yang Q, Cogswell ME, Hamner HC, Carriquiry A, Bailey LB, Pfeiffer CM, et al. Folic acid source, usual intake, and folate and vitamin B-12 status in US adults: National Health and Nutrition Examination Survey (NHANES) 2003-2006. Am J Clin Nutr. 2010;91(1):64-72. PMID: 19828716. http://www.ncbi.nlm.nih.gov/pubmed/19828716.
143. Bailey RL, Dodd KW, Gahche JJ, Dwyer JT, McDowell MA, Yetley EA, et al. Total folate and folic acid intake from foods and dietary supplements in the United States: 2003-2006. Am J Clin Nutr. 2010;91(1):231-7. PMID: 19923379. http://www.ncbi.nlm.nih.gov/pubmed/19923379.
144. U. S. Preventive Services Task Force. Folic Acid to Prevent Neural Tube Defects: Preventive Medication. Recommendation Summary May 2009 [cited 2014 November]. Available from: http://www.uspreventiveservicestaskforce.org/uspstf/uspsnrfol.htm.
145. Food and Nutrition Board - Institute of Medicine. Dietary Reference Intakes: The Essential Guide to Nutrient Requirements. Washington, DC: The National Academies Press; 2006.

146. Carmichael SL, Yang W, Feldkamp ML, Munger RG, Siega-Riz AM, Botto LD, et al. Reduced risks of neural tube defects and orofacial clefts with higher diet quality. Arch Pediatr Adolesc Med. 2012;166(2):121-6. PMID: 21969361. http://www.ncbi.nlm.nih.gov/pubmed/21969361.

147. Sotres-Alvarez D, Siega-Riz AM, Herring AH, Carmichael SL, Feldkamp ML, Hobbs CA, et al. Maternal dietary patterns are associated with risk of neural tube and congenital heart defects. Am J Epidemiol. 2013;177(11):1279-88. PMID: 23639938. http://www.ncbi.nlm.nih.gov/pubmed/23639938.

148. Vujkovic M, Ocke MC, van der Spek PJ, Yazdanpanah N, Steegers EA, Steegers-Theunissen RP. Maternal Western dietary patterns and the risk of developing a cleft lip with or without a cleft palate. Obstet Gynecol. 2007;110(2 Pt 1):378-84. PMID: 17666614. http://www.ncbi.nlm.nih.gov/pubmed/17666614.

149. Vujkovic M, Steegers EA, Looman CW, Ocke MC, van der Spek PJ, Steegers-Theunissen RP. The maternal Mediterranean dietary pattern is associated with a reduced risk of spina bifida in the offspring. BJOG. 2009;116(3):408-15. PMID: 19187373. http://www.ncbi.nlm.nih.gov/pubmed/19187373.

150. Obermann-Borst SA, Vujkovic M, de Vries JH, Wildhagen MF, Looman CW, de Jonge R, et al. A maternal dietary pattern characterised by fish and seafood in association with the risk of congenital heart defects in the offspring. BJOG. 2011;118(10):1205-15. PMID: 21585642. http://www.ncbi.nlm.nih.gov/pubmed/21585642.

151. Nutrition Evidence Library; U.S. Department of Agriculture. What are the effects of maternal dietary intake of omega-3 fatty acids on breast milk composition and infant health outcomes? [cited 2014 November]. Available from: http://nel.gov/evidence.cfm?evidence_summary_id=250144.

152. Hebert LE, Weuve J, Scherr PA, Evans DA. Alzheimer disease in the United States (2010-2050) estimated using the 2010 census. Neurology. 2013;80(19):1778-83. PMID: 23390181. http://www.ncbi.nlm.nih.gov/pubmed/23390181.

153. Dorsey ER, George BP, Leff B, Willis AW. The coming crisis: obtaining care for the growing burden of neurodegenerative conditions. Neurology. 2013;80(21):1989-96. PMID: 23616157. http://www.ncbi.nlm.nih.gov/pubmed/23616157.

154. Centers for Disease Control and Prevention. QuickStats: Prevalence of Current Depression* Among Persons Aged ≥12 Years, by Age Group and Sex — United States, National Health and Nutrition Examination Survey, 2007–2010. Morbidity and Mortality Weekly Report (MMWR) [Internet]. 2012; 60(51):[1747 p.]. Available from: http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6051a7.htm?s_cid=mm6051a7_w.

155. Barnes JL, Tian M, Edens NK, Morris MC. Consideration of nutrient levels in studies of cognitive decline. Nutr Rev. 2014;72(11):707-19. PMID: 25323849. http://www.ncbi.nlm.nih.gov/pubmed/25323849.

156. Rita Cardoso B, Silva Bandeira V, Jacob-Filho W, Franciscato Cozzolino SM. Selenium status in elderly: Relation to cognitive decline. J Trace Elem Med Biol. 2014. PMID: 25220532. http://www.ncbi.nlm.nih.gov/pubmed/25220532.

157. Gelenberg AJ, Freeman MP, Markowitz JC, Rosenbaum JF, Thase ME, Trivedi MH, et al. Practice Guideline for the Treatment of Patients With Major Depressive Disorder, Third Edition. page 51 and page 92. 2010. Available from: http://psychiatryonline.org/pb/assets/raw/sitewide/practice_guidelines/guidelines/mdd.pdf.

158. Grosso G, Pajak A, Marventano S, Castellano S, Galvano F, Bucolo C, et al. Role of omega-3 fatty acids in the treatment of depressive disorders: a comprehensive meta-analysis of randomized clinical trials. PLoS One. 2014;9(5):e96905. PMID: 24805797. http://www.ncbi.nlm.nih.gov/pubmed/24805797.
159. Cherbuin N, Anstey KJ. The Mediterranean diet is not related to cognitive change in a large prospective investigation: the PATH Through Life study. Am J Geriatr Psychiatry. 2012;20(7):635-9. PMID: 21937919. http://www.ncbi.nlm.nih.gov/pubmed/21937919.
160. Eskelinen MH, Ngandu T, Tuomilehto J, Soininen H, Kivipelto M. Midlife healthy-diet index and late-life dementia and Alzheimer's disease. Dement Geriatr Cogn Dis Extra. 2011;1(1):103-12. PMID: 22163237. http://www.ncbi.nlm.nih.gov/pubmed/22163237.
161. Féart C, Samieri C, Rondeau V, Amieva H, Portet F, Dartigues JF, et al. Adherence to a Mediterranean diet, cognitive decline, and risk of dementia. JAMA. 2009;302(6):638-48. PMID: 19671905. http://www.ncbi.nlm.nih.gov/pubmed/19671905.
162. Gardener SL, Rainey-Smith SR, Barnes MB, Sohrabi HR, Weinborn M, Lim YY, et al. Dietary patterns and cognitive decline in an Australian study of ageing. Mol Psychiatry. 2014. PMID: 25070537. http://www.ncbi.nlm.nih.gov/pubmed/25070537.
163. Gu Y, Luchsinger JA, Stern Y, Scarmeas N. Mediterranean diet, inflammatory and metabolic biomarkers, and risk of Alzheimer's disease. J Alzheimers Dis. 2010;22(2):483-92. PMID: 20847399. http://www.ncbi.nlm.nih.gov/pubmed/20847399.
164. Gu Y, Nieves JW, Stern Y, Luchsinger JA, Scarmeas N. Food combination and Alzheimer disease risk: a protective diet. Arch Neurol. 2010;67(6):699-706. PMID: 20385883. http://www.ncbi.nlm.nih.gov/pubmed/20385883.
165. Kesse-Guyot E, Amieva H, Castetbon K, Henegar A, Ferry M, Jeandel C, et al. Adherence to nutritional recommendations and subsequent cognitive performance: findings from the prospective Supplementation with Antioxidant Vitamins and Minerals 2 (SU.VI.MAX 2) study. Am J Clin Nutr. 2011;93(1):200-10. PMID: 21106918. http://www.ncbi.nlm.nih.gov/pubmed/21106918.
166. Kesse-Guyot E, Andreeva VA, Jeandel C, Ferry M, Hercberg S, Galan P. A healthy dietary pattern at midlife is associated with subsequent cognitive performance. J Nutr. 2012;142(5):909-15. PMID: 22457391. http://www.ncbi.nlm.nih.gov/pubmed/22457391.
167. Kesse-Guyot E, Andreeva VA, Lassale C, Ferry M, Jeandel C, Hercberg S, et al. Mediterranean diet and cognitive function: a French study. Am J Clin Nutr. 2013;97(2):369-76. PMID: 23283500. http://www.ncbi.nlm.nih.gov/pubmed/23283500.
168. Kesse-Guyot E, Andreeva VA, Ducros V, Jeandel C, Julia C, Hercberg S, et al. Carotenoid-rich dietary patterns during midlife and subsequent cognitive function. Br J Nutr. 2014;111(5):915-23. PMID: 24073964. http://www.ncbi.nlm.nih.gov/pubmed/24073964.
169. Koyama A, Houston DK, Simonsick EM, Lee JS, Ayonayon HN, Shahar DR, et al. Association Between the Mediterranean Diet and Cognitive Decline in a Biracial Population. J Gerontol A Biol Sci Med Sci. 2014. PMID: 24994847. http://www.ncbi.nlm.nih.gov/pubmed/24994847.
170. Martínez-Lapiscina EH, Clavero P, Toledo E, Estruch R, Salas-Salvadó J, San Julián B, et al. Mediterranean diet improves cognition: the PREDIMED-NAVARRA randomised trial. J Neurol Neurosurg Psychiatry. 2013;84(12):1318-25. PMID: 23670794. http://www.ncbi.nlm.nih.gov/pubmed/23670794.

171. Nicolas A, Faisant C, Nourhashemi F, Lanzmann-Petithory D, Vellas B. Associations between nutritional intake and cognitive function in a healthy ageing sample: A 4-year reassessment. . European Journal of Geriatrics. 2000;2(3):114-9.

172. Olsson E, Karlström B, Kilander L, Byberg L, Cederholm T, Sjögren P. Dietary Patterns and Cognitive Dysfunction in a 12-Year Follow-up Study of 70 Year Old Men. J Alzheimers Dis. 2014. PMID: 25062901. http://www.ncbi.nlm.nih.gov/pubmed/25062901.

173. Ozawa M, Ninomiya T, Ohara T, Doi Y, Uchida K, Shirota T, et al. Dietary patterns and risk of dementia in an elderly Japanese population: the Hisayama Study. Am J Clin Nutr. 2013;97(5):1076-82. PMID: 23553168. http://www.ncbi.nlm.nih.gov/pubmed/23553168.

174. Parrott MD, Shatenstein B, Ferland G, Payette H, Morais JA, Belleville S, et al. Relationship between diet quality and cognition depends on socioeconomic position in healthy older adults. J Nutr. 2013;143(11):1767-73. PMID: 23986363. http://www.ncbi.nlm.nih.gov/pubmed/23986363.

175. Psaltopoulou T, Kyrozis A, Stathopoulos P, Trichopoulos D, Vassilopoulos D, Trichopoulou A. Diet, physical activity and cognitive impairment among elders: the EPIC-Greece cohort (European Prospective Investigation into Cancer and Nutrition). Public Health Nutr. 2008;11(10):1054-62. PMID: 18205988. http://www.ncbi.nlm.nih.gov/pubmed/18205988.

176. Roberts RO, Geda YE, Cerhan JR, Knopman DS, Cha RH, Christianson TJ, et al. Vegetables, unsaturated fats, moderate alcohol intake, and mild cognitive impairment. Dement Geriatr Cogn Disord. 2010;29(5):413-23. PMID: 20502015. http://www.ncbi.nlm.nih.gov/pubmed/20502015.

177. Samieri C, Grodstein F, Rosner BA, Kang JH, Cook NR, Manson JE, et al. Mediterranean diet and cognitive function in older age. Epidemiology. 2013;24(4):490-9. PMID: 23676264. http://www.ncbi.nlm.nih.gov/pubmed/23676264.

178. Samieri C, Okereke OI, E Devore E, Grodstein F. Long-term adherence to the Mediterranean diet is associated with overall cognitive status, but not cognitive decline, in women. J Nutr. 2013;143(4):493-9. PMID: 23365105. http://www.ncbi.nlm.nih.gov/pubmed/23365105.

179. Samieri C, Sun Q, Townsend MK, Chiuve SE, Okereke OI, Willett WC, et al. The association between dietary patterns at midlife and health in aging: an observational study. Ann Intern Med. 2013;159(9):584-91. PMID: 24189593. http://www.ncbi.nlm.nih.gov/pubmed/24189593.

180. Scarmeas N, Stern Y, Mayeux R, Manly JJ, Schupf N, Luchsinger JA. Mediterranean diet and mild cognitive impairment. Arch Neurol. 2009;66(2):216-25. PMID: 19204158. http://www.ncbi.nlm.nih.gov/pubmed/19204158.

181. Scarmeas N, Stern Y, Tang MX, Mayeux R, Luchsinger JA. Mediterranean diet and risk for Alzheimer's disease. Ann Neurol. 2006;59(6):912-21. PMID: 16622828. http://www.ncbi.nlm.nih.gov/pubmed/16622828.

182. Shatenstein B, Ferland G, Belleville S, Gray-Donald K, Kergoat MJ, Morais J, et al. Diet quality and cognition among older adults from the NuAge study. Exp Gerontol. 2012;47(5):353-60. PMID: 22386581. http://www.ncbi.nlm.nih.gov/pubmed/22386581.

183. Smith PJ, Blumenthal JA, Babyak MA, Craighead L, Welsh-Bohmer KA, Browndyke JN, et al. Effects of the dietary approaches to stop hypertension diet, exercise, and caloric restriction on neurocognition in overweight adults with high blood pressure.

Hypertension. 2010;55(6):1331-8. PMID: 20305128. http://www.ncbi.nlm.nih.gov/pubmed/20305128.

184. Tangney CC, Kwasny MJ, Li H, Wilson RS, Evans DA, Morris MC. Adherence to a Mediterranean-type dietary pattern and cognitive decline in a community population. Am J Clin Nutr. 2011;93(3):601-7. PMID: 21177796. http://www.ncbi.nlm.nih.gov/pubmed/21177796.

185. Titova OE, Ax E, Brooks SJ, Sjögren P, Cederholm T, Kilander L, et al. Mediterranean diet habits in older individuals: associations with cognitive functioning and brain volumes. Exp Gerontol. 2013;48(12):1443-8. PMID: 24126083. http://www.ncbi.nlm.nih.gov/pubmed/24126083.

186. Tsivgoulis G, Judd S, Letter AJ, Alexandrov AV, Howard G, Nahab F, et al. Adherence to a Mediterranean diet and risk of incident cognitive impairment. Neurology. 2013;80(18):1684-92. PMID: 23628929. http://www.ncbi.nlm.nih.gov/pubmed/23628929.

187. Wengreen H, Munger RG, Cutler A, Quach A, Bowles A, Corcoran C, et al. Prospective study of Dietary Approaches to Stop Hypertension- and Mediterranean-style dietary patterns and age-related cognitive change: the Cache County Study on Memory, Health and Aging. Am J Clin Nutr. 2013;98(5):1263-71. PMID: 24047922. http://www.ncbi.nlm.nih.gov/pubmed/24047922.

188. Wengreen HJ, Neilson C, Munger R, Corcoran C. Diet quality is associated with better cognitive test performance among aging men and women. J Nutr. 2009;139(10):1944-9. PMID: 19675102. http://www.ncbi.nlm.nih.gov/pubmed/19675102.

189. Akbaraly TN, Brunner EJ, Ferrie JE, Marmot MG, Kivimaki M, Singh-Manoux A. Dietary pattern and depressive symptoms in middle age. Br J Psychiatry. 2009;195(5):408-13. PMID: 19880930. http://www.ncbi.nlm.nih.gov/pubmed/19880930.

190. Akbaraly TN, Sabia S, Shipley MJ, Batty GD, Kivimaki M. Adherence to healthy dietary guidelines and future depressive symptoms: evidence for sex differentials in the Whitehall II study. Am J Clin Nutr. 2013;97(2):419-27. PMID: 23283506. http://www.ncbi.nlm.nih.gov/pubmed/23283506.

191. Chan R, Chan D, Woo J. A Prospective Cohort Study to Examine the Association between Dietary Patterns and Depressive Symptoms in Older Chinese People in Hong Kong. PLoS One. 2014;9(8):e105760. PMID: 25148515. http://www.ncbi.nlm.nih.gov/pubmed/25148515.

192. Chatzi L, Melaki V, Sarri K, Apostolaki I, Roumeliotaki T, Georgiou V, et al. Dietary patterns during pregnancy and the risk of postpartum depression: the mother-child 'Rhea' cohort in Crete, Greece. Public Health Nutr. 2011;14(9):1663-70. PMID: 21477412. http://www.ncbi.nlm.nih.gov/pubmed/21477412.

193. Chocano-Bedoya PO, O'Reilly EJ, Lucas M, Mirzaei F, Okereke OI, Fung TT, et al. Prospective study on long-term dietary patterns and incident depression in middle-aged and older women. Am J Clin Nutr. 2013;98(3):813-20. PMID: 23885043. http://www.ncbi.nlm.nih.gov/pubmed/23885043.

194. Jacka FN, Cherbuin N, Anstey KJ, Butterworth P. Dietary patterns and depressive symptoms over time: examining the relationships with socioeconomic position, health behaviours and cardiovascular risk. PLoS One. 2014;9(1):e87657. PMID: 24489946. http://www.ncbi.nlm.nih.gov/pubmed/24489946.

195. Le Port A, Gueguen A, Kesse-Guyot E, Melchior M, Lemogne C, Nabi H, et al. Association between dietary patterns and depressive symptoms over time: a 10-year follow-up study of the GAZEL cohort. PLoS One. 2012;7(12):e51593. PMID: 23251585. http://www.ncbi.nlm.nih.gov/pubmed/23251585.

196. Lucas M, Chocano-Bedoya P, Shulze MB, Mirzaei F, O'Reilly É, Okereke OI, et al. Inflammatory dietary pattern and risk of depression among women. Brain Behav Immun. 2014;36:46-53. PMID: 24095894. http://www.ncbi.nlm.nih.gov/pubmed/24095894.

197. McMartin SE, Kuhle S, Colman I, Kirk SF, Veugelers PJ. Diet quality and mental health in subsequent years among Canadian youth. Public Health Nutr. 2012;15(12):2253-8. PMID: 22414240. http://www.ncbi.nlm.nih.gov/pubmed/22414240.

198. Okubo H, Miyake Y, Sasaki S, Tanaka K, Murakami K, Hirota Y, et al. Dietary patterns during pregnancy and the risk of postpartum depression in Japan: the Osaka Maternal and Child Health Study. Br J Nutr. 2011;105(8):1251-7. PMID: 21144112. http://www.ncbi.nlm.nih.gov/pubmed/21144112.

199. Rienks J, Dobson AJ, Mishra GD. Mediterranean dietary pattern and prevalence and incidence of depressive symptoms in mid-aged women: results from a large community-based prospective study. Eur J Clin Nutr. 2013;67(1):75-82. PMID: 23212131. http://www.ncbi.nlm.nih.gov/pubmed/23212131.

200. Ruusunen A, Lehto SM, Mursu J, Tolmunen T, Tuomainen TP, Kauhanen J, et al. Dietary patterns are associated with the prevalence of elevated depressive symptoms and the risk of getting a hospital discharge diagnosis of depression in middle-aged or older Finnish men. J Affect Disord. 2014;159:1-6. PMID: 24679382. http://www.ncbi.nlm.nih.gov/pubmed/24679382.

201. Sánchez-Villegas A, Delgado-Rodríguez M, Alonso A, Schlatter J, Lahortiga F, Serra Majem L, et al. Association of the Mediterranean dietary pattern with the incidence of depression: the Seguimiento Universidad de Navarra/University of Navarra follow-up (SUN) cohort. Arch Gen Psychiatry. 2009;66(10):1090-8. PMID: 19805699. http://www.ncbi.nlm.nih.gov/pubmed/19805699.

202. Sánchez-Villegas A, Martínez-González MA, Estruch R, Salas-Salvadó J, Corella D, Covas MI, et al. Mediterranean dietary pattern and depression: the PREDIMED randomized trial. BMC Med. 2013;11:208. PMID: 24229349. http://www.ncbi.nlm.nih.gov/pubmed/24229349.

203. Skarupski KA, Tangney CC, Li H, Evans DA, Morris MC. Mediterranean diet and depressive symptoms among older adults over time. J Nutr Health Aging. 2013;17(5):441-5. PMID: 23636545. http://www.ncbi.nlm.nih.gov/pubmed/23636545.

204. Torres SJ, Nowson CA. A moderate-sodium DASH-type diet improves mood in postmenopausal women. Nutrition. 2012;28(9):896-900. PMID: 22480799. http://www.ncbi.nlm.nih.gov/pubmed/22480799.

205. Vilela AA, Farias DR, Eshriqui I, Vaz JoS, Franco-Sena AB, Castro MB, et al. Prepregnancy Healthy Dietary Pattern Is Inversely Associated with Depressive Symptoms among Pregnant Brazilian Women. J Nutr. 2014;144(10):1612-8. PMID: 25143375. http://www.ncbi.nlm.nih.gov/pubmed/25143375.

206. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium; Edited by A Catharine Ross CLT, Ann L Yaktine, and Heather B Del Valle. Dietary Reference Intakes for Calcium and Vitamin D. Washington (DC): National Academies Press (US); 2011.

207. Benetou V, Orfanos P, Pettersson-Kymmer U, Bergstrom U, Svensson O, Johansson I, et al. Mediterranean diet and incidence of hip fractures in a European cohort. Osteoporos Int. 2013;24(5):1587-98. PMID: 23085859. http://www.ncbi.nlm.nih.gov/pubmed/23085859.

208. Bullo M, Amigo-Correig P, Marquez-Sandoval F, Babio N, Martinez-Gonzalez MA, Estruch R, et al. Mediterranean diet and high dietary acid load associated with mixed nuts: effect on bone metabolism in elderly subjects. J Am Geriatr Soc. 2009;57(10):1789-98. PMID: 19807791. http://www.ncbi.nlm.nih.gov/pubmed/19807791.

209. Dai Z, Butler LM, van Dam RM, Ang LW, Yuan JM, Koh WP. Adherence to a vegetable-fruit-soy dietary pattern or the Alternative Healthy Eating Index is associated with lower hip fracture risk among Singapore Chinese. J Nutr. 2014;144(4):511-8. PMID: 24572035. http://www.ncbi.nlm.nih.gov/pubmed/24572035.

210. Feart C, Lorrain S, Ginder Coupez V, Samieri C, Letenneur L, Paineau D, et al. Adherence to a Mediterranean diet and risk of fractures in French older persons. Osteoporos Int. 2013;24(12):3031-41. PMID: 23783645. http://www.ncbi.nlm.nih.gov/pubmed/23783645.

211. Langsetmo L, Hanley DA, Prior JC, Barr SI, Anastassiades T, Towheed T, et al. Dietary patterns and incident low-trauma fractures in postmenopausal women and men aged ≥ 50 y: a population-based cohort study. Am J Clin Nutr. 2011;93(1):192-9. PMID: 21068350. http://www.ncbi.nlm.nih.gov/pubmed/21068350.

212. Langsetmo L, Poliquin S, Hanley DA, Prior JC, Barr S, Anastassiades T, et al. Dietary patterns in Canadian men and women ages 25 and older: relationship to demographics, body mass index, and bone mineral density. BMC Musculoskelet Disord. 2010;11:20. PMID: 20109205. http://www.ncbi.nlm.nih.gov/pubmed/20109205.

213. McTiernan A, Wactawski-Wende J, Wu L, Rodabough RJ, Watts NB, Tylavsky F, et al. Low-fat, increased fruit, vegetable, and grain dietary pattern, fractures, and bone mineral density: the Women's Health Initiative Dietary Modification Trial. Am J Clin Nutr. 2009;89(6):1864-76. PMID: 19403636. http://www.ncbi.nlm.nih.gov/pubmed/19403636.

214. Monjardino T, Lucas R, Ramos E, Barros H. Associations between a priori-defined dietary patterns and longitudinal changes in bone mineral density in adolescents. Public Health Nutr. 2014;17(1):195-205. PMID: 23149164. http://www.ncbi.nlm.nih.gov/pubmed/23149164.

215. Monma Y, Niu K, Iwasaki K, Tomita N, Nakaya N, Hozawa A, et al. Dietary patterns associated with fall-related fracture in elderly Japanese: a population based prospective study. BMC Geriatr. 2010;10:31. PMID: 20513246. http://www.ncbi.nlm.nih.gov/pubmed/20513246.

216. Noh HY, Song YJ, Lee JE, Joung H, Park MK, Li SJ, et al. Dietary patterns are associated with physical growth among school girls aged 9-11 years. Nutr Res Pract. 2011;5(6):569-77. PMID: 22259683. http://www.ncbi.nlm.nih.gov/pubmed/22259683.

217. Park SJ, Joo SE, Min H, Park JK, Kim Y, Kim SS, et al. Dietary patterns and osteoporosis risk in postmenopausal korean women. Osong Public Health Res Perspect. 2012;3(4):199-205. PMID: 24159515. http://www.ncbi.nlm.nih.gov/pubmed/24159515.

218. Samieri C, Ginder Coupez V, Lorrain S, Letenneur L, Alles B, Feart C, et al. Nutrient patterns and risk of fracture in older subjects: results from the Three-City Study. Osteoporos Int. 2013;24(4):1295-305. PMID: 22976577. http://www.ncbi.nlm.nih.gov/pubmed/22976577.

219. Wosje KS, Khoury PR, Claytor RP, Copeland KA, Hornung RW, Daniels SR, et al. Dietary patterns associated with fat and bone mass in young children. Am J Clin Nutr. 2010;92(2):294-303. PMID: 20519562. http://www.ncbi.nlm.nih.gov/pubmed/20519562.

220. George SM, Ballard-Barbash R, Manson JE, Reedy J, Shikany JM, Subar AF, et al. Comparing indices of diet quality with chronic disease mortality risk in postmenopausal women in the Women's Health Initiative Observational Study: evidence to inform national dietary guidance. Am J Epidemiol. 2014;180(6):616-25. PMID: 25035143. http://www.ncbi.nlm.nih.gov/pubmed/25035143.

Table D2.1. *AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults, 2013*

Critical Question 4a. Among overweight and obese adults, what is the efficacy/effectiveness of a comprehensive lifestyle intervention program (i.e., comprised of diet, physical activity, and behavior therapy) in facilitating weight loss or maintenance of lost weight?
Critical Question 4b. What characteristics of delivering comprehensive lifestyle interventions (e.g., frequency and duration of treatment, individual versus group sessions, onsite versus telephone/email contact) are associated with greater weight loss or weight loss maintenance?

Intervention/Question	Included Studies	Evidence Statement (Strength of Evidence)
3.4.1. Description of the Diet, Physical Activity, and Behavior Therapy Components in High-Intensity, Onsite Lifestyle Interventions	12 RCTs	ES1. The principal components of an effective high-intensity, on-site comprehensive-lifestyle intervention include: 1) prescription of a moderately-reduced calorie diet; 2) a program of increased physical activity; and 3) the use of behavioral strategies to facilitate adherence to diet and activity recommendations. *(High)*
3.4.2. Comprehensive Interventions Compared with Usual Care, Minimal Care, or No-Treatment Control	15 RCTs	ES 2a (Short-Term Weight Loss). In overweight and obese individuals in whom weight loss is indicated and who wish to lose weight, comprehensive lifestyle interventions consisting of diet, physical activity, and behavior therapy (all 3 components) produce average weight losses of up to 8 kg in 6 months of frequent (i.e., initially weekly), onsite treatment provided by a trained interventionist* in group or individual sessions. Such losses (which can approximate reductions of 5% to 10% of initial weight) are greater than those produced by usual care (i.e., characterized by the limited provision of advice or educational materials). Comparable 6-month weight losses have been observed in treatment comparison studies of comprehensive lifestyle interventions, which did not include a usual care group. *(High)* ES 2b (Intermediate-Term Weight Loss). Longer-term comprehensive lifestyle interventions, which additionally provide weekly to monthly on-site treatment for another 6 months, produce average weight losses of up to 8 kg at 1 year, losses which are greater than those resulting from usual care. Comparable 1-year weight losses have been observed in treatment comparison studies of comprehensive lifestyle interventions, which did not include a usual care group. *(Moderate)* ES 2c (Long-Term Weight Loss). Comprehensive lifestyle interventions which, after the first year, continue to provide bimonthly or more frequent intervention contacts, are associated with gradual weight regain of 1 to 2 kg/year (on average), from the weight loss achieved at 6 to 12 months. Long-term (>1 year) weight losses, however, remain larger than those associated with usual care. Comparable findings have been observed in treatment comparison studies of comprehensive lifestyle interventions, which did not include a usual care group. *(High)*
3.4.3. Efficacy/Effectiveness of Electronically Delivered, Comprehensive Interventions in Achieving Weight Loss Evidence Statement	13 RCTs	ES 3. Electronically delivered, comprehensive weight loss interventions developed in academic settings, which include frequent self-monitoring of weight, food intake, and physical activity—as well as personalized feedback from a trained interventionist*—can produce weight loss of up to 5 kg at 6 to 12 months, a loss which is greater than that resulting from no or minimal intervention (i.e., primarily knowledge based) offered on the internet or in print. *(Moderate)*
3.4.4. Efficacy/Effectiveness of Comprehensive, Telephone-Delivered Lifestyle Interventions in Achieving Weight Loss	3 RCTs	ES 4. In comprehensive lifestyle interventions that are delivered by telephone or face-to-face counseling, and which also include the use of either commercially-prepared prepackaged meals or an interactive web based program, the telephone delivered and face-to-face delivered interventions produced similar mean net weight losses of approximately 5 kg at 6 months and 24 months, compared with a usual care control group.

		(Low)
3.4.5. Efficacy/Effectiveness of Comprehensive Weight Loss Programs in Patients Within a Primary Care Practice Setting Compared With Usual Care	4 RCTs	ES 5. In studies to date, low to moderate-intensity lifestyle interventions for weight loss provided to overweight or obese adults by primary care practices alone, have not been shown to be effective. *(Low)*
3.4.6. Efficacy/Effectiveness of Commercial-Based, Comprehensive Lifestyle Interventions in Achieving Weight Loss	4 RCTs	ES 6. Commercial-based, comprehensive weight loss interventions that are delivered in person have been shown to induce an average weight loss of 4.8 kg to 6.6 kg at 6 months in 2 trials when conventional foods are consumed and 6.6 kg to 10.1 kg at 12 months in 2 trials with provision of prepared food, losses that are greater than those produced by minimal-treatment control interventions. *(Low)*
3.4.7. Efficacy/Effectiveness of Very Low-Calorie Diets, as Used as Part of a Comprehensive Lifestyle Intervention, in Achieving Weight Loss	4 RCTs	ES 7a. Comprehensive, high intensity on-site lifestyle interventions that include a medically supervised very low-calorie diet (often defined as <800 kcal/day), as provided by complete meal replacement products, produce total weight loss of approximately 14.2 kg to 21 kg over 11 to 14 weeks, which is larger than that produced by no intervention or a usual care control group (i.e., advice and education only). *(High)*
		ES 7b. Following the cessation of a high intensity lifestyle intervention with a medically supervised very-low calorie diet of 11 to 14 weeks, weight regain of 3.1 kg to 3.7 kg has been observed during the ensuing 21 to 38 weeks of non-intervention follow-up. *(High)*
		ES 7c. The prescription of various types (resistance or aerobic training) and doses of moderate intensity exercise training (e.g., brisk walking 135 to 250 minutes/week), delivered in conjunction with weight loss maintenance therapy does not reduce the amount of weight regained after the cessation of the very-low calorie diet, as compared with weight loss maintenance therapy alone. *(Low)*
3.4.8. Efficacy/Effectiveness of Comprehensive Lifestyle Interventions in Maintaining Lost Weight	14 RCTs	ES 8a. After initial weight loss, some weight regain can be expected, on average, with greater regain observed over longer periods of time. Continued provision of a comprehensive weight loss maintenance program (onsite or by telephone), for periods of up to 2.5 years following initial weight loss, reduces weight regain, as compared to the provision of minimal intervention (e.g., usual care). The optimal duration of weight loss maintenance programs has not been determined. *(Moderate)*
		ES 8b. 35% to 60% of overweight/obese adults who participate in a high intensity long-term comprehensive lifestyle intervention maintain a loss of ≥5% of initial body weight at ≥2 year's follow-up (post-randomization). *(Moderate)*
3.4.9. Characteristics of Lifestyle Intervention Delivery That May Affect Weight Loss: Intervention	10 RCTs	ES 9a (Moderate-Intensity Interventions). Moderate intensity, on-site comprehensive lifestyle interventions, which provide an average of 1 to 2 treatment sessions per month typically produce mean weight losses of 2 kg to 4 kg in 6 to 12 months, losses which generally are greater than those produced by usual care (i.e., minimal intervention control group). *(High)*
		ES 9b (Low-intensity Interventions). Low intensity, on-site comprehensive lifestyle interventions, which provide fewer than monthly treatment sessions do not consistently produce weight loss when compared to usual care. *(Moderate)*
		ES 9c (Effect of intervention intensity). When weight loss with each intervention intensity (i.e., low, moderate, and high) is compared to usual care, high-intensity lifestyle interventions (≥14 sessions in 6 months) typically produce greater net-of-control weight losses than low-to-moderate intensity interventions. *(Moderate)*

3.4.10. Characteristics of Lifestyle Intervention Delivery That May Affect Weight Loss or Weight	15 RCTs	ES 10. There do not appear to be substantial differences in the size of the weight losses produced by individual- and group-based sessions in high-intensity, comprehensive lifestyle intervention delivered on site by a trained interventionist*. *(Low)*
3.4.11. Characteristics of Lifestyle Intervention Delivery That May Affect Weight Loss or Weight Loss Maintenance: Onsite Versus Electronically Delivered Interventions		ES 11. Weight losses observed in comprehensive lifestyle interventions, which are delivered onsite by a trained interventionist* in initially weekly and then biweekly group or individual sessions, are generally greater than weight losses observed in comprehensive interventions that are delivered by Internet or email and which include feedback from a trained interventionist. *(Low)*

Part D. Chapter 3: Individual Diet and Physical Activity Behavior Change

INTRODUCTION

Individual behavior change lies at the inner core of the social-ecological model that forms the basis of the 2015 Dietary Guidelines for American Advisory Committee (DGAC) conceptual model (see ***Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence***). For this reason, it is crucial to identify the behavioral strategies that individuals living in the United States can follow to improve their healthy lifestyle behaviors as well as the key contextual factors that facilitate the ability of individuals to consume healthy diets.

In the past, American families seldom consumed food prepared outside their homes and, for the most part, consumed their meals as a family unit. However, these behaviors have changed dramatically in recent years. Today, 33 percent of calories are consumed outside the home and it is becoming more common for individuals to eat alone and to bring meals prepared outside into their homes (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). Eating away from home is associated with increased caloric intake and poorer dietary quality compared to eating at home.[1] As recognized by the 2010 DGAC these major changes in eating behaviors can be expected to have a negative impact on the quality of the diets consumed and the risk of obesity among the U.S. population.[2]

Other individual lifestyle behaviors related to dietary intakes and obesity risk also have changed in recent decades. The U.S. population has become increasingly sedentary,[3] with daily hours of screen time exposure becoming a serious public health concern due to its potential negative influence on dietary and weight outcomes. For example, it has been hypothesized that TV viewing time has a negative influence on dietary habits of individuals because of unhealthy snacking while watching TV and through exposure to advertisements of unhealthy food products.[4] In turn, excess caloric intake coupled with sedentary time directly resulting from excessive TV may increase the risk of obesity. Suboptimal sleep patterns associated with today's busy lives also have been identified as a potential risk factor for poor dietary behaviors and body weight outcomes.[5]

In response to these trends, interest has grown in the potential of behavioral strategies that individuals can use to improve their dietary behaviors. Specifically, self-monitoring of diet, physical activity, and body weight has been identified as a potential key component of successful healthy lifestyle interventions.[6] Diet self-monitoring may, in turn, be facilitated by the availability and use of menus displaying calorie labels and the Nutrition Facts label on packaged foods.

Recognizing the importance of these dietary and lifestyle behaviors to the health and well-being of the U.S. population, the DGAC reviewed recent evidence to address questions on the relationship between eating out, family shared meals, sedentary behavior, and diet and weight outcomes. The DGAC also sought to examine associations between sleep patterns, dietary intakes, and obesity risk. However, after conducting preliminary literature searches, the Committee determined sleep patterns was an emerging area with an insufficient body of evidence and did not include specific questions on this topic.

The DGAC also focused on identifying evidence that could provide individuals with tools to improve their dietary choices and body weight status. Specifically, the Committee reviewed recent evidence on the impact of diet and weight self-monitoring, and on use of food and menu labels on dietary intake and weight outcomes. The DGAC was interested in reviewing the evidence on the use of mobile health (m-health) technologies to improve dietary and weight outcomes, and after a preliminary review was conducted, determined that this, too, was an emerging area and that a full evidence review was premature. However, key m-health studies focused on self-monitoring were identified, and thus were reviewed as part of the body of evidence on self-monitoring. This chapter addresses sedentary behaviors, but not physical activity behaviors in general because these are addressed in ***Part D. Chapter 7: Physical Activity***.

Consistent with the DGAC conceptual model presented in ***Part B. Chapter 1: Introduction***, this chapter also addresses major contextual factors that influence the ability of individuals to implement healthy dietary and other lifestyles, including the prevention of sedentary behaviors. The Committee focused on the association between diet, body weight, and chronic disease outcomes and two contextual factors that are highly relevant in the United States—household food insecurity and acculturation.

Household food insecurity is defined as "access to enough food for an active, healthy life. It includes at a minimum (a) the ready availability of nutritionally adequate and safe foods, and (b) an assured ability to acquire acceptable foods in socially acceptable ways (e.g., without resorting to emergency food supplies, scavenging, stealing, or other coping strategies)".[7] Thus, household food insecurity is a condition that exists whenever the availability of nutritionally adequate and safe foods, or the ability to acquire acceptable foods in socially acceptable ways, is limited or uncertain.[7] In 2013, 49.1 million people in the United States lived in food insecure households, and of these, 8.6 million are children.[1] Household food insecurity is suggested to be an independent risk factor for poor physical and mental health outcomes across the lifespan.[8, 9]

The second contextual factor the DGAC addressed—acculturation—reflects that the United States continues to be a nation of immigrants.[10, 11] Acculturation has been defined both as the "process by which immigrants adopt the attitudes, values, customs, beliefs, and behaviors of a new culture",[12] and as the "gradual exchange between immigrants' original attitudes and behavior and those of the host culture".[13] Acculturation is relevant for individual dietary behaviors because evidence suggests that the healthy lifestyles with which immigrants arrive deteriorate as they integrate or assimilate into mainstream American culture.[14] Moreover, evidence suggests that to be effective in helping immigrants retain their healthy lifestyles, nutrition education programs, including those that are a part of food assistance programs, must be tailored to their different levels of acculturation.[14]

Given the strong relevance of household food insecurity and acculturation as contextual factors influencing healthy lifestyles, the DGAC examined associations between them and diet, obesity risk, and whenever possible, corresponding chronic disease risk factors.

LIST OF QUESTIONS

Eating Out

1. What is the relationship between eating out and/or take away meals and body weight in children and adults?

Family Shared Meals

2. What is the relationship between frequency and regularity of family shared meals and measures of dietary intake in U.S. population groups?
3. What is the relationship between frequency and regularity of family shared meals and measures of body weight and obesity in U.S. population groups?

Sedentary Behavior, Including Screen Time

4. What is the relationship between sedentary behavior and measures of dietary intake and body weight in adults?
5. How effective are behavioral interventions in youth that focus on reducing recreational sedentary screen time and improving physical activity and/or diet?

Self-Monitoring

6. What is the relationship between use of diet and body weight self-monitoring strategies and body weight outcomes in adults and youth?

Food and Menu Labeling

7. What is the relationship between knowledge and use of food and menu labels and measures of dietary intake in U.S. population groups?

Household Food Insecurity (HFI)

8. What is the relationship between household food insecurity (HFI) and measures of dietary intake and body weight?

Acculturation

9. What is the relationship between acculturation and measures of dietary intake?
10. What is the relationship between acculturation and body weight?
11. What is the relationship between acculturation and risk of cardiovascular disease (CVD)?
12. What is the relationship between acculturation and risk of type 2 diabetes?

METHODOLOGY

All of the questions covered in this chapter— eating out, family shared meals, sedentary behavior, self-monitoring, food and menu labeling, household food insecurity, and acculturation—were answered using Nutrition Evidence Library (NEL) systematic reviews. A description of the NEL process is provided in ***Part C: Methodology***. All reviews were conducted in accordance with NEL methodology, and the DGAC made all substantive decisions required throughout the process to ensure that the most complete and relevant body of evidence was identified and evaluated to answer each question. All steps in the process were documented to ensure transparency and reproducibility. Specific information about individual systematic reviews can be found at www.NEL.gov, including the search strategy, inclusion and exclusion criteria, a complete list of included and excluded articles, and detailed documentation describing the included studies and the body of evidence. A link to this website is provided following each evidence review.

EATING OUT

The majority of Americans consume meals outside of the home one or more times per week (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). The 2010 DGAC concluded that "strong and consistent evidence indicates that children and adults who eat fast food are at increased risk of weight gain, overweight, and obesity".[2] With this relationship as a foundation, the 2015 DGAC updated and expanded the review of the "eating out" topic. Specifically, the "fast food" category was broadened to capture other types of eating out venues (e.g., quick serve, casual, formal restaurants, and grocery store take-out).

Terminology used to define the exposure was modified from "eating out," to the broader term "eating out and/or take away meals" to reflect the inclusion of meals eaten out at a broader array of restaurant venues as well as takeout or ready-to-eat foods or meals purchased and consumed either away from or in the home. The population of interest remained healthy individuals ages 2 years and older.

Question 1: What is the relationship between eating out and/or take away meals and body weight in children and adults?

Source of evidence: Update to 2010 DGAC's NEL systematic review

Conclusion

Among adults, moderate evidence from prospective cohort studies in populations ages 40 years or younger at baseline indicates higher frequency of fast food consumption is associated with higher body weight, body mass index (BMI), and risk for obesity. **DGAC Grade: Moderate**

Among children, limited evidence from prospective cohort studies in populations ages 8 to 16 years at baseline suggests that higher frequency of fast food consumption is associated with increased adiposity, BMI z-score, or risk of obesity during childhood, adolescence, and during the transition from adolescence into adulthood. **DGAC Grade: Limited**

Insufficient evidence is available to assess the relationship between frequency of other types of restaurant and takeout meals and body weight outcomes in children and adults. **DGAC Grade: Grade Not assignable**

Implications

Given that one-third of calories are consumed outside of the home (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends)***, individuals should limit the frequency of eating at fast-food establishments. When eating out, one should choose healthy foods and beverages within their calorie needs to avoid increases in body weight.

Review of the Evidence

Fifteen prospective studies examined the relationship between eating out and/or take away meals and measures of body weight in adults and children.[15-29] Eleven studies in the United States [16-18, 20-23, 25-28] and four international studies (one each from Canada, the United Kingdom, Australia, and Spain)[15, 19, 24, 29] were reviewed. Men and women and boys and girls were well represented and the majority of studies within the United States included diverse populations.

In children, seven prospective cohort studies[19, 21, 22, 24, 27-29] examined the relationship between frequency of fast-food meals, or consumption of other types of meals and anthropometric outcomes and, overall, found mixed results. Six studies examined fast-food meals[19, 21, 22, 24, 28, 29]: three studies[19, 28, 29] indicated increased fast food intake, particularly more than twice per week, was associated with increased risk of obesity, BMI/BMI z-score or body fat, two[22, 24] found no association, and one[21] found no association in boys and a negative association in girls. Two studies looked at a variety of non-fast-food meals away from home, using varying definitions of food establishments and meal types and reported mixed findings for a relationship with weight-related outcomes.[27, 28]

In adolescents transitioning to adulthood, one study found high baseline frequency of fast food intake was associated with increased BMI z-scores at 5-year follow-up.[25] In adults, evidence consistently demonstrated a relationship between higher frequency of fast-food meal consumption and body weight outcomes. Five prospective cohort studies (three cohorts) reported a higher frequency of intake of meals from fast food locations, or intake exceeding once per week, was associated with higher weight gain, BMI, and risk of obesity.[17, 18, 20, 23, 26] A "moderate" grade was assigned (as opposed to the "strong" grade assigned by the 2010 DGAC) because the evidence based was small (five studies focused on fast food, three from the same cohort), all of which were prospective cohort studies; few studies controlled for energy intake and no study reported actual food consumed; and the method of measurement of "eating out" varied among studies. Evidence related to the association between frequency of meals from other types of restaurants and intake of all takeout meals and weight is limited, but indicates traditional restaurant meal frequency may not be associated with weight outcomes.[17, 18] Two studies[15, 16] examined total meals away from home or meal types eaten away from home, which came from both fast food and restaurant locations, and reported frequency was associated with increased body weight outcomes for most meal types. Two studies from the same cohort found no significant relationship between frequency of meals from restaurants (non-fast-food establishments), and weight-related outcomes.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3371

FAMILY SHARED MEALS

Data from cross-sectional studies suggest that when families share meals, they achieve better diet quality and improved nutrient intake, and to some extent, are better able to maintain appropriate body weight.[30-36] The definition of family shared meals in the literature varies, with some defining it as the number of a specific meal eaten together (e.g., dinner), or any meal, prepared at home or outside of home, that is shared among individuals living in the same household.[37] Family mealtime may act as a protective factor for many nutritional health-related problems. For example, they provide an opportunity for parents to model good eating behaviors and create a positive atmosphere by providing time for social interaction and thus a sense of social support for all members.[38, 39] Shared meals may be important in every stage of the lifecycle to support healthy growth, development, and weight, though the evidence for adults is mixed. The importance of the family in supporting positive behaviors is clearly part of the life course approach embodied in the DGAC's conceptual model (see ***Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence***). As a result, the Committee decided to explore the relationship between family shared meals and dietary intake as well as weight outcomes from high-quality epidemiological studies to determine if there is a cause and effect association.

Question 2: What is the relationship between frequency/regularity of family shared meals and measures of dietary intake in U.S. population groups?

Source of evidence: NEL systematic review

Conclusion

Insufficient evidence on the association between frequency of family shared meals and measures of dietary intake is available to draw a conclusion. **DGAC Grade: Grade not assignable**

Implications

The DGAC determined that a grade was not assignable due to the insufficient evidence for this question. Therefore, no implications were developed.

Review of the Evidence

Two studies in the United States with the duration of 5 to 10 years from one prospective cohort examined the relationship between frequency/regularity of family meals and measures of dietary intake in U.S. population groups.[40, 41] The studies included adolescents transitioning from early to middle adolescence (middle school to high school)[40] and adolescents transitioning to early adulthood.[41] These studies found more frequent consumption of family meals was associated with improved dietary intake, specifically an increase in fruits and/or vegetables, and calcium-rich or milk-based foods.[40, 41] Given that the evidence is limited to these two studies using data from the same cohort at two time points, the Committee could not assign a grade.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250455

Question 3: What is the relationship between frequency/regularity of family shared meals and measures of body weight in U.S. population groups?

Source of evidence: NEL systematic review

Conclusion

Limited evidence from prospective studies shows inconsistent relationships between the number of family shared meals and body weight of children and adolescents. **DGAC Grade: Limited**

Implications

The very limited evidence available on the relationship between family shared meals and measures of body weight precludes developing implications for this question. Shared meals may be important in every stage of the lifecycle to support healthy growth, development, and weight; however, more studies are warranted to determine if there is a direct effect. In the absence of such studies, meal times may still be an optimal time for parents to provide role modeling behaviors in terms of what foods to eat and, for the elderly encouragement to eat given the social support of other individuals.

Review of the Evidence

Six studies, which included one randomized control trial (RCT)[42] and five prospective cohort studies (4 cohorts)[43-47] examined the relationship between frequency/regularity of family meals and measures of body weight in U.S. populations. The study duration for the RCT was 6 months[42] and the prospective cohort studies[43-47] ranged in duration from 1 to 5 years. The study population was children and adolescents ages 4 to 15 years.

Three out of four prospective cohort studies found no significant association between the frequency of family shared meals, BMI, or overweight status. Evidence from one prospective study (two articles) showed that an increase in the frequency of family shared meals lowered the likelihood of becoming overweight or the persistence of overweight. One study found that among overweight children, eating more family breakfast and dinner meals was associated with lower likelihood of becoming overweight or remaining overweight over a 4-year period. Another article reported children who typically ate more breakfast meals with their families had a lower rate of increase in BMI over 5 years. The number of dinner meals eaten with the family was not associated with a change in BMI.

One RCT included an intervention that simultaneously focused on four household routines, including family shared meals.[42] Although a reduction in body weight occurred, family meal frequency did not change.[42]

This body of evidence had several limitations, including that studies did not use a standard definition for family shared meals, two studies assessed only family dinners, two studies assessed breakfast and dinner meals, and two studies assessed all meals. No study assessed the quality or source of meals consumed.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250460

SEDENTARY BEHAVIOR, INCLUDING SCREEN TIME

The *Physical Activity Guidelines for Americans* recommend that adults engage in at least 150 minutes (2.5 hours) of moderate- to vigorous-intensity physical activity each week and two days a week of strength training.[48] Youth ages 6 to 17 years should engage in 60 minutes or more of daily physical activity.[48] Unfortunately, the vast majority of Americans do not get the physical activity they need; only 20 percent of adults meet both the aerobic and strength training recommendations and less than 20 percent of adolescents meet the youth guideline. [49, 50] In addition, one-third of adults engage in no leisure-time physical activity.[51] Regular physical activity is associated with myriad health benefits, including reduced risk of chronic disease, and physical, mental, and cognitive benefits, irrespective of body weight.[48] Physical inactivity is associated with increased risk of overweight and obesity, CVD, type 2 diabetes, breast and colon cancer, and overall all-cause mortality.[52]

Sedentary behavior, which refers to any waking activity predominantly done while in a sitting or reclining posture, is gaining considerable public health interest as a chronic disease risk factor and therefore a potential area for interventions to target, with reducing screen time often a focus. The American Academy of Pediatrics (AAP) recommends no more than 2 hours a day of screen time (including television and other types of media) for children ages 2 years and older and none for children younger than age 2 years.[53] However, children ages 8 to 18 years spend an average of 7 hours on screen time each day.[54] The U.S. Report Card on Physical Activity for Youth gave the sedentary behavior indicator a grade of "D" for youth meeting the AAP's screen time recommendation.[55] Rates of screen time are similar among males and females, yet disproportionately higher for African American youth compared to Caucasian youth (63.3 percent not meeting AAP recommendation vs. 44.6 percent).[56] For this topic, two questions were addressed by the DGAC, the first with a NEL systematic review focused on the transition from childhood to adulthood and sedentary behavior in adults. The second question used the 2014 Community Preventive Services Task Force Obesity Prevention and Control (Community Guide) systematic review to examine the effectiveness of interventions among youth to reduce sedentary screen time and increase physical activity.

Question 4: What is the relationship between sedentary behavior and dietary intake and body weight in adults?

Source of evidence: NEL systematic review

Conclusion

Moderate and consistent evidence from prospective studies that followed cohorts of youth into adulthood supports that adults have a higher body weight and incidence of overweight and obesity when the amount of TV viewing is higher in childhood and adolescence. **DGAC Grade: Moderate**

Moderate evidence from prospective studies suggests no association between sedentary behavior in adulthood and change in body weight, body composition, or incidence of overweight or obesity in adulthood. **DGAC Grade: Moderate**

Insufficient evidence exists to address the association between sedentary behavior and dietary intake in adults. **DGAC Grade: Grade Not Assignable**

Implications

Sedentary behavior, including TV watching and screen time, should be limited during childhood to lower the likelihood of excess body weight or overweight and obesity in adulthood. Federal, state, and local policies and programs to support school and community-based programs to identify and reduce sedentary behavior among children and adolescents are needed to help them achieve and maintain healthy weight status as they transition into adulthood. Although an apparent lack of association exists between sedentary behavior and change in body weight status in adulthood, adults are encouraged to adopt and sustain levels of physical activity consistent with the *Physical Activity Guidelines for Americans* to promote health and to achieve and sustain a healthy weight status.

Review of the Evidence

This evidence review included 23 studies from 18 prospective cohorts that examined the relationship between sedentary behavior and body weight status in adults.[57-79] Study locations included six studies from Australia,[59, 60, 65, 74, 75, 77] six studies from the United Kingdom,[61, 69, 70, 73, 76, 78] seven studies from the United States,[57, 58, 62, 66, 67, 71, 79] two studies from New Zealand,[63, 64] and one study each from Canada[72] and Spain.[68] The mean age of participants ranged from 23 years to 60 years. Longitudinal studies followed participants from childhood (5 to 16 years) to adulthood (21 to 45 years). Three studies (two cohorts)[57, 59, 75] had an all-female sample and the remainder of the studies included both males and females.

Increasing levels of TV viewing during childhood and adolescence predicted higher BMI[64, 65, 69, 76] and increased incidence of overweight and obesity in adulthood.[58, 64, 65, 76] The lack of association between adult sedentary behavior (TV viewing, commute time or composite measures of sedentary behavior) and body weight change or body weight status are mostly consistent, despite methodological differences in measurement of sedentary behavior. Among two studies that assessed the relationship between sedentary behavior in adulthood and dietary intake, one study found an association between TV viewing and lower compliance with recommended dietary guidance.[66] The other study found that more TV viewing was associated with greater intake of calories from fat, but not total calories or calories from sweets.[71]

Methodological approaches differed with regard to population and cohort size, types of sedentary behavior considered, and timeframes studied. Only one study directly measured sedentary behavior[61] and few studies adjusted analysis for energy intake and other potential mediators, such as dietary intake. The majority of studies were conducted in Caucasian populations; therefore diverse ethnic and racial groups were underrepresented.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3343

Question 5: How effective are behavioral interventions in youth that focus on reducing recreational sedentary screen time and improving physical activity and/or diet?

Source of evidence: *Community Preventive Services Task Force Obesity Prevention and Control: Behavioral Interventions that Aim to Reduce Recreational Sedentary Screen Time (Community Guide)*[80] Available at: http://www.thecommunityguide.org/obesity/RRbehavioral.html

Conclusion

The DGAC concurs with the Community Guide,[80] which found strong evidence that behavioral interventions are effective in reducing recreational sedentary screen time among children ages 13 years and younger. Limited evidence was available to assess the effectiveness of these interventions among adults and no evidence was available for adolescents ages 14 years and older.

DGAC Grade: Strong

Implications

The Community Guide identified effective behavioral interventions to reduce recreational screen time and recommended that they be implemented in a variety of settings. The DGAC concurs with this recommendation because of the potential for these interventions to have beneficial effects on children's diet and weight status. Multifaceted interventions to reduce recreational sedentary screen time may include home, school, neighborhood, and pediatric primary care settings, and emphasize parental, family, and peer-based social support, coaching or counseling sessions, and electronic tracking and monitoring of the use of screen-based technologies.

Review of the Evidence

The Community Guide review classified behavioral screen time interventions as: 1) screen-time-only interventions that focus only on reducing recreational sedentary screen time, and 2) screen-time-plus interventions, which focus on reducing recreational sedentary screen time and increasing physical activity and/or improving diet. These interventions are used to teach behavioral self-management skills through one or more of the following components: classroom-based education, tracking and monitoring, coaching or counseling sessions, and family-based or peer social support. The Community Guide review focused on both high- and low-intensity interventions to reduce sedentary behavior in youth. High-intensity interventions included the use of an electronic monitoring device to limit screen time or at least three personal or computer-tailored interactions. Low-intensity interventions included two or fewer personal or computer-tailored interactions. This review included 49 studies with 61 arms. Studies were included that had an intervention component with one or more outcomes of interest. Study duration was 1.5 months to 2 years.

The study populations were mostly children younger than age 13 years and collectively were racially and ethnically diverse. All studies were conducted in the United States within a variety of settings, including schools (20 studies), homes (8 studies), communities (6 studies), primary care clinics (4 studies), research institutes (5 studies), and in multiple settings (4 studies). Settings were a mix of urban and suburban areas.

Evidence indicated that behavioral screen time interventions are effective in reducing recreational sedentary screen time (47 study arms), improving physical activity (42 study arms), improving diet (37 study arms), and improving or maintaining weight status (38 study arms). Studies were found to be effective among children ages 13 years and younger. The evidence showed that both screen-time-only and screen-time-plus interventions are both effective at reducing recreational sedentary screen time. However, screen-time-only interventions showed greater reductions in TV viewing and composite screen time compared to screen-time-plus interventions. All studies demonstrated effectiveness among both males and females. Forty-five studies that reported racial distribution showed intervention effectiveness in all groups: white (20 studies), black (14 studies), Hispanic (11 studies), Asian/Pacific Islander (10 studies), American Indian or Alaska Native (3 studies), and other (7 studies).

For additional details on this body of evidence, visit:
http://www.thecommunityguide.org/obesity/RRbehavioral.html

SELF-MONITORING

In the context of comprehensive behavioral lifestyle interventions for weight management, self-monitoring refers to the process by which an individual observes and records specific information reflecting his or her dietary intake, physical activity, and/or body weight. As a component of behavioral weight-management programs, self-monitoring is typically coupled with goal setting and performance feedback. Goal setting involves specifying a target or recommended level for dietary intake, physical activity, and/or body weight. Self-monitoring provides information that allows the individual to judge whether targets have been met, and if not, to use the feedback from self-monitoring to adjust future actions so as to meet the target. A high frequency of self-monitoring is commonly associated with greater adherence to other weight management strategies and with greater success in lifestyle programs for weight management.[81]

The goal of this systematic review was to determine whether self-monitoring of diet and/or weight is associated with body weight outcomes. This review included studies examining the effect of self-weighing or self-monitoring of diet, such as counting calories and/or monitoring foods consumed. Although paper diaries are the traditional method for self-monitoring new technological approaches are emerging, such as the use of websites, smart phone “apps,” and interactive voice response phone calls. Because self-monitoring is often a component of weight loss and weight maintenances interventions, it is important to understand its effect on body weight outcomes.

Question 6: What is the relationship between use of diet and weight self-monitoring strategies and body weight outcomes in adults and youth?

Source of evidence: NEL systematic review

Conclusion

Moderate evidence, primarily in overweight adult women living in the United States, indicates that self-monitoring of diet, weight, or both, in the context of a behavioral weight management intervention, incorporating goal setting and performance feedback, improves weight-loss outcomes. **DGAC Grade: Moderate**

Limited but consistent evidence suggests that higher frequency or greater adherence to self-monitoring of diet, weight, or both, in the context of a behavioral weight management program, is associated with better weight-loss outcomes. **DGAC Grade: Limited**

Implications

Self-monitoring coupled with goal setting and performance feedback can be used to enhance outcomes in weight management programs and should be incorporated into these programs for weight management.

Review of the Evidence

Twenty studies (4 RCTs,[82-85] 15 prospective cohort studies,[86-100] and 1 retrospective cohort study[101]) examined the relationship between diet and weight self-monitoring strategies and body weight outcomes in adults and youth. The study durations ranged from 3 months to 3.25 years. The study samples predominantly included women. Five studies were exclusively in women, one study was in pregnant women,[88] and one study was in children.[83] Sixteen studies were conducted in the United States[84-87, 89-100] and four were international (one each from the United Kingdom, Australia, Netherlands, and Japan).[82, 83, 88, 101]

Three RCTs showed that weight management interventions, delivered through mail or email which included self-monitoring of diet, weight, or both, coupled with behavioral change strategies, such as goal setting, personalized feedback, shaping, stimulus control, and problem solving, resulted in significantly greater weight losses than did interventions that did not emphasize self-monitoring.[82, 84, 85] One weight loss maintenance study in children found no effect for self-monitoring through Short Message Service on BMI.[83]

Sixteen cohort studies in adults found higher frequency or greater adherence to diet and weight self-monitoring was associated with favorable body weight outcomes.[86-101] One study with overweight pregnant women provided a four-session behavior change program with a gestational weight gain chart and a recommendation for regular self-weighing.[88] The women in the intervention arm lost more weight 6 weeks after delivery compared to a control group that received one brief education session. Four studies assessed different methods of self-monitoring, including paper diaries, Internet-based or mobile applications, and found that no specific method was superior to others.[87, 93, 94, 98]

The limitations of the evidence were that study participants were predominately overweight or obese, educated, Caucasian, females between the ages of 30 to 60 years, thus limiting generalizability to broader population groups.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3374

FOOD AND MENU LABELING

Food and menu labels can provide information that improves an individual's food selection and potentially improves body weight outcomes. Research focusing upon the impact of food labeling on body weight and other health outcomes is beginning to emerge. The U.S. Food and Drug Administration (FDA) recently finalized regulations requiring calorie information to be listed on menus and menu boards in chain restaurants, similar retail establishments, and vending machines with 20 or more locations. Studying the effects of this regulation on dietary choices, weight and chronic disease outcomes will provide an opportunity to understand how policy works in real-world conditions.

Some studies, including existing reviews, have examined the impact of restaurant calorie labeling on free-living consumer food selection and have had mixed results. Few studies have actually measured calories consumed as a result of menu labeling. A recent systematic review including 17 studies with experimental or quasi-experimental designs evaluated whether menu-based nutrition information affects the selection and consumption of calories in restaurants and other foodservice establishments.[102] Five of these studies measured the association between the introduction of menu labeling and average calories purchased per transaction in fast-food restaurants before and after implementation of policies that required restaurants to add calorie values to menus. Data collection varied in terms of duration (2 weeks to 6 months) and time from menu changes (from 4 weeks to one year after menu calorie labeling took place). Only one of the five reported a statistically significant association between the introduction of menu labeling and the selection of fewer calories.

Overall, however, the review concluded that menu labeling of calories alone did not decrease calories selected or consumed but that the addition of contextual or interpretive information on menus, such as daily caloric recommendations or physical activity equivalents, assisted consumers to select and consume fewer calories. [102] Additionally, there appeared to be a difference in sex response such that women tended to use the information to select and consumer fewer calories than men.

The intent of this NEL systematic review was to focus on controlled trials that isolated the impact of menu labeling on food selection and consumption at the individual level. The Committee was also interested in the effects of menu labeling on body weight outcomes; however there was insufficient evidence from RCTs examining the association between food and menu labels and body weight to complete a systematic review with body weight as the outcome.

Question 7: What is the effect of use of food and menu labels on measures of food selection and dietary intake in U.S. population groups?

Source of evidence: NEL systematic review

Conclusion

Limited and inconsistent evidence exists to support an association between menu calorie labels and food selection or consumption. **DGAC Grade: Limited**

Implications

The impact of food and menu labeling on food selection and health outcomes is limited by the heterogeneous approaches and the modest number of high quality studies, particularly RCTs. Thus, no implication could be drawn from the RCTs although policy level studies suggest that menu labeling of calories alone will not decrease calories selected or consumed but that addition of contextual or interpretive information on menus, such as daily caloric recommendations or physical activity equivalents, can assist consumers to select and consume fewer calories.[102] The new menu labeling regulations recently finalized by the FDA will provide an opportunity for further food and nutrition policy research in real-world settings.

Review of the Evidence

Ten RCTs[103-112] were included in this body of evidence that compared menu calorie labeling on food selection. Three of the ten studies also measured calorie intake of a test meal.[107-109] Results were mixed regarding the influence of menu calorie labeling on food selection. Five studies found no effect of calorie information alone on food selection.[104, 105, 107, 108, 110] Three studies found calorie labeling led to selection of fewer calories.[103, 109, 112] Two studies showed mixed results. One[106] found an impact of calorie labeling with women, but not men, and another[111] found that parents ordered fewer calories for their children, but not for themselves when calorie information was included on a test menu.

Two studies found that providing calorie labels with either recommended daily caloric intake information[109] or physical activity equivalents[108] resulted in the consumption of fewer calories at a test meal. One study did not find an effect of calorie labeling on calorie consumption.[107] Two studies examining physical activity equivalents as a component of the calorie labeling found a decrease in the calorie content of selected food items.[104, 108] One study that examined the effect of calorie labeling and value pricing (structuring product prices such that the per unit cost decreases as portion size increases) also showed no association between calorie labeling and food selection or consumption.

This body of evidence has many limitations: two of the ten studies were conducted in actual restaurant settings, limiting the external validity of the findings; three studies measured food intake; some studies included pricing as a confounder, while others did not; and all studies were conducted in one session. The methodological complexities of laboratory studies limit generalizability to free living populations.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3379

HOUSEHOLD FOOD INSECURITY

Food insecurity is a leading nutrition-related public health issue that is associated with reduced food intake or hunger because the household lacks money and other resources for food. Food insecurity can compromise nutritional intake, potentially leading to increased risk of chronic diseases.[9] In addition, food insecurity may promote anxiety and psychological distress, further affecting the health and well-being of an individual or family.[113, 114] Food insecurity is typically measured by survey questionnaires, such as the U.S. Household Food Security Survey Module, an 18-item questionnaire that assesses characteristics at the household level and severity of food insecurity (e.g., moderate or severe) over the past 12 months. The standard method of scoring consists of households being considered food secure if respondents affirm less than 3 scale items, food insecure if 3 to 7 items are affirmed, and severely food insecure if 8 or more items are affirmed.[9] Surveys in the United States indicate that 14.3 percent or more of households experienced food insecurity at least once during 2013.[1] Rates of food insecurity are substantially higher than the national average for those households with incomes near or below the Federal poverty line (38.4 percent vs. 14.3 percent), those households with children and a single parent, and for African American- and Hispanic-headed households.[1] Rates of food insecurity are more common in rural areas and large cities compared to suburban and exurban areas surrounding cities.[1] Among food-insecure households, 62 percent are participating in one or more of the three largest Federal food and nutrition assistance programs (Supplemental Nutrition Assistance Program [SNAP], Special Supplementation Program for Women, Infants, and Children [WIC], and the National School Breakfast and Lunch Programs).[1] The causes of food insecurity are multifactorial and the types of nutrition-related problems resulting from food insecurity are diverse, differing across the life cycle. Among food insecure households, the cycle of having enough food followed by inadequate amounts has been associated with stress in pregnant women,[113] poor diet quality among adults,[115, 116] poor glycemic control among diabetics,[117] and high visceral body fat and body weight gain in some but not all cross-sectional studies of children and adults.[118-120] Each of these conditions has a well-documented impact in the development of chronic diseases.[121, 122] Thus, the 2015 DGAC chose to examine the relationship between food insecurity and diet quality as well as the causal nature of this public health issue on body weight with a systematic review of prospective cohorts.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3372

Question 8: What is the relationship between household food insecurity (HFI) and measures of diet quality and body weight?

Source of evidence: NEL systematic review

Conclusion

Limited and inconsistent evidence from studies conducted in adults and children ages 3 to 6 years suggests that a positive association may exist between persistent and/or progressing household food insecurity and higher body weight in older adults, pregnant women, and young children. No studies reported a relationship with lower body weight. **DGAC Grade: Limited**

Insufficient evidence was available from prospective studies to assess the relationship between household food insecurity and dietary intake. **DGAC Grade: Grade Not assignable**

Implications

Federal food assistance programs, which play an important role in providing relief to families in economic distress, should carefully document and monitor food insecurity and nutritional risk in program participants. Participants should receive tailored counseling to choose foods with their limited budgets that meet the *Dietary Guidelines for Americans* and to achieve or maintain a healthy body weight. Federal food assistance programs should also regularly assess, evaluate, and update the methods they use to help recipients select healthier foods, consistent with best practices.

Review of the Evidence

This systematic review included nine prospective cohort studies examining the relationship between household food insecurity and body weight status.[118, 123-130] In adults, four prospective cohort studies assessed the relationship between household food insecurity and measures of body weight, with one study focusing on elderly men and women[126] and three studies focusing only on women.[118, 128, 130] The study of older adults derived data from two large cohorts including the Health and Retirement Survey and the Asset and Health Dynamics among the Oldest Old.[126] The studies on women ranged in size from 303 to 1,707, with the data derived from relatively small cohort study populations, including the Bassett Mothers Health Project cohort study,[128] the Pregnancy, Infection, and Nutrition cohort,[118] and the Fragile Families and Child Wellbeing Study.[130] The study of older adults focused on a relatively homogenous population who were mostly Caucasian.[126] Of the studies of women, two assessed diverse populations,[118, 130] while one had a study population almost entirely composed of Caucasian women.[128]

In children, a total of five prospective cohort studies (three cohorts)[123-125, 127, 129] assessed the relationship between household food insecurity and measures of body weight, with one of the five studies assessing household food insufficiency, a similar measure considered more severe than the concept of food security, although not as severe as hunger.[124] Four of the studies were conducted on populations in the United States[123, 125, 127, 129] and one study in a Canadian population.[124] The studies ranged in size from 1,514 to 28,353 subjects. The data were derived from nationally representative cohorts, including three studies using data from the Early Child Longitudinal Study-Kindergarten Cohort,[123, 125, 129] one study using data from the Longitudinal Study of Child Development in Quebec,[124] and one study deriving data from a large cohort participating in the Massachusetts WIC Program.[127]

Based on this evidence, the impact of food insecurity on body weight is not clear. Among older adults, becoming food insecure during follow-up was positively associated with BMI in one large cohort but there was no association in a different cohort from the same study.[126] Among pregnant women, findings were inconsistent, with 1 of 2 studies suggesting no association between food insecurity and pregnancy weight gain outcomes.[128] One study found null findings among the marginally food secure, but greater weight gain (absolute and relative to the 2009 IOM Guidelines),[131] and severe pre-gravid obesity among food insecure women.[118] Among children, findings were inconsistent. Two studies found no association between food insecurity and body weight outcomes.[123, 129] Dubois et al. found that food insufficiency was associated greater likelihood of overweight and obesity in preschool-aged children.[124] One study found that persistent food insecurity without hunger was associated with child obesity but non-persistent food insecurity with hunger was not associated with obesity risk.[127] Jyoti et al. reported that there was an association between food insecurity and weight gain for girls but not boys.[125] However, the data provided some suggestion of an association between food insecurity and higher body weight among girls and those who are of low birth weight.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3372

ACCULTURATION

Immigrants continue to represent a significant proportion of the United States population and evidence indicates that immigrants adopt the dietary habits and disease patterns of host cultures.[14] Federal food assistance and nutrition education programs are aware of the need to tailor services and messaging according to the level of acculturation of immigrant communities. It is essential for this acculturation-sensitive tailoring to take into account the level of dietary acculturation and the socio-economic characteristics such as health literacy, language, and other cultural preferences of immigrant communities. Thus, understanding how dietary habits, body weight, and chronic disease outcomes are influenced by the process of acculturation is an important public health issue for the United States. However, because immigrants can take different paths during the process of acculturation, this construct has proven to be difficult to conceptualize and measure. The four paths of acculturation (assimilation, integration, segregation, and marginalization) refer to the degree in which immigrants retain their host culture and adopt the culture of their new country.[14] This explains, at least in part, why the evidence from prospective studies continues to be limited in nature, as shown in this chapter.

Question 9: What is the relationship between acculturation and measures of dietary intake?

Source of evidence: NEL systematic review

Conclusion

Limited evidence from cross-sectional studies suggests that in adults of Latino/Hispanic national origin, particularly among women and persons of Mexican origin, higher acculturation to the United States is associated with lower fruit and vegetable intake, as well as higher intake of fast food. Insufficient evidence is available for children, Asians and African Americans in general, and among populations of diverse Latino/Hispanic national origin to draw a conclusion regarding the association between measures of acculturation and dietary intake. **DGAC Grade: Limited**

Implications

Federal food assistance and nutrition education programs need to support immigrants in maintaining the healthy dietary habits they had when they arrived and in not acquiring unhealthy dietary patterns as they acculturate to mainstream America. This can be achieved by, among other things, effectively reaching out to immigrant families to facilitate their enrollment in programs such as SNAP and WIC and ensuring access to fresh vegetables and fruits. These community outreach programs are needed because in addition to their risk of adopting unhealthy dietary behaviors, immigrants may also have language limitations and/or a lack of understanding of the program enrollment procedures.

Review of the Evidence

This systematic review included 17 studies, 15 cross-sectional studies,[132-146] and two longitudinal studies[147, 148] that examined the relationship between multidimensional or multiple proxy measures of acculturation and dietary intake. Study populations included ten Latino/Hispanic populations[132-136, 138-140, 144, 145] (five in Mexican Americans) and [132, 133, 135, 136, 140] six Asian populations;[137, 141-143, 146, 147] one study included both Asian and Latino/Hispanic populations.[148] Two studies included children[135, 148] and three studies included only women.[134, 138, 140] Study locations included one national[140] and one U.S.-Mexican border state study,[136] ten studies from California,[132, 133, 135, 137-139, 143, 145, 146, 148] and one study each from Massachusetts, Hawaii,[147] New York,[141] and a Midwestern city.

In adults of Latino/Hispanic national origin, evidence from nine cross-sectional analyses suggests that higher acculturation to the United States is associated with lower adherence to recommended dietary patterns. Among adults of Latino/Hispanic national origin, primarily women and those of Mexican origin, higher acculturation is consistently associated with lower fruit and vegetable intake, as well as higher intake of fast food. In children and youth of Latino/Hispanic national origin, emerging evidence was identified from two cross-sectional studies suggesting a negative association between acculturation and dietary behaviors. In a study of children ages 3 to 5 years who were proxied by caregiver acculturation, acculturation was associated with higher intake of sweets. In a study among adolescents, acculturation was associated with higher intake of fast foods.

Among Asian populations, emerging evidence from five cross-sectional and two longitudinal studies suggests that higher acculturation is associated with lower adherence to recommended dietary patterns. In adults, six studies among Asian populations (mainly Korean, Chinese and Filipino) suggest higher acculturation is associated with higher fast food and alcohol consumption.[137, 141-143, 146, 147] One study suggests higher acculturation is associated with increased fast food consumption among Asian adolescents.[148]

Insufficient evidence is available among children, those of Latino/Hispanic national origin (other than Mexican-Americans), and among immigrant populations from Asia, Africa, Europe, and the Middle East regarding the association between measures of acculturation and dietary intake.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250436

Question 10: What is the relationship between acculturation and body weight?

Source of evidence: NEL systematic review

Conclusion

Limited evidence suggests a relationship between higher acculturation to the United States and increased body weight. This relationship varies by national origin and gender. Specifically, findings were mixed in both Asian and Latino/Hispanic populations. In Asians, the association was stronger in women than men and in Latino/Hispanic populations; associations were stronger in Mexican-born women. **DGAC Grade: Limited**

Implications

Federal food assistance and nutrition education programs need to support immigrants against the risk of becoming overweight or obese as they acculturate to mainstream America. This can be achieved by among other things, effectively reaching out to immigrant families to facilitate their enrollment in programs such as SNAP and WIC and ensuring access to low-energy and high-nutrient dense dietary patterns rich in vegetables and fruits and whole grain foods. These community outreach programs are needed because in addition to their risk of adopting unhealthy dietary behaviors, immigrants may also have language limitations and/or a lack of understanding of the program enrollment procedures.

Review of the Evidence

This systematic review includes 13 studies:[133, 137, 141, 143, 144, 146, 147, 149-154] 12 cross-sectional studies,[133, 137, 141, 143, 144, 146, 149-154] and one longitudinal study.[147] The populations included seven Asian,[137, 141, 143, 146, 147, 150, 151] five Latino/Hispanic (four Mexican-American and one Puerto Rican),[133, 144, 149, 152, 153] and included adults ranging in age from 35 to 75 years. Five studies were analyzed by gender.[141, 143, 146, 153, 154] Three of the studies included national samples,[149, 152, 154] five studies were from California,[133, 137, 143, 146, 153] and one study each was from Hawaii,[147] Louisiana,[151] Maryland,[150] Massachusetts,[144] New York.[141] Two studies included samples from the country of origin (Vietnam and Korea).[143, 151]

Among Asian populations, the majority of the data suggest a positive relationship between acculturation and increased body weight, but results are not consistent. Among Latinos/Hispanic populations, the association has been documented mostly among women of Mexican origin.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250437

Question 11: What is the relationship between acculturation and risk of cardiovascular disease (CVD)?

Source of evidence: NEL systematic review

Conclusion

No conclusion can be drawn regarding the relationship between acculturation to the United States and the risk of CVD. This is due to the small number of studies, wide variation in methodology used to assess acculturation, and limited representation of ethnic groups in the body of evidence. Very limited evidence from a small number of cross-sectional studies conducted in Latino/Hispanic populations suggest a positive relationship between language acculturation and elevation in LDL cholesterol and no relationship between acculturation and blood pressure.

Insufficient evidence is available for other race/ethnic populations and among children for these outcomes and other CVD outcomes. **DGAC Grade: Grade not assignable**

Implications

The DGAC determined that a grade was not assignable due to the insufficient evidence for this question. Therefore, no implications were developed.

Review of the Evidence

This systematic review includes six cross-sectional studies in adult men and women between the ages of 40 to 60 years.[144, 154-158] The study populations included five Latino/Hispanic[144, 155-158] and one multicultural population[154] and the data were predominately derived from large, multi-state or national data sets.

Three studies found a positive relationship between language acculturation and elevated blood lipid levels,[154, 156, 157] but results varied by acculturation indicator. Two studies assessed the association between acculturation and blood pressure in Latino/Hispanic populations and no association was found.[156, 157] Two studies assessed the relationship between acculturation and hypertension in Latino/Hispanic and a multicultural population and found no association.[144, 154] Two studies suggest a positive association between language acculturation and CVD risk factors,[155, 158] but results varied as a function of language acculturation indicator used. The studies used different methods to assess acculturation, including three studies that used multidimensional scales[144, 155, 157] and three studies that relied on the assessment of acculturation proxies.[154, 156, 158]

The preponderance of evidence was in predominately Mexican American populations, but other Hispanic/Latino national origin groups were represented.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250438

Question 12: What is the relationship between acculturation and risk of type 2 diabetes?

Source of evidence: NEL systematic review

Conclusion

Conclusions regarding the relationship between acculturation and type 2 diabetes cannot be drawn due to limited evidence from a very small number of cross-sectional studies and study populations, limitations in acculturation assessment methodology that did not take into account potential confounders and effect modifiers, and lack of standardized assessment of outcomes. **DGAC Grade: Grade not assignable**

Implications

The DGAC determined that a grade was not assignable due to the insufficient evidence for this question. Therefore, no implications were developed.

Review of the Evidence

This systematic review included four cross-sectional studies.[144, 152, 159, 160] Two of the studies used National Health and Nutrition Examination Survey (NHANES) data on Hispanic/Latino participants,[152, 160] one study used the Multi-Ethnic Study of Atherosclerosis (MESA) cohort,[159] which included Mexican, other Hispanic, and Chinese populations, and one study used the Boston Puerto Rican Health Study cohort.[144]

The studies used different methods to assess acculturation. Four different multidimensional scales were used[144, 159, 160] and one study relied on the assessment of two acculturation proxies.[152] All measures took into consideration language usage with some only using this proxy and others including additional proxies for acculturation.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250439

CHAPTER SUMMARY

The individual is at the innermost core of the social-ecological model. In order for policy recommendations such as the *Dietary Guidelines for Americans* to be fully implemented, motivating and facilitating behavioral change at the individual level is required. The collective work presented in this chapter suggests a number of promising behavior change strategies that can be used to favorably impact a range of health related outcomes and to enhance the effectiveness of interventions. These include reducing screen time, reducing the frequency of eating out at fast- food restaurants, increasing frequency of family shared meals, and self-monitoring of diet and body weight as well as effective food labeling to target healthier food choices. These strategies complement comprehensive lifestyle interventions and nutrition counseling by qualified nutrition professionals. Timely feedback from registered dietitians/nutritionists and other qualified health professionals and engagement of the individual as appropriate in individual and group counseling will enhance outcomes. For this approach to work, it will be essential for the food environments where low-income individuals live to facilitate access to the selection of healthy food choices that respect their cultural preferences. Likewise, food and calorie label education should be designed to be understood for low literacy audiences some of which may have additional English language fluency limitations. While viable approaches are available now, additional research is necessary to improve the scientific foundation for more effective guidelines on individual level behavior change for all individuals living in the United States, taking into account the social, economic and cultural environments in which they live.

The evidence reviewed in this chapter indicates that the social, economic, and cultural context in which individuals live may facilitate or hinder their ability to choose and consume dietary patterns that are consistent with the Dietary Guidelines. Specifically household food insecurity hinders the access to healthy diets for millions of Americans. Also, immigrants are at high risk of losing the healthier dietary patterns characteristic of their cultural background as they acculturate into mainstream America. Furthermore, preventive nutrition services that take into account the social determinants of health are largely unavailable in our health system to systematically address the nutrition-related health problems of Americans including overweight and obesity, CVD, type 2 diabetes, and other chronic diseases. In summary, this chapter calls for: a) continuous support of Federal programs to help alleviate the consequences of household food insecurity, b) food and nutrition assistance programs to take into account the risk that immigrants have of giving up their healthier dietary habits soon after arriving in the United States, and c) efforts to provide all individuals living in the United States with the environments, knowledge, and tools needed to implement effective individual- or family-level behavioral change strategies to improve the quality of their diets and reduce sedentary behaviors. As indicated in ***Part D Chapter 4: Food Environment and Settings*** and ***Part D Chapter 5: Food Sustainability and Safety***, achieving these goals will require changes at all levels of the social-ecological model through coordinated efforts among health care and social and food systems from the national to the local level.

NEEDS FOR FUTURE RESEARCH

Eating Out

1. Develop a standard methodology to collect and characterize various types of eating venues.

   Rationale: This recommendation is fundamental to conducting rigorous research, evaluating findings from multiple studies, and developing policies to promote healthy eating among people who frequent eating out venues and/or consume take away meals.

2. Conduct rigorously designed research to examine the longitudinal impact of obtaining or consuming meals away from home from various types of commonly frequented venues on changes in food and beverage intakes (frequency, quantity, and composition), body weight, adiposity, and health profiles from childhood to adulthood in diverse (racial/ethnic, socioeconomic, cultural, and geographic) groups of males and females.

Rationale: Most groups in the U.S. population regularly consume meals that are prepared away from home and the landscape of fast food and other types of food procurement and consumption venues is increasingly complex. The potential for eating out and/or take away meals to influence diet quality, energy balance, body mass and composition, and the risks of health-related morbidities across the lifespan among our diverse population underscores the importance of understanding this issue.

Family Shared Meals

3. Conduct studies in diverse populations that assess not only frequency of family shared meals, but also quality of family shared meals.

   Rationale: Our understanding of the importance of family shared meals in terms of how they contribute in a positive way to body weight and overall health and well-being requires a rigorous examination of the dietary quality of these meals compared to other meals consumed by family members.

4. Conduct RCTs to isolate the effect of interventions that increase the frequency of family meals from other health and parenting behaviors that may be associated with dietary intake and weight status.

   Rationale: Family shared meals are commonly implemented as one component of lifestyle interventions that include an array of other behavioral and parenting strategies for weight management. To improve our understanding of the causal pathway of how family shared meals contributes to maintaining or achieving a health weight, the specific contribution of family shared meals to weight outcomes independent of other behavioral strategies needs to be ascertained.

Sedentary Behavior

5. Develop improved and better standardized and validated tools to assess sedentary behaviors and activities that children, adolescents, and adults regularly engage in.

   Rationale: Our understanding of the impact of sedentary behaviors on diet, energy balance, body mass, adiposity, and health is currently compromised by reliance on subjective assessments, including self-reports of daily activity patterns, and by inadequate techniques to document and quantify the array of sedentary activities people engage in (beyond TV viewing and (or) computer screen time). It also would be beneficial for researchers to document the potential benefits and implications of reducing one type of sedentary behavior (e.g. screen time) on other sedentary behaviors (e.g., reading for leisure, arts and crafts, listening to music) and indices of health (e.g. sleep quality and duration).

6. Conduct prospective research to examine the effects and mechanisms of the quantity, patterns, and changes of sedentary behaviors on diet quality, energy balance, body weight, adiposity, and health across the life span in groups within the U.S. population with diverse personal, cultural, economic, and geographic characteristics.

   Rationale: Emerging, but limited, evidence implicates sedentary behaviors with adverse health-related outcomes, especially in children and adolescents as they transition into adulthood. However, an improved understanding of why these relationships exist will help in developing appropriate and effective approaches and policies to reduce the amount of time people spend engaging in sedentary behaviors.

Self-Monitoring

7. Evaluate the impact of different types, modalities, and frequencies of self-monitoring on body weight outcomes during both the weight loss intervention and maintenance periods.

   Rationale: Self-monitoring is associated with improved weight management. However, the current practice of recommending daily self-monitoring may represent a barrier to its implementation and/or continued use. Hence, it is important to determine whether lower frequencies of self-monitoring can produce beneficial effects on weight outcomes.

8. Evaluate the comparative effectiveness of performance feedback from self-monitoring delivered through automated systems versus personal interactions with a counselor.

   Rationale: Automated feedback derived from self-monitoring data and delivered electronically can produce beneficial changes on weight outcomes. However, the comparative effectiveness and cost efficiency of feedback delivered through non-personal modalities versus personal interactions has yet to be determined.

9. Test the effectiveness of self-monitoring on weight outcomes in understudied groups, including ethnic/racial minorities, low education, low literacy, and low numeracy populations, males, and subjects younger than age 30 years and older than age 60 years.

   Rationale: Evidence regarding the effectiveness of self-monitoring has been derived largely from research conducted on well educated, middle-class, white women. Hence, it is important to determine whether the beneficial effects of self-monitoring on weight outcomes are generalizable to understudied groups.

10. Conduct RCTs based on sound behavioral change theories that incorporate self-monitoring, employ heterogeneous populations, and are powered for small effect sizes and high attrition rates, to test the short- (e.g., 3 months) and long-term (e.g., 12 months) effects of mobile health technologies on dietary and weight outcomes.

Rationale: Mobile health technologies have the potential to reach larger portions of the populations than face-to-face interventions, but the effect sizes of mobile technologies may be small and the attrition rates may be large. Larger, more representative study populations and longer study periods will permit an assessment of the generalizability and sustainability of mobile health technologies.

Food and Menu Labeling

11. Develop novel labeling approaches to provide informative strategies to convey caloric intake values on food items consumed at home and in restaurant settings.

Rationale: Menu labels can include different types of information in addition to calories. These include physical activity equivalents, and daily caloric needs. Very few studies have been designed to examine the optimal combination of menu label information to prevent excessive caloric intake. This will be very valuable evidence to inform the calorie label policy that has just been enacted by the FDA.

12. Compare labeling strategies across various settings, such as restaurants, stores, and the home to determine their efficacy in altering food selection and health outcomes, including weight.

Rationale: The great majority of menu labeling RCT's have been conducted under laboratory conditions. Given the recent FDA regulations, future studies will be able to impact the effectiveness of these polices across settings as accessed by diverse free living populations.

13. Evaluate the process and impact of recent FDA menu labeling regulation.

Rationale: The new FDA regulation provides a unique opportunity to understand the impact of menu labeling on consumers dietary behaviors in "real world" settings.

Household Food Insecurity

14. Conduct prospective cohort studies that cover a wide age range and include children, families, older adults, and ethnically/racially diverse populations and describe potential effect modifiers such as gender, ethnic and cultural factors, family structure, area of residence (i.e., urban vs. rural), employment, and use of social support systems while examining the relationship between household food insecurity, dietary intake, and body weight.

Rationale: Understanding the temporal process of when and how long food insecurity occurs within a family/individual’s lifetime and their response to this economic stressor is critical to conducting rigorous research and comparing finding across studies in order to develop and implement intervention studies and policies to alleviate this public health problem.

15. Standardize research methodology, including developing a consistent approach to measuring food insecurity and use of measured height and weight to reduce the likelihood of responder bias.

    Rationale: The measurement error issues related to the use of self-reported weight have been well documented in the literature. In order to conduct rigorous studies in this area that can be compared and evaluated as to the causal nature of the role of food insecurity on body weight, standard methodology is warranted both in the measurement of the exposure as well as the outcome.

Acculturation

16. Conduct prospective longitudinal studies including those that start in early childhood to track dietary intake, sedentary behaviors, body weight, and chronic disease outcomes across the lifespan. Include the diversity of ethnic/racial groups in the United States, including individuals and families of diverse national origins. Include comparison groups in countries of origin to rule out, among other things, the potential confounding by internal migration from rural to urban area within the country of origin.

    Rationale: Acculturation is a time-dependent life course process that requires longitudinal studies to be properly understood. Because the impact of acculturation on dietary, weight and health outcomes can be expected to be modified by the life course stage of life when individuals migrate to the United States, prospective acculturation studies need to start following individuals from very early childhood.

17. Develop a standard tool to measure acculturation or validation of multidimensional acculturation scales in different immigrant groups and in different languages.

    Rationale: Acculturation is a complex construct that is seldom measured with multidimensional scales that can capture the different paths that migrant scan take with regards to the acculturation process, including assimilation, integration, segregation, and marginalization. Although research in acculturation measurement has been conducted among Hispanic/Latinos, it has been predominantly based on Mexican American populations and little acculturation measurement research has been conducted among other groups, including individuals from Asia, Africa, Europe, and the Middle East.

Sleep Patterns

18. Conduct prospective studies that start in childhood (including transition to adulthood), to investigate the longitudinal effect of sleep patterns on diet and body weight outcomes while accounting for confounders, mediators, and moderators including: physical activity, socioeconomic variables (such as education, employment, household income), sex, alcohol

intake, smoking status (including new smoker, new non-smoker), media use/screen time, and depression.

Rationale: While research associates short sleep duration and disordered sleep patterns with adverse differences and changes in food and beverage consumption, body weight, and indices of metabolic and cardiovascular health, less is known about the impact of potential modifying lifestyle factors. This research will help delineate the role of sleep patterns, duration and quality, i.e., mediator or moderator, on diet and weigh-related outcomes. Research in children shows that sleep deprivation and weight are related but this relationship is not apparent in adult studies. This may be due to the fact that energy intake increases during transition to short sleep duration, but levels off when short sleep duration becomes consistent.

19. Conduct studies to assess the effects of diet on sleep quality to examine the mechanism by which dietary intake, energy intake, and energy expenditure may impact sleep.

Rationale: Most research has focused on sleep quality and duration as modifying factors on diet, body weight, and health. A paucity of research exists on the potential impact of diet on sleep-related outcomes. This line of research would use diet as the means to improve indices of sleep, which in turn may subsequently improve health-related outcomes.

REFERENCES

1. Coleman-Jensen A, Gregory C, Singh A. Household food security in the United States in 2013. 2014. http://www.ers.usda.gov/media/1565415/err173.pdf.
2. United States. Department of Agriculture. Human Nutrition Information Service. Dietary Guidelines Advisory Committee., United States. Agricultural Research Service. Report of the Dietary Guidelines Advisory Committee on the dietary guidelines for Americans, 2010 : to the Secretary of Agriculture and the Secretary of Health and Human Services. 2010:vi, 445 p. PMID: 16634830. http://www.cnpp.usda.gov/sites/default/files/dietary_guidelines_for_americans/2010DGACReport-camera-ready-Jan11-11.pdf.
3. Owen N, Healy GN, Matthews CE, Dunstan DW. Too much sitting: the population health science of sedentary behavior. Exerc Sport Sci Rev. 2010;38(3):105-13. PMID: 20577058. http://www.ncbi.nlm.nih.gov/pubmed/20577058.
4. McGinnis JM, Gootman JA, Kraak VI. Food marketing to children and youth: threat or opportunity? 2006. http://static.publico.pt/docs/pesoemedida/Iom_Food_Marketing_To_Children_Exec_Summary_2006.pdf.
5. Golem DL, Martin-Biggers JT, Koenings MM, Davis KF, Byrd-Bredbenner C. An integrative review of sleep for nutrition professionals. Adv Nutr. 2014;5(6):742-59. PMID: 25398735. http://www.ncbi.nlm.nih.gov/pubmed/25398735.
6. Butryn ML, Webb V, Wadden TA. Behavioral treatment of obesity. Psychiatr Clin North Am. 2011;34(4):841-59. PMID: 22098808. http://www.ncbi.nlm.nih.gov/pubmed/22098808.

7. Core indicators of nutritional state for difficult-to-sample populations. J Nutr. 1990;120 Suppl 11:1559-600. PMID: 2243305. http://www.ncbi.nlm.nih.gov/pubmed/2243305.
8. Perez-Escamilla R. Food insecurity in children: impact on physical, psychoemotional and social development. In: Ross CA, Caballero RJ, Cousins RJ, Tucker KL, Ziegler TR, editors. Modern Nutrition in Health and Disease. Baltimore, MD: Lippincott Williams & Wilkins; 2013. p. 1006-15.
9. Lee JS, Gundersen C, Cook J, Laraia B, Johnson MA. Food insecurity and health across the lifespan. Adv Nutr. 2012;3(5):744-5. PMID: 22983862. http://www.ncbi.nlm.nih.gov/pubmed/22983862.
10. Statistical portrait of the foreign-born population in the United States, 2010. Pew Research Hispanic Trends Project. 2012. http://www.pewhispanic.org/2014/04/29/statistical-portrait-of-the-foreign-born-population-in-the-united-states-2012/.
11. Yearbook of immigration statistics: 2013. Table 1 Persons Obtaining Lawful Permanet Resident Status: Fiscal Years 1820 to 2013. http://www.dhs.gov/yearbook-immigration-statistics-2013-lawful-permanent-residents.
12. Abraido-Lanza AF, White K, Vasques E. Immigrant populations and health. In: Anderson N, editor. Encyclopedia of Health and Behavior. Newbury Park, CA: Sage; 2004. p. 533-7.
13. Redfield R, Linton R, Herskovits MJ. Memorandum for the study of acculturation. American Anthropologist. 1936;38(1):149-52.
14. Perez-Escamilla R, Putnik P. The role of acculturation in nutrition, lifestyle, and incidence of type 2 diabetes among Latinos. J Nutr. 2007;137(4):860-70. PMID: 17374645. http://www.ncbi.nlm.nih.gov/pubmed/17374645.
15. Bes-Rastrollo M, Basterra-Gortari FJ, Sanchez-Villegas A, Marti A, Martinez JA, Martinez-Gonzalez MA. A prospective study of eating away-from-home meals and weight gain in a Mediterranean population: the SUN (Seguimiento Universidad de Navarra) cohort. Public Health Nutr. 2010;13(9):1356-63. PMID: 19954575. http://www.ncbi.nlm.nih.gov/pubmed/19954575.
16. Boggs DA, Rosenberg L, Coogan PF, Makambi KH, Adams-Campbell LL, Palmer JR. Restaurant foods, sugar-sweetened soft drinks, and obesity risk among young African American women. Ethn Dis. 2013;23(4):445-51. PMID: 24392607. http://www.ncbi.nlm.nih.gov/pubmed/24392607.
17. Duffey KJ, Gordon-Larsen P, Jacobs DR, Jr., Williams OD, Popkin BM. Differential associations of fast food and restaurant food consumption with 3-y change in body mass index: the Coronary Artery Risk Development in Young Adults Study. Am J Clin Nutr. 2007;85(1):201-8. PMID: 17209197. http://www.ncbi.nlm.nih.gov/pubmed/17209197.
18. Duffey KJ, Gordon-Larsen P, Steffen LM, Jacobs DR, Jr., Popkin BM. Regular consumption from fast food establishments relative to other restaurants is differentially associated with metabolic outcomes in young adults. J Nutr. 2009;139(11):2113-8. PMID: 19776183. http://www.ncbi.nlm.nih.gov/pubmed/19776183.
19. Fraser LK, Clarke GP, Cade JE, Edwards KL. Fast food and obesity: a spatial analysis in a large United Kingdom population of children aged 13-15. Am J Prev Med. 2012;42(5):e77-85. PMID: 22516506. http://www.ncbi.nlm.nih.gov/pubmed/22516506.
20. French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates. Int J Obes Relat

Metab Disord. 2000;24(10):1353-9. PMID: 11093299. http://www.ncbi.nlm.nih.gov/pubmed/11093299.

21. Haines J, Neumark-Sztainer D, Wall M, Story M. Personal, behavioral, and environmental risk and protective factors for adolescent overweight. Obesity (Silver Spring). 2007;15(11):2748-60. PMID: 18070766. http://www.ncbi.nlm.nih.gov/pubmed/18070766.
22. Laska MN, Murray DM, Lytle LA, Harnack LJ. Longitudinal associations between key dietary behaviors and weight gain over time: transitions through the adolescent years. Obesity (Silver Spring). 2012;20(1):118-25. PMID: 21701567. http://www.ncbi.nlm.nih.gov/pubmed/21701567.
23. Li F, Harmer P, Cardinal BJ, Bosworth M, Johnson-Shelton D, Moore JM, et al. Built environment and 1-year change in weight and waist circumference in middle-aged and older adults: Portland Neighborhood Environment and Health Study. Am J Epidemiol. 2009;169(4):401-8. PMID: 19153214. http://www.ncbi.nlm.nih.gov/pubmed/19153214.
24. MacFarlane A, Cleland V, Crawford D, Campbell K, Timperio A. Longitudinal examination of the family food environment and weight status among children. Int J Pediatr Obes. 2009;4(4):343-52. PMID: 19922051. http://www.ncbi.nlm.nih.gov/pubmed/19922051.
25. Niemeier HM, Raynor HA, Lloyd-Richardson EE, Rogers ML, Wing RR. Fast food consumption and breakfast skipping: predictors of weight gain from adolescence to adulthood in a nationally representative sample. J Adolesc Health. 2006;39(6):842-9. PMID: 17116514. http://www.ncbi.nlm.nih.gov/pubmed/17116514.
26. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR, Jr., et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet. 2005;365(9453):36-42. PMID: 15639678. http://www.ncbi.nlm.nih.gov/pubmed/15639678.
27. Taveras EM, Berkey CS, Rifas-Shiman SL, Ludwig DS, Rockett HR, Field AE, et al. Association of consumption of fried food away from home with body mass index and diet quality in older children and adolescents. Pediatrics. 2005;116(4):e518-24. PMID: 16199680. http://www.ncbi.nlm.nih.gov/pubmed/16199680.
28. Thompson OM, Ballew C, Resnicow K, Must A, Bandini LG, Cyr H, et al. Food purchased away from home as a predictor of change in BMI z-score among girls. Int J Obes Relat Metab Disord. 2004;28(2):282-9. PMID: 14647177. http://www.ncbi.nlm.nih.gov/pubmed/14647177.
29. Bisset S, Gauvin L, Potvin L, Paradis G. Association of body mass index and dietary restraint with changes in eating behaviour throughout late childhood and early adolescence: a 5-year study. Public Health Nutr. 2007;10(8):780-9. PMID: 17381909. http://www.ncbi.nlm.nih.gov/pubmed/17381909.
30. Anderson SE, Whitaker RC. Household routines and obesity in US preschool-aged children. Pediatrics. 2010;125(3):420-8. PMID: 20142280. http://www.ncbi.nlm.nih.gov/pubmed/20142280.
31. Ayala GX, Baquero B, Arredondo EM, Campbell N, Larios S, Elder JP. Association between family variables and Mexican American children's dietary behaviors. J Nutr Educ Behav. 2007;39(2):62-9. PMID: 17346653. http://www.ncbi.nlm.nih.gov/pubmed/17346653.

32. Gillman MW, Rifas-Shiman SL, Frazier AL, Rockett HR, Camargo CA, Jr., Field AE, et al. Family dinner and diet quality among older children and adolescents. Arch Fam Med. 2000;9(3):235-40. PMID: 10728109. http://www.ncbi.nlm.nih.gov/pubmed/10728109.

33. Videon TM, Manning CK. Influences on adolescent eating patterns: the importance of family meals. J Adolesc Health. 2003;32(5):365-73. PMID: 12729986. http://www.ncbi.nlm.nih.gov/pubmed/12729986.

34. Hammons AJ, Fiese BH. Is frequency of shared family meals related to the nutritional health of children and adolescents? Pediatrics. 2011;127(6):e1565-74. PMID: 21536618. http://www.ncbi.nlm.nih.gov/pubmed/21536618.

35. Sobal J, Hanson K. Family meals and body weight in US adults. Public Health Nutr. 2011;14(9):1555-62. PMID: 21356147. http://www.ncbi.nlm.nih.gov/pubmed/21356147.

36. Chan JC, Sobal J. Family meals and body weight. Analysis of multiple family members in family units. Appetite. 2011;57(2):517-24. PMID: 21763740. http://www.ncbi.nlm.nih.gov/pubmed/21763740.

37. Fulkerson JA, Larson N, Horning M, Neumark-Sztainer D. A review of associations between family or shared meal frequency and dietary and weight status outcomes across the lifespan. J Nutr Educ Behav. 2014;46(1):2-19. PMID: 24054888. http://www.ncbi.nlm.nih.gov/pubmed/24054888.

38. Savage JS, Fisher JO, Birch LL. Parental influence on eating behavior: conception to adolescence. J Law Med Ethics. 2007;35(1):22-34. PMID: 17341215. http://www.ncbi.nlm.nih.gov/pubmed/17341215.

39. Ventura AK, Birch LL. Does parenting affect children's eating and weight status? Int J Behav Nutr Phys Act. 2008;5:15. PMID: 18346282. http://www.ncbi.nlm.nih.gov/pubmed/18346282.

40. Burgess-Champoux TL, Larson N, Neumark-Sztainer D, Hannan PJ, Story M. Are family meal patterns associated with overall diet quality during the transition from early to middle adolescence? J Nutr Educ Behav. 2009;41(2):79-86. PMID: 19304252. http://www.ncbi.nlm.nih.gov/pubmed/19304252.

41. Larson N, Fulkerson J, Story M, Neumark-Sztainer D. Shared meals among young adults are associated with better diet quality and predicted by family meal patterns during adolescence. Public Health Nutr. 2013;16(5):883-93. PMID: 22857517. http://www.ncbi.nlm.nih.gov/pubmed/22857517.

42. Haines J, McDonald J, O'Brien A, Sherry B, Bottino CJ, Schmidt ME, et al. Healthy Habits, Happy Homes: randomized trial to improve household routines for obesity prevention among preschool-aged children. JAMA Pediatr. 2013;167(11):1072-9. PMID: 24019074. http://www.ncbi.nlm.nih.gov/pubmed/24019074.

43. Fulkerson JA, Neumark-Sztainer D, Hannan PJ, Story M. Family meal frequency and weight status among adolescents: cross-sectional and 5-year longitudinal associations. Obesity (Silver Spring). 2008;16(11):2529-34. PMID: 18719674. http://www.ncbi.nlm.nih.gov/pubmed/18719674.

44. Gable S, Chang Y, Krull JL. Television watching and frequency of family meals are predictive of overweight onset and persistence in a national sample of school-aged children. J Am Diet Assoc. 2007;107(1):53-61. PMID: 17197271. http://www.ncbi.nlm.nih.gov/pubmed/17197271.

45. Miller DP. Associations between the home and school environments and child body mass index. Soc Sci Med. 2011;72(5):677-84. PMID: 21227558. http://www.ncbi.nlm.nih.gov/pubmed/21227558.

46. Sen B. Frequency of family dinner and adolescent body weight status: evidence from the national longitudinal survey of youth, 1997. Obesity (Silver Spring). 2006;14(12):2266-76. PMID: 17189555. http://www.ncbi.nlm.nih.gov/pubmed/17189555.

47. Taveras EM, Rifas-Shiman SL, Berkey CS, Rockett HR, Field AE, Frazier AL, et al. Family dinner and adolescent overweight. Obes Res. 2005;13(5):900-6. PMID: 15919844. http://www.ncbi.nlm.nih.gov/pubmed/15919844.

48. United States. Department of Health and Human Services. 2008 physical activity guidelines for Americans : be active, healthy, and happy! ODPHP publication. 2008(no U0036):ix, 61 p. PMID: 15630651. http://www.health.gov/paguidelines/pdf/paguide.pdf.

49. Healthy People 2020. PA-2. Increase the proportion of adults who meet current federal physical activity guidelines for aeorbic physical activity and for muscle-strengthening activity. Washington, DC: U.S. Department of Health and Human Services. Office of Disease Prevention and Health Promotion; [cited 2014]. Available from: https://www.healthypeople.gov/2020/topics-objectives/topic/physical-activity/objectives.

50. Healthy People 2020. PA-3. Increase the proportion of adolescents who meet current federal physical activity guidelines for aerobic physical activity and for muscle-strengthening activity. Washington, DC: U.S. Department of Health and Human Services. Office of Disease Prevention and Health Promotion; [cited 2014]. Available from: https://www.healthypeople.gov/2020/topics-objectives/topic/physical-activity/objectives.

51. Healthy People 2020. PA-1. Reduce the proportion of adults who engage in no leisure-time physical activity. Washington, DC: U.S. Department of Health and Human Services. Office of Disease Prevention and Health Promotion; [cited 2014]. Available from: https://www.healthypeople.gov/node/5052/data_details.

52. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. Lancet. 2012;380(9838):219-29. PMID: 22818936. http://www.ncbi.nlm.nih.gov/pubmed/22818936.

53. Policy Statement: children, adolescents, and the media. Pediatrics. 2013;132(5):958-61. http://pediatrics.aappublications.org/content/132/5/958.full.html.

54. Rideout V. Zero to eight: children's media use in America 2013. 2013. https://www.commonsensemedia.org/sites/default/files/research/zero-to-eight-2013.pdf.

55. 2014 United States Report Card on Physical Activity for Children and Youth. 2014. http://www.physicalactivityplan.org/reportcard/NationalReportCard_longform_final%20for%20web.pdf.

56. Fakhouri TH, Hughes JP, Brody DJ, Kit BK, Ogden CL. Physical activity and screen-time viewing among elementary school-aged children in the United States from 2009 to 2010. JAMA Pediatr. 2013;167(3):223-9. PMID: 23303439. http://www.ncbi.nlm.nih.gov/pubmed/23303439.

57. Blanck HM, McCullough ML, Patel AV, Gillespie C, Calle EE, Cokkinides VE, et al. Sedentary behavior, recreational physical activity, and 7-year weight gain among postmenopausal U.S. women. Obesity (Silver Spring). 2007;15(6):1578-88. PMID: 17557996. http://www.ncbi.nlm.nih.gov/pubmed/17557996.

58. Boone JE, Gordon-Larsen P, Adair LS, Popkin BM. Screen time and physical activity during adolescence: longitudinal effects on obesity in young adulthood. Int J Behav Nutr Phys Act. 2007;4:26. PMID: 17559668. http://www.ncbi.nlm.nih.gov/pubmed/17559668.
59. De Cocker KA, van Uffelen JG, Brown WJ. Associations between sitting time and weight in young adult Australian women. Prev Med. 2010;51(5):361-7. PMID: 20638408. http://www.ncbi.nlm.nih.gov/pubmed/20638408.
60. Ding D, Sugiyama T, Owen N. Habitual active transport, TV viewing and weight gain: a four year follow-up study. Prev Med. 2012;54(3-4):201-4. PMID: 22342646. http://www.ncbi.nlm.nih.gov/pubmed/22342646.
61. Ekelund U, Brage S, Besson H, Sharp S, Wareham NJ. Time spent being sedentary and weight gain in healthy adults: reverse or bidirectional causality? Am J Clin Nutr. 2008;88(3):612-7. PMID: 18779275. http://www.ncbi.nlm.nih.gov/pubmed/18779275.
62. French SA, Mitchell NR, Hannan PJ. Decrease in television viewing predicts lower body mass index at 1-year follow-up in adolescents, but not adults. J Nutr Educ Behav. 2012;44(5):415-22. PMID: 22591582. http://www.ncbi.nlm.nih.gov/pubmed/22591582.
63. Hancox RJ, Milne BJ, Poulton R. Association between child and adolescent television viewing and adult health: a longitudinal birth cohort study. Lancet. 2004;364(9430):257-62. PMID: 15262103. http://www.ncbi.nlm.nih.gov/pubmed/15262103.
64. Landhuis CE, Poulton R, Welch D, Hancox RJ. Childhood sleep time and long-term risk for obesity: a 32-year prospective birth cohort study. Pediatrics. 2008;122(5):955-60. PMID: 18977973. http://www.ncbi.nlm.nih.gov/pubmed/18977973.
65. Mamun AA, O'Callaghan MJ, Williams G, Najman JM. Television watching from adolescence to adulthood and its association with BMI, waist circumference, waist-to-hip ratio and obesity: a longitudinal study. Public Health Nutr. 2013;16(1):54-64. PMID: 22687709. http://www.ncbi.nlm.nih.gov/pubmed/22687709.
66. Meyer AM, Evenson KR, Couper DJ, Stevens J, Pereria MA, Heiss G. Television, physical activity, diet, and body weight status: the ARIC cohort. Int J Behav Nutr Phys Act. 2008;5:68. PMID: 19091124. http://www.ncbi.nlm.nih.gov/pubmed/19091124.
67. Mortensen LH, Siegler IC, Barefoot JC, Gronbaek M, Sorensen TI. Prospective associations between sedentary lifestyle and BMI in midlife. Obesity (Silver Spring). 2006;14(8):1462-71. PMID: 16988090. http://www.ncbi.nlm.nih.gov/pubmed/16988090.
68. Nunez-Cordoba JM, Bes-Rastrollo M, Pollack KM, Segui-Gomez M, Beunza JJ, Sayon-Orea C, et al. Annual motor vehicle travel distance and incident obesity: a prospective cohort study. Am J Prev Med. 2013;44(3):254-9. PMID: 23415122. http://www.ncbi.nlm.nih.gov/pubmed/23415122.
69. Parsons TJ, Manor O, Power C. Television viewing and obesity: a prospective study in the 1958 British birth cohort. Eur J Clin Nutr. 2008;62(12):1355-63. PMID: 17717536. http://www.ncbi.nlm.nih.gov/pubmed/17717536.
70. Pulsford RM, Stamatakis E, Britton AR, Brunner EJ, Hillsdon MM. Sitting behavior and obesity: evidence from the Whitehall II study. Am J Prev Med. 2013;44(2):132-8. PMID: 23332328. http://www.ncbi.nlm.nih.gov/pubmed/23332328.
71. Raynor DA, Phelan S, Hill JO, Wing RR. Television viewing and long-term weight maintenance: results from the National Weight Control Registry. Obesity (Silver Spring). 2006;14(10):1816-24. PMID: 17062812. http://www.ncbi.nlm.nih.gov/pubmed/17062812.

72. Saunders TJ, Tremblay MS, Despres JP, Bouchard C, Tremblay A, Chaput JP. Sedentary behaviour, visceral fat accumulation and cardiometabolic risk in adults: a 6-year longitudinal study from the Quebec Family Study. PLoS One. 2013;8(1):e54225. PMID: 23326600. http://www.ncbi.nlm.nih.gov/pubmed/23326600.

73. Stamatakis E, Hamer M, Mishra GD. Early adulthood television viewing and cardiometabolic risk profiles in early middle age: results from a population, prospective cohort study. Diabetologia. 2012;55(2):311-20. PMID: 22057195. http://www.ncbi.nlm.nih.gov/pubmed/22057195.

74. Sugiyama T, Ding D, Owen N. Commuting by car: weight gain among physically active adults. Am J Prev Med. 2013;44(2):169-73. PMID: 23332335. http://www.ncbi.nlm.nih.gov/pubmed/23332335.

75. van Uffelen JG, Watson MJ, Dobson AJ, Brown WJ. Sitting time is associated with weight, but not with weight gain in mid-aged Australian women. Obesity (Silver Spring). 2010;18(9):1788-94. PMID: 20111018. http://www.ncbi.nlm.nih.gov/pubmed/20111018.

76. Viner RM, Cole TJ. Television viewing in early childhood predicts adult body mass index. J Pediatr. 2005;147(4):429-35. PMID: 16227025. http://www.ncbi.nlm.nih.gov/pubmed/16227025.

77. Wijndaele K, Healy GN, Dunstan DW, Barnett AG, Salmon J, Shaw JE, et al. Increased cardiometabolic risk is associated with increased TV viewing time. Med Sci Sports Exerc. 2010;42(8):1511-8. PMID: 20139784. http://www.ncbi.nlm.nih.gov/pubmed/20139784.

78. Pinto Pereira SM, Power C. Sedentary behaviours in mid-adulthood and subsequent body mass index. PLoS One. 2013;8(6):e65791. PMID: 23762427. http://www.ncbi.nlm.nih.gov/pubmed/23762427.

79. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 2011;364(25):2392-404. PMID: 21696306. http://www.ncbi.nlm.nih.gov/pubmed/21696306.

80. Increasing physical activity: behavioral and social approaches: Guide to Community Preventive Services; [cited 2014 September 10]. Available from: http://www.thecommunityguide.org/pa/behavioral-social/index.html.

81. Burke LE, Wang J, Sevick MA. Self-monitoring in weight loss: a systematic review of the literature. J Am Diet Assoc. 2011;111(1):92-102. PMID: 21185970. http://www.ncbi.nlm.nih.gov/pubmed/21185970.

82. Adachi Y, Sato C, Yamatsu K, Ito S, Adachi K, Yamagami T. A randomized controlled trial on the long-term effects of a 1-month behavioral weight control program assisted by computer tailored advice. Behav Res Ther. 2007;45(3):459-70. PMID: 16713991. http://www.ncbi.nlm.nih.gov/pubmed/16713991.

83. de Niet J, Timman R, Bauer S, van den Akker E, de Klerk C, Kordy H, et al. Short message service reduces dropout in childhood obesity treatment: a randomized controlled trial. Health Psychol. 2012;31(6):797-805. PMID: 22468714. http://www.ncbi.nlm.nih.gov/pubmed/22468714.

84. Steinberg DM, Tate DF, Bennett GG, Ennett S, Samuel-Hodge C, Ward DS. The efficacy of a daily self-weighing weight loss intervention using smart scales and e-mail. Obesity (Silver Spring). 2013;21(9):1789-97. PMID: 23512320. http://www.ncbi.nlm.nih.gov/pubmed/23512320.

85. Wing RR, Crane MM, Thomas JG, Kumar R, Weinberg B. Improving weight loss outcomes of community interventions by incorporating behavioral strategies. Am J Public Health. 2010;100(12):2513-9. PMID: 20966375. http://www.ncbi.nlm.nih.gov/pubmed/20966375.

86. Bartfield JK, Stevens VJ, Jerome GJ, Batch BC, Kennedy BM, Vollmer WM, et al. Behavioral transitions and weight change patterns within the PREMIER trial. Obesity (Silver Spring). 2011;19(8):1609-15. PMID: 21455122. http://www.ncbi.nlm.nih.gov/pubmed/21455122.

87. Burke LE, Styn MA, Sereika SM, Conroy MB, Ye L, Glanz K, et al. Using mHealth technology to enhance self-monitoring for weight loss: a randomized trial. Am J Prev Med. 2012;43(1):20-6. PMID: 22704741. http://www.ncbi.nlm.nih.gov/pubmed/22704741.

88. Harrison CL, Teede HJ, Lombard CB. How effective is self-weighing in the setting of a lifestyle intervention to reduce gestational weight gain and postpartum weight retention? Aust N Z J Obstet Gynaecol. 2014;54(4):382-5. PMID: 24738837. http://www.ncbi.nlm.nih.gov/pubmed/24738837.

89. Kong A, Beresford SA, Alfano CM, Foster-Schubert KE, Neuhouser ML, Johnson DB, et al. Self-monitoring and eating-related behaviors are associated with 12-month weight loss in postmenopausal overweight-to-obese women. J Acad Nutr Diet. 2012;112(9):1428-35. PMID: 22795495. http://www.ncbi.nlm.nih.gov/pubmed/22795495.

90. Linde JA, Jeffery RW, French SA, Pronk NP, Boyle RG. Self-weighing in weight gain prevention and weight loss trials. Ann Behav Med. 2005;30(3):210-6. PMID: 16336072. http://www.ncbi.nlm.nih.gov/pubmed/16336072.

91. Peterson ND, Middleton KR, Nackers LM, Medina KE, Milsom VA, Perri MG. Dietary self-monitoring and long-term success with weight management. Obesity (Silver Spring). 2014;22(9):1962-7. PMID: 24931055. http://www.ncbi.nlm.nih.gov/pubmed/24931055.

92. Steinberg DM, Levine EL, Lane I, Askew S, Foley PB, Puleo E, et al. Adherence to self-monitoring via interactive voice response technology in an eHealth intervention targeting weight gain prevention among Black women: randomized controlled trial. J Med Internet Res. 2014;16(4):e114. PMID: 24780934. http://www.ncbi.nlm.nih.gov/pubmed/24780934.

93. Turk MW, Elci OU, Wang J, Sereika SM, Ewing LJ, Acharya SD, et al. Self-monitoring as a mediator of weight loss in the SMART randomized clinical trial. Int J Behav Med. 2013;20(4):556-61. PMID: 22936524. http://www.ncbi.nlm.nih.gov/pubmed/22936524.

94. Turner-McGrievy GM, Beets MW, Moore JB, Kaczynski AT, Barr-Anderson DJ, Tate DF. Comparison of traditional versus mobile app self-monitoring of physical activity and dietary intake among overweight adults participating in an mHealth weight loss program. J Am Med Inform Assoc. 2013;20(3):513-8. PMID: 23429637. http://www.ncbi.nlm.nih.gov/pubmed/23429637.

95. VanWormer JJ, Linde JA, Harnack LJ, Stovitz SD, Jeffery RW. Self-weighing frequency is associated with weight gain prevention over 2 years among working adults. Int J Behav Med. 2012;19(3):351-8. PMID: 21732212. http://www.ncbi.nlm.nih.gov/pubmed/21732212.

96. VanWormer JJ, Martinez AM, Martinson BC, Crain AL, Benson GA, Cosentino DL, et al. Self-weighing promotes weight loss for obese adults. Am J Prev Med. 2009;36(1):70-3. PMID: 18976879. http://www.ncbi.nlm.nih.gov/pubmed/18976879.

97. Wing RR, Hamman RF, Bray GA, Delahanty L, Edelstein SL, Hill JO, et al. Achieving weight and activity goals among diabetes prevention program lifestyle participants. Obes Res. 2004;12(9):1426-34. PMID: 15483207. http://www.ncbi.nlm.nih.gov/pubmed/15483207.

98. Wing RR, Papandonatos G, Fava JL, Gorin AA, Phelan S, McCaffery J, et al. Maintaining large weight losses: the role of behavioral and psychological factors. J Consult Clin Psychol. 2008;76(6):1015-21. PMID: 19045969. http://www.ncbi.nlm.nih.gov/pubmed/19045969.

99. Ma J, Xiao L, Blonstein AC. Measurement of self-monitoring web technology acceptance and use in an e-health weight-loss trial. Telemed J E Health. 2013;19(10):739-45. PMID: 23952787. http://www.ncbi.nlm.nih.gov/pubmed/23952787.

100. Krukowski RA, Harvey-Berino J, Bursac Z, Ashikaga T, West DS. Patterns of success: online self-monitoring in a web-based behavioral weight control program. Health Psychol. 2013;32(2):164-70. PMID: 22545978. http://www.ncbi.nlm.nih.gov/pubmed/22545978.

101. Johnson F, Wardle J. The association between weight loss and engagement with a web-based food and exercise diary in a commercial weight loss programme: a retrospective analysis. Int J Behav Nutr Phys Act. 2011;8:83. PMID: 21810222. http://www.ncbi.nlm.nih.gov/pubmed/21810222.

102. Sinclair SE, Cooper M, Mansfield ED. The influence of menu labeling on calories selected or consumed: a systematic review and meta-analysis. J Acad Nutr Diet. 2014;114(9):1375-88 e15. PMID: 25037558. http://www.ncbi.nlm.nih.gov/pubmed/25037558.

103. Burton S, Creyer EH, Kees J, Huggins K. Attacking the obesity epidemic: the potential health benefits of providing nutrition information in restaurants. Am J Public Health. 2006;96(9):1669-75. PMID: 16873758. http://www.ncbi.nlm.nih.gov/pubmed/16873758.

104. Dowray S, Swartz JJ, Braxton D, Viera AJ. Potential effect of physical activity based menu labels on the calorie content of selected fast food meals. Appetite. 2013;62:173-81. PMID: 23220355. http://www.ncbi.nlm.nih.gov/pubmed/23220355.

105. Ellison B, Lusk JL, Davis D. Looking at the label and beyond: the effects of calorie labels, health consciousness, and demographics on caloric intake in restaurants. Int J Behav Nutr Phys Act. 2013;10:21. PMID: 23394433. http://www.ncbi.nlm.nih.gov/pubmed/23394433.

106. Gerend MA. Does calorie information promote lower calorie fast food choices among college students? J Adolesc Health. 2009;44(1):84-6. PMID: 19101463. http://www.ncbi.nlm.nih.gov/pubmed/19101463.

107. Harnack LJ, French SA, Oakes JM, Story MT, Jeffery RW, Rydell SA. Effects of calorie labeling and value size pricing on fast food meal choices: results from an experimental trial. Int J Behav Nutr Phys Act. 2008;5:63. PMID: 19061510. http://www.ncbi.nlm.nih.gov/pubmed/19061510.

108. James A, Adams-Huet B, Shah M. Menu Labels Displaying the Kilocalorie Content or the Exercise Equivalent: Effects on Energy Ordered and Consumed in Young Adults. Am J Health Promot. 2014. PMID: 24575727. http://www.ncbi.nlm.nih.gov/pubmed/24575727.

109. Roberto CA, Larsen PD, Agnew H, Baik J, Brownell KD. Evaluating the impact of menu labeling on food choices and intake. Am J Public Health. 2010;100(2):312-8. PMID: 20019307. http://www.ncbi.nlm.nih.gov/pubmed/20019307.
110. Roseman MG, Mathe-Soulek K, Higgins JA. Relationships among grocery nutrition label users and consumers' attitudes and behavior toward restaurant menu labeling. Appetite. 2013;71:274-8. PMID: 24012965. http://www.ncbi.nlm.nih.gov/pubmed/24012965.
111. Tandon PS, Wright J, Zhou C, Rogers CB, Christakis DA. Nutrition menu labeling may lead to lower-calorie restaurant meal choices for children. Pediatrics. 2010;125(2):244-8. PMID: 20100765. http://www.ncbi.nlm.nih.gov/pubmed/20100765.
112. Wisdom J, Downs JS, Loewenstein G. Promoting health choices: information versus convenience. Am Econ J Appl Econ. 2010;2(2):164-78.
113. Laraia BA, Siega-Riz AM, Gundersen C, Dole N. Psychosocial factors and socioeconomic indicators are associated with household food insecurity among pregnant women. J Nutr. 2006;136(1):177-82. PMID: 16365079. http://www.ncbi.nlm.nih.gov/pubmed/16365079.
114. Perez-Escamilla R, Pinheiro de Toledo Vianna R. Food insecurity and the behavioral and intellectual development of children: a review of the evidence. J Appl Res Child. 2012;3(1). http://digitalcommons.library.tmc.edu/childrenatrisk/vol3/iss1/9/.
115. Leung CW, Epel ES, Ritchie LD, Crawford PB, Laraia BA. Food Insecurity Is Inversely Associated with Diet Quality of Lower-Income Adults. J Acad Nutr Diet. 2014;114(12):1943-53 e2. PMID: 25091796. http://www.ncbi.nlm.nih.gov/pubmed/25091796.
116. Hartline-Grafton H. Food Insecurity and Obesity: understanding the connections. 2011. http://frac.org/pdf/frac_brief_understanding_the_connections.pdf.
117. Seligman HK, Jacobs EA, Lopez A, Tschann J, Fernandez A. Food insecurity and glycemic control among low-income patients with type 2 diabetes. Diabetes Care. 2012;35(2):233-8. PMID: 22210570. http://www.ncbi.nlm.nih.gov/pubmed/22210570.
118. Laraia BA, Siega-Riz AM, Gundersen C. Household food insecurity is associated with self-reported pregravid weight status, gestational weight gain, and pregnancy complications. J Am Diet Assoc. 2010;110(5):692-701. PMID: 20430130. http://www.ncbi.nlm.nih.gov/pubmed/20430130.
119. Eisenmann JC, Gundersen C, Lohman BJ, Garasky S, Stewart SD. Is food insecurity related to overweight and obesity in children and adolescents? A summary of studies, 1995-2009. Obes Rev. 2011;12(5):e73-83. PMID: 21382151. http://www.ncbi.nlm.nih.gov/pubmed/21382151.
120. Hunger and obesity: understanding a food insecurity paradigm: workshop summary. 2011. http://www.nap.edu/openbook.php?record_id=13102.
121. Adam TC, Epel ES. Stress, eating and the reward system. Physiol Behav. 2007;91(4):449-58. PMID: 17543357. http://www.ncbi.nlm.nih.gov/pubmed/17543357.
122. Laraia BA. Food insecurity and chronic disease. Adv Nutr. 2013;4(2):203-12. PMID: 23493536. http://www.ncbi.nlm.nih.gov/pubmed/23493536.
123. Bhargava A, Jolliffe D, Howard LL. Socio-economic, behavioural and environmental factors predicted body weights and household food insecurity scores in the Early Childhood Longitudinal Study-Kindergarten. Br J Nutr. 2008;100(2):438-44. PMID: 18275621. http://www.ncbi.nlm.nih.gov/pubmed/18275621.

124. Dubois L, Farmer A, Girard M, Porcherie M. Family food insufficiency is related to overweight among preschoolers. Soc Sci Med. 2006;63(6):1503-16. PMID: 16777308. http://www.ncbi.nlm.nih.gov/pubmed/16777308.
125. Jyoti DF, Frongillo EA, Jones SJ. Food insecurity affects school children's academic performance, weight gain, and social skills. J Nutr. 2005;135(12):2831-9. PMID: 16317128. http://www.ncbi.nlm.nih.gov/pubmed/16317128.
126. Kim K, Frongillo EA. Participation in food assistance programs modifies the relation of food insecurity with weight and depression in elders. J Nutr. 2007;137(4):1005-10. PMID: 17374668. http://www.ncbi.nlm.nih.gov/pubmed/17374668.
127. Metallinos-Katsaras E, Must A, Gorman K. A longitudinal study of food insecurity on obesity in preschool children. J Acad Nutr Diet. 2012;112(12):1949-58. PMID: 23174682. http://www.ncbi.nlm.nih.gov/pubmed/23174682.
128. Olson CM, Strawderman MS. The relationship between food insecurity and obesity in rural childbearing women. J Rural Health. 2008;24(1):60-6. PMID: 18257872. http://www.ncbi.nlm.nih.gov/pubmed/18257872.
129. Rose D, Bodor JN. Household food insecurity and overweight status in young school children: results from the Early Childhood Longitudinal Study. Pediatrics. 2006;117(2):464-73. PMID: 16452367. http://www.ncbi.nlm.nih.gov/pubmed/16452367.
130. Whitaker RC, Sarin A. Change in food security status and change in weight are not associated in urban women with preschool children. J Nutr. 2007;137(9):2134-9. PMID: 17709454. http://www.ncbi.nlm.nih.gov/pubmed/17709454.
131. Weight gain during pregnancy. 2009. http://books.nap.edu/openbook.php?record_id=12584&page=R1.
132. Corral I, Landrine H. Acculturation and ethnic-minority health behavior: a test of the operant model. Health Psychol. 2008;27(6):737-45. PMID: 19025269. http://www.ncbi.nlm.nih.gov/pubmed/19025269.
133. Creighton MJ, Goldman N, Pebley AR, Chung CY. Durational and generational differences in Mexican immigrant obesity: is acculturation the explanation? Soc Sci Med. 2012;75(2):300-10. PMID: 22575698. http://www.ncbi.nlm.nih.gov/pubmed/22575698.
134. Dubowitz T, Subramanian SV, Acevedo-Garcia D, Osypuk TL, Peterson KE. Individual and neighborhood differences in diet among low-income foreign and U.S.-born women. Womens Health Issues. 2008;18(3):181-90. PMID: 18222706. http://www.ncbi.nlm.nih.gov/pubmed/18222706.
135. Erinosho TO, Berrigan D, Thompson FE, Moser RP, Nebeling LC, Yaroch AL. Dietary intakes of preschool-aged children in relation to caregivers' race/ethnicity, acculturation, and demographic characteristics: results from the 2007 California Health Interview Survey. Matern Child Health J. 2012;16(9):1844-53. PMID: 22160613. http://www.ncbi.nlm.nih.gov/pubmed/22160613.
136. Ghaddar S, Brown CJ, Pagan JA, Diaz V. Acculturation and healthy lifestyle habits among Hispanics in United States-Mexico border communities. Rev Panam Salud Publica. 2010;28(3):190-7. PMID: 20963266. http://www.ncbi.nlm.nih.gov/pubmed/20963266.
137. Gomez SL, Kelsey JL, Glaser SL, Lee MM, Sidney S. Immigration and acculturation in relation to health and health-related risk factors among specific Asian subgroups in a health maintenance organization. Am J Public Health. 2004;94(11):1977-84. PMID: 15514240. http://www.ncbi.nlm.nih.gov/pubmed/15514240.

138. Kasirye OC, Walsh JA, Romano PS, Beckett LA, Garcia JA, Elvine-Kreis B, et al. Acculturation and its association with health-risk behaviors in a rural Latina population. Ethn Dis. 2005;15(4):733-9. PMID: 16259501. http://www.ncbi.nlm.nih.gov/pubmed/16259501.
139. Matias SL, Stoecklin-Marois MT, Tancredi DJ, Schenker MB. Adherence to dietary recommendations is associated with acculturation among Latino farm workers. J Nutr. 2013;143(9):1451-8. PMID: 23864507. http://www.ncbi.nlm.nih.gov/pubmed/23864507.
140. Montez JK, Eschbach K. Country of birth and language are uniquely associated with intakes of fat, fiber, and fruits and vegetables among Mexican-American women in the United States. J Am Diet Assoc. 2008;108(3):473-80. PMID: 18313430. http://www.ncbi.nlm.nih.gov/pubmed/18313430.
141. Parikh NS, Fahs MC, Shelley D, Yerneni R. Health behaviors of older Chinese adults living in New York City. J Community Health. 2009;34(1):6-15. PMID: 18830807. http://www.ncbi.nlm.nih.gov/pubmed/18830807.
142. Shin CN, Lach H. Nutritional issues of Korean Americans. Clin Nurs Res. 2011;20(2):162-80. PMID: 21160079. http://www.ncbi.nlm.nih.gov/pubmed/21160079.
143. Song YJ, Hofstetter CR, Hovell MF, Paik HY, Park HR, Lee J, et al. Acculturation and health risk behaviors among Californians of Korean descent. Prev Med. 2004;39(1):147-56. PMID: 15207996. http://www.ncbi.nlm.nih.gov/pubmed/15207996.
144. van Rompay MI, McKeown NM, Castaneda-Sceppa C, Falcon LM, Ordovas JM, Tucker KL. Acculturation and sociocultural influences on dietary intake and health status among Puerto Rican adults in Massachusetts. J Acad Nutr Diet. 2012;112(1):64-74. PMID: 22389874. http://www.ncbi.nlm.nih.gov/pubmed/22389874.
145. Van Wieren AJ, Roberts MB, Arellano N, Feller ER, Diaz JA. Acculturation and cardiovascular behaviors among Latinos in California by country/region of origin. J Immigr Minor Health. 2011;13(6):975-81. PMID: 21626297. http://www.ncbi.nlm.nih.gov/pubmed/21626297.
146. Wang S, Quan J, Kanaya AM, Fernandez A. Asian Americans and obesity in California: a protective effect of biculturalism. J Immigr Minor Health. 2011;13(2):276-83. PMID: 21153765. http://www.ncbi.nlm.nih.gov/pubmed/21153765.
147. Novotny R, Chen C, Williams AE, Albright CL, Nigg CR, Oshiro CE, et al. US acculturation is associated with health behaviors and obesity, but not their change, with a hotel-based intervention among Asian-Pacific Islanders. J Acad Nutr Diet. 2012;112(5):649-56. PMID: 22709769. http://www.ncbi.nlm.nih.gov/pubmed/22709769.
148. Unger JB, Reynolds K, Shakib S, Spruijt-Metz D, Sun P, Johnson CA. Acculturation, physical activity, and fast-food consumption among Asian-American and Hispanic adolescents. J Community Health. 2004;29(6):467-81. PMID: 15587346. http://www.ncbi.nlm.nih.gov/pubmed/15587346.
149. Ahluwalia IB, Ford ES, Link M, Bolen JC. Acculturation, weight, and weight-related behaviors among Mexican Americans in the United States. Ethn Dis. 2007;17(4):643-9. PMID: 18072373. http://www.ncbi.nlm.nih.gov/pubmed/18072373.
150. Chen L, Juon HS, Lee S. Acculturation and BMI among Chinese, Korean and Vietnamese adults. J Community Health. 2012;37(3):539-46. PMID: 21922164. http://www.ncbi.nlm.nih.gov/pubmed/21922164.
151. Fu H, VanLandingham MJ. Disentangling the effects of migration, selection and acculturation on weight and body fat distribution: results from a natural experiment

involving Vietnamese Americans, returnees, and never-leavers. J Immigr Minor Health. 2012;14(5):786-96. PMID: 22427069. http://www.ncbi.nlm.nih.gov/pubmed/22427069.

152. Garcia L, Gold EB, Wang L, Yang X, Mao M, Schwartz AV. The relation of acculturation to overweight, obesity, pre-diabetes and diabetes among U.S. Mexican-American women and men. Ethn Dis. 2012;22(1):58-64. PMID: 22774310. http://www.ncbi.nlm.nih.gov/pubmed/22774310.

153. Hubert HB, Snider J, Winkleby MA. Health status, health behaviors, and acculturation factors associated with overweight and obesity in Latinos from a community and agricultural labor camp survey. Prev Med. 2005;40(6):642-51. PMID: 15850860. http://www.ncbi.nlm.nih.gov/pubmed/15850860.

154. Koya DL, Egede LE. Association between length of residence and cardiovascular disease risk factors among an ethnically diverse group of United States immigrants. J Gen Intern Med. 2007;22(6):841-6. PMID: 17503110. http://www.ncbi.nlm.nih.gov/pubmed/17503110.

155. Daviglus M, Talavera G, Avilés-Santa M, Allison M, Cai J, Criqui M, et al. Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States. JAMA. 2012;308(17):1775-84. PMID: WOS:000310726500024. http://www.ncbi.nlm.nih.gov/pubmed/23117778.

156. Eamranond PP, Legedza AT, Diez-Roux AV, Kandula NR, Palmas W, Siscovick DS, et al. Association between language and risk factor levels among Hispanic adults with hypertension, hypercholesterolemia, or diabetes. Am Heart J. 2009;157(1):53-9. PMID: 19081396. http://www.ncbi.nlm.nih.gov/pubmed/19081396.

157. Eamranond PP, Wee CC, Legedza AT, Marcantonio ER, Leveille SG. Acculturation and cardiovascular risk factor control among Hispanic adults in the United States. Public Health Rep. 2009;124(6):818-24. PMID: 19894424. http://www.ncbi.nlm.nih.gov/pubmed/19894424.

158. Kershaw KN, Greenlund KJ, Stamler J, Shay CM, Daviglus ML. Understanding ethnic and nativity-related differences in low cardiovascular risk status among Mexican-Americans and non-Hispanic Whites. Prev Med. 2012;55(6):597-602. PMID: 23036519. http://www.ncbi.nlm.nih.gov/pubmed/23036519.

159. Kandula NR, Diez-Roux AV, Chan C, Daviglus ML, Jackson SA, Ni H, et al. Association of acculturation levels and prevalence of diabetes in the multi-ethnic study of atherosclerosis (MESA). Diabetes Care. 2008;31(8):1621-8. PMID: 18458142. http://www.ncbi.nlm.nih.gov/pubmed/18458142.

160. Mainous AG, 3rd, Majeed A, Koopman RJ, Baker R, Everett CJ, Tilley BC, et al. Acculturation and diabetes among Hispanics: evidence from the 1999-2002 National Health and Nutrition Examination Survey. Public Health Rep. 2006;121(1):60-6. PMID: 16416699. http://www.ncbi.nlm.nih.gov/pubmed/16416699.

Part D. Chapter 4: Food Environment and Settings

INTRODUCTION

Few American children, adolescents or adults have dietary patterns that are consistent with the Dietary Guidelines for Americans. The reasons for this are numerous, as what people eat is influenced by many complex factors, as discussed in ***Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence***. These factors span from individual levels of influence to dimensions of our environment. Improving dietary and lifestyle patterns and reducing diet-related chronic diseases, including obesity, will require actions at the individual behavioral and population and environmental levels. Behavioral strategies are needed to motivate and enhance the capacity of the individual to adopt and improve their lifestyle behaviors. Specific behavioral efforts related to eating and food[♦] and beverage choices include improving knowledge, attitudes, motivations, and food and cooking skills. Environmental change also is important because the environmental context and conditions affect what and how much people eat and what food choices are available. In addition, actions are needed to address the disparity gaps that currently exist in availability and access to healthy foods in low-income and rural communities.

Health and optimal nutrition and weight management cannot be achieved without a focus on the synergistic linkages and interactions between individuals and their environments, and understanding the different domains of food-related environmental influences. The social environment includes social networks and support systems, such as those provided by family, friends, and community cohesion. The physical environment includes the multiple settings where people obtain and consume food, such as their homes, work places, schools, restaurants, and grocery stores. The macro-environment operates within the broader society and includes food marketing, economic and price structures, food production and distribution systems, transportation, and agricultural practices and policies. Collectively, these environments influence what food choices we make, and where and how much we eat. Although personal responsibility is important, food choices are intertwined with and dependent on the community and environment context.

Interest is growing in the role of the environment in promoting or hindering healthy eating. Although it is up to individuals to decide what and how much they eat and drink, individual behavior to make healthy choices is enhanced when there is a supportive environment with accessible and affordable healthy choices. Thus, individual change is more likely to be facilitated and sustained if the environments within which food choices are made supports healthful options. As with other major public health issues, such as smoking reduction, injury prevention,

[♦] Note: Throughout this chapter, references to "foods" should be taken to mean "foods and beverages."

and infectious disease prevention, greater success at the individual and population levels for reducing obesity and diet-related chronic diseases are not as likely to occur unless environmental influences are identified and modified.

Meaningful solutions to improve diet and health cannot only be focused just on individuals, or families but must take into account the need for environmental and policy change. Environmental and policy changes can have a sustaining effect on individual behavior change because they can become incorporated into organizational structures and systems, and lead to alterations in sociocultural and societal norms. Both policy and environmental changes also can help reduce disparities by improving access to and availability of healthy food in underserved neighborhoods and communities. Federal nutrition assistance programs, in particular, play a vital role in achieving this objective through access to affordable foods that help millions of Americans meet Dietary Guidelines recommendations.

The Nation's ultimate goal should be neighborhoods and communities where healthy, affordable food and beverages are available to everyone in the United States in multiple settings, where healthy foods rather than unhealthy foods are the likely choice (optimal default), where social norms embrace and support healthy eating, and where children grow up enjoying the taste of vegetables, fruits, whole grains, and nonfat or low-fat dairy products and water instead of energy-dense foods with low nutrient density and that are high in refined grains, saturated fats, sodium, and added sugars. So too, it is important that these behaviors can be sustained throughout the lifespan and in settings where adults and older adult populations work or are served and reside.

The questions asked and reviewed in this chapter address place-based environments that influence the foods that individuals, families and households obtain and consume, and on the community settings in which they spend much of their time. The DGAC considered several settings but prioritized four key settings to examine for this report: neighborhood and community food access; child care (early care and education); schools; and worksites. The Committee examined the relationship of these settings to diet quality and weight status. Because of the need to identify effective population-level strategies, the Committee focused specifically on reviewing the scientific literature to determine the impact of place-based obesity prevention and dietary interventions. Because of time demands, the Committee could not address other important settings, such as after-school settings, recreational settings, and faith-based institutions, as well as more macro-environmental influences such as food marketing and economic impacts. Despite the lack of time to examine these settings, the DGAC considers them to be very important environmental influencers on dietary intake.

LIST OF QUESTIONS

Food Access

1. What is the relationship between neighborhood and community access to food retail settings and individuals' dietary intake and quality?
2. What is the relationship between neighborhood and community access to food retail settings and weight status?

Early Care and Education

3. What is the impact of obesity prevention approaches in early care and education programs on the weight status of children ages 2 to 5 years?

Schools

4. What is the impact of school-based approaches on the dietary intake, quality, behaviors, and/or preference of school-aged children?
5. What is the impact of school-based policies on the dietary intake, quality, behaviors, and/or preferences of school-aged children?
6. What is the impact of school-based approaches on the weight status of school-aged children?
7. What is the impact of school-based policies on the weight status of school-aged children?

Worksite

8. What is the impact of worksite-based approaches on the dietary intake, quality, behaviors and/or preferences of employees?
9. What is the impact of worksite policies on the dietary intake, quality, behaviors and/or preferences of employees?
10. What is the impact of worksite-based approaches on the weight status of employees?
11. What is the impact of worksite policies on weight status of employees?

METHODOLOGY

Questions related to food access were answered using Nutrition Evidence Library (NEL) systematic reviews, while questions related to schools and worksites were answered using existing systematic reviews. The early care and education question was answered using an existing systematic review with a NEL systematic review update. Descriptions of the NEL process and the use of existing systematic reviews are provided in ***Part C: Methodology***. All NEL reviews were conducted in accordance with NEL methodology, and the DGAC made all substantive decisions required throughout the process to ensure that the most complete and relevant body of evidence was identified and evaluated to answer each question. All steps in the process were documented to ensure transparency and reproducibility. Specific information about individual systematic reviews can be found at www.NEL.gov, including the search strategy, inclusion and exclusion criteria, a complete list of included and excluded articles, and a detailed write-up describing the included studies and the body of evidence. Specific information about the use of existing systematic reviews, including the search strategy, inclusion and exclusion criteria, and a detailed write-up describing the included studies and the body of evidence can be found at www.DietaryGuidelines.gov. A link for each question is provided following each evidence review.

FOOD ACCESS

Understanding how access to nutritious and affordable food at various retail establishments--from convenience stores, to farmers markets, to large box stores--support individuals in their consumption of a high quality diet and ability to achieve a healthy body weight was the focus of the food access questions. Because the two food access questions are complementary, the DGAC choose to develop only one implication statement for both questions.

Question 1: What is the relationship between neighborhood and community access to food retail settings and individuals' dietary intake and quality?

Source of evidence: NEL systematic review

Conclusion

Emerging evidence suggests that the relationship between access to farmers' markets/produce stands and dietary intake and quality is favorable. The body of evidence regarding access to other food outlets, such as supermarkets, grocery stores, and convenience/corner stores, and dietary intake and quality is limited and inconsistent. **DGAC Grade: Grade not assignable**

Review of the Evidence

This systematic review included 18 studies published between 2007 and 2013, including 15 cross-sectional studies,[1-15] by independent investigators with sufficient sample sizes, 1 longitudinal study[16] and 2 controlled trials[17, 18] (one RCT and one non-randomized) examining the relationship between food access and dietary intake and/or quality.

The studies used multiple approaches to assess food access and dietary intake, quality, and variety. The majority of studies measured food access by the density of food outlets within a specified distance from a participant's residence and/or proximity to various food outlets. The majority of studies assessed dietary intake by focusing on vegetable and fruit consumption; diet quality and variety were predominantly determined by various validated diet indices including, but not limited to, the Healthy Eating Index (HEI).

Although food access was assessed across wide-ranging geographic, ethnic, racial, and income groups, due to the wide variation in methods used to determine food access, making comparisons across studies was challenging. Despite this variability, a consistent relationship was identified between farmers' markets/produce stands and dietary intake.[6, 15] Two cross-sectional studies found statistically significant, favorable associations between access to farmers' markets/produce stands and dietary intake (assessed by individual vegetable and fruit consumption) and diet variety and quality (both assessed by the HEI). Due to the variability of studies and paucity of data, no consistent associations regarding dietary outcomes and access to other food outlets were evident.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250425

Question 2: What is the relationship between neighborhood and community access to food retail settings and weight status?

Source of evidence: NEL systematic review

Conclusion

Limited but consistent evidence suggests that the relationship between access to convenience stores and weight status is unfavorable, with closer proximity and greater access being associated with significantly higher body mass index (BMI) and/or increased odds of overweight or obesity.
DGAC Grade: Limited

The body of evidence on access to other food outlets, such as supermarkets, grocery stores, and farmers' markets/produce stands, and weight status is limited and inconsistent. **DGAC Grade: Grade not assignable**

Review of the Evidence

This systematic review included 26 studies published between 2005 and 2013, including 19 cross-sectional studies[1, 6, 8, 14, 19-33] and 7 longitudinal studies[34-40] examining the relationship between food access and weight status.

The studies used multiple approaches to assess food access and measures of weight status. The majority of studies measured food access by the density of food outlets within a specified distance from a participant's residence and/or proximity to various food outlets. The primary weight status outcome was BMI, which was derived from height and weight.

Due to the wide variation in methods used to determine food access, making comparison across studies was challenging. Despite this variability, the relationship between convenience stores and weight status was consistent across the evidence. Seven studies[19, 23, 24, 26-28, 37] (six cross-sectional and one longitudinal) found statistically significant associations between access to convenience stores and BMI and/or increased odds of overweight or obesity. Five of these studies were completed in an adult sample; two assessed this relationship among children. Due to the variability of studies and paucity of data, no consistent associations regarding weight status and access to other food outlets were evident.

The evidence base included several studies of weaker design, mostly cross-sectional, by independent investigators with sufficient sample sizes. The findings across studies were inconsistent for all food outlet types, except for convenience stores, which were evaluated in only seven studies. Although food access was assessed across geographic, ethnic, racial and income groups, the variability in methodology made it difficult to compare studies.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250459

Implications for the Food Access Topic Area

For people to improve their diets and health, they need to have access to high quality and affordable healthy foods in environments where they live, work, learn, and/or play across the lifespan. Limited access to affordable and healthy food is a challenge, particularly for families living in rural areas and low-income communities. Innovative approaches to bring healthy food retail options into communities have proliferated, especially in underserved areas. These include creating financing programs to incentivize grocery store development; improving availability of healthy food at corner stores and bodegas, farmers markets and mobile markets, shelters, food banks, community gardens/cooperatives, and youth-focused gardens; and creating new forms of wholesale distribution through food hubs. However, most of these approaches lack adequate evaluation. These and other promising equity-oriented efforts need to continue and be evaluated and then successfully scaled up to other communities.

To ensure healthy food access to everyone in the United States, action is needed across all levels—Federal, state, and local—to create private-public partnerships and business models, with the highest priority on those places with greatest need. Similar efforts are needed to reduce access to, and consumption of, calorie-dense, nutrient-poor foods and sugar-sweetened beverages in community settings. These efforts need to be seamlessly integrated with food assistance programs, such as food banks, soup kitchens, and Federal nutrition assistance programs, such as the Special Supplemental Program for Women, Infants and Children (WIC) and the Supplemental Nutrition Assistance Program (SNAP) and elder nutrition.

EARLY CARE AND EDUCATION

About one in five preschool children are overweight or obese,[41] and growing evidence indicates that preschoolers who are overweight or obese experience negative physical consequences, including cardio-metabolic abnormalities,[42] making evident the need for effective efforts to prevent excessive weight gain for this age group.

Question 3: What is the impact of obesity prevention approaches in early care and education programs on the weight status of children ages 2 to 5 years?

Source of evidence: Existing systematic review with a NEL systematic review update

Conclusion

Moderate evidence suggests that multi-component obesity prevention approaches implemented in child care settings improve weight-related outcomes in preschoolers. A combination of dietary and physical activity interventions is effective for preventing or slowing excess weight gain and reducing the proportion of young children ages 2 to 5 years who become overweight or obese.
DGAC Grade: Moderate

Implications

Existing evidence indicates that multi-component interventions that incorporate both nutrition and physical activity are effective in reducing excessive weight gain in preschool children. Successful strategies include: curricular enhancements of classroom education for children on both nutrition education and physical activity, outreach engagement to parents about making positive changes in the home, improvements in the nutrition quality of meals and snacks served in the child care program, modifying food service practices, improving the mealtime environment, increasing physical activity play, reducing sedentary behaviors, and improving outdoor playground environments. Evidenced-based healthy eating and physical activity practices should be implemented in child care settings with training and technical assistance for staff. At the Federal, state, and local levels, policies are needed that create strong nutrition and physical activity standards and guidelines in child care settings. There is a need to strengthen policies at the Federal, state, and local levels for strong nutrition and physical activity standards and guidelines in child care settings.

It is important that child care facilities provide meals and snacks that are consistent with the meal patterns in the Federal Child and Adult Care Food Program (CACFP)[43] to ensure that young children have access to healthy meals and snacks and age-appropriate portions. Drinking water also needs to be readily available and accessible to children. Government agencies should ensure access to affordable, nutritious foods through CACFP and maximize participation in the program.

Review of the Evidence

This evidence portfolio included one existing systematic review from Zhou et al.[44] and a de novo NEL systematic review updating the evidence base. The Zhou et al. review included 15 controlled trials published between 2000 and 2012; the NEL review included seven studies[45-52] (eight publications) published between 2012 and 2014. Both reviews examined the impact of obesity prevention approaches on the weight status of children ages 2 to 5 years.

The studies used a variety of intervention strategies targeting behaviors that affect body weight. Most approaches were multi-component, with a combination of interventions targeting children, their parents, and/or staff of early care and education programs. The primary weight status outcomes of interest were BMI and BMI z-score.

The body of available evidence describes a large variation in excessive weight gain prevention approaches, making comparison across studies challenging. Despite this variability, multi-component interventions were effective in reducing BMI and preventing excess weight gain. Seven of 10 multi-component studies included in the Zhou et al. review demonstrated improvements in weight-related outcomes. Six of the seven interventions included in the NEL review demonstrated that multi-component interventions effectively reduce BMI or prevent excess weight gain in children ages 2 to 5 years.

The evidence base included several studies of strong design by independent investigators, specifically controlled trials, with sufficient sample sizes. Some inconsistency was evident across studies and may be explained by differences in the populations sampled, outcome measures, duration or exposure of intervention, and follow-up periods. Although the majority of the studies included in the evidence portfolio effectively reduced BMI or prevented excess weight gain, the magnitude of the effect as well as the clinical and public health significance was difficult to assess because of the differences in measures and methodology.

For additional details on this body of evidence, visit: http://NEL.gov/topic.cfm?cat=3355

SCHOOLS

There are 49.6 million children aged 6-17 years in the United States, and the vast majority are educated in public or private school settings. School-based programs and policies at the local, state, and federal levels are cornerstones of food accessibility, availability, and consumption at schools, which underscore why this setting is a major determinant of nutritional intake and growth, development, and health of school-aged children. Because the schools questions are complementary, the DGAC choose to develop only one implication statement for the four questions.

Question 4: What is the impact of school-based approaches on the dietary intake, quality, behaviors, and/or preferences of school-aged children?

Source of evidence: Existing systematic reviews

Conclusion

Moderate evidence indicates that multi-component school-based approaches can increase daily vegetable and fruit consumption in children in grades kindergarten through 8th. Sufficient school-based studies have not been conducted with youth in grades 9 to 12. Vegetable and fruit consumption individually, as well as in combination, can be targeted with specific school-based approaches. **DGAC Grade: Moderate**

Review of the Evidence

This evidence portfolio included three systematic reviews;[53-55] two of which included meta-analyses,[53, 55] which collectively evaluated 75 studies published between 1985 and 2011. Forty-nine studies were conducted in the United States and the remaining studies were completed in other highly developed countries. The systematic reviews examined the impact of school-based approaches targeting the dietary intake, quality, behaviors and/or preferences of school-aged children.

The studies used a variety of intervention strategies. Some approaches were multi-component, with a combination of interventions targeting children, their parents, and/or the school environment. The primary dietary outcome of interest was vegetable and fruit intake.

In the body of available evidence, the school-based approaches were diverse, making comparison across studies challenging. Despite this variability, multi-component interventions, and in particular those that engaged both children and their families, were more effective than single-component interventions for eliciting significant dietary improvements. Broadly, school-based intervention programs moderately increased total daily vegetable and fruit intakes and fruit (with and without fruit juice) intake alone. Furthermore, results showed that school-based economic incentive programs can effectively increase vegetable and fruit consumption and reduce consumption of low-nutrient-dense foods while children are at school. Nutrition education programs that include gardening effectively increased the consumption of vegetables in school-aged children, along with small, but significant increases in fruit intake.

The evidence base included three reviews evaluating several studies by independent investigators with sufficient sample sizes. Some inconsistency was evident across studies and may be explained by differences in the populations sampled, outcome measures, duration or exposure of intervention and follow-up periods. Although findings indicated that school-based approaches effectively increased the combined intake of vegetable and fruit, the magnitude of the effect as well as the public health significance was difficult to assess because of differences in measures and methodology.

For additional details on this body of evidence, visit: Appendix E-2.29a and Appendix E-2.29b

Question 5: What is the impact of school-based policies on the dietary intake, quality, behaviors, and/or preferences of school-aged children?

Source of evidence: Existing systematic reviews

Conclusion

Strong evidence demonstrates that implementing school policies for nutrition standards to improve the availability, accessibility, and consumption of healthy foods and beverages sold outside the school meal programs (competitive foods and beverages) and (or) reducing or eliminating unhealthy foods and beverages are associated with improved purchasing behavior and result in higher quality dietary intake by children while at school. **DGAC Grade: Strong**

Review of the Evidence

This evidence portfolio includes two systematic reviews,[54, 56] which collectively evaluated 52 studies published between 1990 and 2013. Forty-one studies were conducted in the United States and the remaining studies were conducted in other highly-developed countries. The systematic reviews examined the impact of school policies, at the state and district levels, on dietary intake and behaviors.

The studies included a variety of policies, including economic incentives and both state and school-district policies, targeting behaviors related to dietary intake. The primary outcomes of interest were vegetable and fruit intakes and availability, purchasing, and consumption of competitive foods and beverages (CF&B).

In the body of available evidence, school policies were diverse, making comparison across studies challenging. Despite this variability, school-based policies targeting the availability of foods and beverages can positively influence the behaviors related to nutrition among children while they are at school. School-based economic incentive programs can effectively increase vegetable and fruit consumption and reduce consumption of low-nutrient-dense foods while children are at school. The implementation of school policies to change the availability and accessibility of healthier foods and beverages versus unhealthy CF&B is associated with the expected changes in consumption within the school setting. In addition, strong and consistent enforcement of more comprehensive policies to change the availability of healthier foods and beverages versus unhealthy CF&B at schools is associated with desired changes in consumption and purchasing within the school setting. Also, policies restricting the use of food as a reward for academic performance or as part of a fundraiser were associated with a reduction in using foods and beverages for these purposes.

The evidence base included two reviews evaluating several studies by independent investigators with sufficient sample sizes. Although findings indicated that school policies can effectively increase the combined intake of vegetables and fruits and/or decrease the availability, purchasing, and consumption of unhealthy CF&B, the magnitude of the effect as well as the public health significance is difficult to ascertain.

For additional details on this body of evidence, visit: Appendix E-2.30 and Appendix E-2.29b

Question 6: What is the impact of school-based approaches on the weight status of school-aged children?

Source of evidence: Existing systematic reviews

Conclusion

Moderate and generally consistent evidence indicates that multi-component school-based approaches have beneficial effects on weight status (BMI or BMI-z reduced on average by 0.15 kg/m2), especially for children ages 6 to 12 years. **DGAC Grade: Moderate**

The body of evidence regarding the impact of school-based approaches on weight status among adolescents is limited due to an insufficient number of studies. **DGAC Grade: Not Assignable**

Review of the Evidence

This evidence portfolio included two systematic reviews;[57, 58] one of which included a meta-analysis.[57] Collectively, 108 studies targeting children in school published before August 2012 were evaluated. Forty-nine studies were conducted in the United States and the remaining studies were completed in other highly developed countries. The systematic reviews examined the impact of school-based approaches targeting obesity prevention among school-aged children.

The studies used a variety of intervention strategies targeting behaviors related to dietary intake and/or physical activity. Some approaches were multi-component, with a combination of interventions targeting children, their parents, and/or the school environment. The primary outcomes of interest were BMI, changes in BMI, rate of weight gain, body fat percentage, waist circumference, skin fold thickness, and prevalence of overweight and obesity.

In the body of available evidence, the school-based approaches were diverse, making comparison across studies challenging. Despite this variability, school-based interventions significantly improved weight-related outcomes. Multi-component interventions, and in particular those implemented longer term (more than 6 months), were more effective than single-component and short-term (3 to 6 months) interventions. Evidence supporting the effectiveness of school-based interventions among children ages 6 to 12 years was robust, while findings among adolescents ages 13 to 18 years were weaker, but trended toward effectiveness.

The evidence base included two reviews evaluating several studies by independent investigators with sufficient sample sizes. Although findings indicated that school-based approaches effectively improve weight-related outcomes, in particular among children between the ages of 6 and 12 years, a high degree of heterogeneity means these findings should be interpreted cautiously. Although the magnitude of the effect was clinically meaningful, the public health significance was difficult to ascertain.

For additional details on this body of evidence, visit: Appendix E-2.31 and Appendix 2.29b

Question 7: What is the impact of school-based policies on the weight status of school-aged children?

Source of evidence: Existing systematic reviews

Conclusion

Although moderate evidence indicates that school policies improve dietary intake, limited evidence suggests that school policies targeting nutrition, alone and in combination with physical activity, may beneficially affect weight-related outcomes. **DGAC Grade: Limited**

Review of the Evidence

This evidence portfolio included two systematic reviews,[56, 59] which collectively evaluated 45 studies published between 2003 and 2013. Forty studies were conducted in the United States and the remaining studies were conducted in other highly developed countries. The systematic reviews examined the impact of school policies, at the state and district levels, on weight-related outcomes.

The studies included a variety of policies at the school, school-district, or state level, targeting behaviors related to dietary intake, alone and in combination with physical activity. The primary outcome of interest was BMI.

Limited research exists to systematically review and quantitatively evaluate the effect of school-based nutrition policies on the weight status of children. In addition, high heterogeneity among studies warrants caution when drawing conclusions from the results. In the body of available evidence, the findings related to the impact of school policies targeting nutrition and physical activity on weight outcomes were mixed. Even so, dietary policies related to the School Breakfast Program were associated with a lower BMI among students who participated in the program in comparison to students who did not participate. Overall, school-based, multi-component interventions including policy elements and policies and laws regarding the availability and accessibility of CF&B in schools warrant further research as ways to target childhood obesity.

The evidence base included two reviews evaluating several studies by independent investigators with sufficient sample sizes. However, most studies were of weaker design (i.e., cross-sectional) and findings were inconsistent.

For additional details on this body of evidence, visit: Appendix E-2.32 and Appendix E-2.29b

Implications for the Schools Topic Area

Existing evidence indicates that school-based programs designed to improve the food environment and support healthy behaviors may effectively promote improved dietary intake and weight status of school-aged children. Programs that emphasize multi-component, multi-dimensional approaches (including increased physical activity) are important to changing behavior and need to be reinforced within the home environment, as well as the community, including neighborhood food retail outlets that surround schools. Policies should strive to support effective programs that increase availability, accessibility, and consumption of healthy foods, while reducing less healthy CF&B. The combination of economic incentives along with specific policies can increase the likelihood that specific approaches will be effective.

The recently updated USDA nutrition standards for school meals, snacks, and beverages sold in schools will ensure that students throughout the United States will have healthier school meals and snack and beverage options, but schools need support and active engagement from students, parents, teachers, administrators, community members, and their districts and states to successfully implement and sustain them.

WORKSITES

Many workplaces are located in areas where food options are limited, which makes the workplace an important setting for approaches focused on dietary intake and environmental modifications. Because the worksite questions are complementary, the DGAC choose to develop only one implication statement for the four questions.

Question 8: What is the impact of worksite-based approaches on the dietary intake, quality, behaviors and/or preferences of employees?

Source of evidence: Existing systematic reviews

Conclusion

Moderate evidence indicates that multi-component worksite approaches can increase vegetable and fruit consumption of employees. **DGAC Grade: Moderate**

Review of the Evidence

This evidence portfolio includes two systematic reviews,[60, 61] which collectively evaluated 35 studies by independent investigators with sufficient sample sizes published before November 2012. The systematic reviews examined the impact of worksite-based approaches targeting the dietary intake, quality, behaviors, and/or preferences of employees.

The studies used a variety of intervention approaches targeting behaviors related to dietary intake; some were delivered in-person and others were delivered through the Internet. Some inconsistencies are evident across studies and may be explained by differences in the populations sampled and methodologies used, including the types and durations of intervention and follow-up periods. Some approaches were multi-component, with a combination of interventions targeting employees and/or the food environment at the worksite. The primary dietary outcome of interest was vegetable and fruit intake.

Among the body of evidence available, multi-component interventions, and in particular those that incorporated face-to-face contact and nutrition education, were more effective than single-component interventions for eliciting significant dietary improvements. Overall, worksite-based intervention programs moderately increase vegetable and fruit intakes, although the magnitude of the effect is difficult to assess. Nutrition education and internet-based programs appear to be promising approaches for eliciting desired dietary modifications when incorporated into multi-component interventions.

For additional details on this body of evidence, visit: Appendix E-2.33a and Appendix E-2.33b

Question 9: What is the impact of worksite-based policies on the dietary intake, quality, behaviors and/or preferences of employees?

Source of evidence: Existing systematic reviews

Conclusion

Moderate and consistent evidence indicates that worksite nutrition policies, alone and in combination with environmental changes and/or individual-level nutrition and health improvement strategies, can improve the dietary intake of employees. Multi-component interventions appear to be more effective than single-component interventions. **DGAC Grade: Moderate**

Review of the Evidence

This evidence portfolio includes one systematic review,[62] which evaluated 27 studies by independent investigators with sufficient sample sizes published between 1985 and 2010. The review examined the evidence for the effectiveness of a variety of worksite health promotion programs using environmental and/or policy changes either alone or in combination with health behavior change strategies focused on individual employees.

Some interventions were multi-component, with a combination of strategies targeting employees and/or the food environment at the worksite. Strategies included point-of-purchase labeling, increased availability of healthy food items, and/or educational programs and materials. The primary dietary outcome of interest was vegetable and fruit intake.

In the body of evidence available, the worksite-based policies were diverse, thus it was challenging to identify the most effective strategies. Despite this variability, multi-component interventions, and in particular those that targeted individual employees in addition to the environment, were more effective than single-component interventions for eliciting significant dietary improvements. Overall, worksite interventions moderately increased vegetable and fruit intakes.

Some inconsistency was evident across studies assessed for the systematic review in regards to scientific rigor and impact. The inconsistencies may be explained by differences in the populations sampled and methodologies used, including duration, exposure of the intervention, and follow-up periods. Although findings indicate that worksite policies increase consumption of vegetables and fruit, the magnitude of the effect was difficult to assess.

For additional details on this body of evidence, visit: Appendix E-2.34 and Appendix E-2.33b

Question 10: What is the impact of worksite-based approaches on the weight status of employees?

Source of evidence: Existing systematic reviews

Conclusion

Moderate and consistent evidence indicates that multi-component worksite approaches targeting physical activity and dietary behaviors favorably affect weight-related outcomes. **DGAC Grade: Moderate**

Review of the Evidence

This evidence portfolio includes two systematic reviews,[61, 63] one of which included meta-analyses.[63] The systematic reviews examined the impact of worksite-based approaches on the weight status of employees. Collectively, 70 studies published before November 2012 were evaluated.

The studies used a variety of intervention strategies targeting behaviors related to weight status; some were delivered in-person and others were delivered through the Internet. The primary outcomes of interest were body weight, BMI, and body fat percentage.

In the body of evidence available, multi-component interventions, and in particular those that incorporated face-to-face contact and targeted behaviors related to diet and physical activity, were more effective than single-component interventions for eliciting significant improvements in weight-related outcomes. Overall, worksite-based intervention programs significantly decreased body weight, BMI, and body fat percentage. Internet-based programs appeared to be promising approaches for eliciting behavior changes and improving related health outcomes.

The evidence base included two reviews evaluating several studies by independent investigators with sufficient sample sizes. Some inconsistencies were evident across studies and may be explained by differences in the populations sampled and methodologies, including duration or exposure of intervention and follow-up periods. Although findings indicated that worksite-based approaches effectively improve the weight status of employees, the magnitude of the effect was difficult to assess.

For additional details on this body of evidence, visit: Appendix E-2.35 and Appendix E-2.33b

Question 11: What is the impact of worksite-based policies on the weight status of employees?

Source of evidence: Existing systematic reviews

Conclusion

The body of evidence assessing the impact of worksite policies on the weight status of employees is very limited. **DGAC Grade: Not Assignable**

Review of the Evidence

This evidence portfolio included one systematic review,[62] which evaluated 27 studies published between 1985 and 2010. The review examined the evidence for the effectiveness of worksite health promotion programs using environmental and/or policy changes either alone or in combination with individually-focused health behavior change strategies.

The studies used a variety of policies targeting behaviors that can influence weight status. Some studies assessed the impact of policies (e.g., catering policies and company policies rewarding employees for healthy behaviors) combined with individual-level strategies. Some interventions were multi-component, with a combination of strategies targeting employees (e.g., point-of-choice messaging including nutrition information in cafeterias and reminders to use stairs) and/or the food environment at the worksite (e.g., increased availability of healthy food options). The health outcomes of interest included BMI, blood pressure, and cholesterol.

In the body of evidence available, worksite policies either alone or in combination with individually-focused health behavior change strategies did not affect the weight status of employees. However, interventions incorporating both environmental and individual strategies can lead to significant improvement in behaviors related to weight status (e.g., dietary intake). The lack of impact may be due to length of exposure or the duration of the follow-up period.

The evidence base included one review evaluating several studies by independent investigators with sufficient sample sizes. The studies were inconsistent in their scientific rigor. Due to the variability of studies and paucity of data, no consistent associations regarding worksite policies and the weight status of employees were evident.

For additional details on this body of evidence, visit: Appendix E-2.36 and Appendix E-2.33b

Implications for the Worksite Topic Area

Existing evidence indicates that worksite approaches focused on dietary intake can increase fruit and vegetable intakes of employees. Multi-component programs targeting nutrition education in combination with dietary modification interventions are found to be effective. Additionally, environmental modifications in conjunction with a variety of worksite policies targeting dietary modification, including point-of-purchase information, catering policies, and menu labeling are effective. Thus, these evidence-based strategies should be implemented in worksites through a variety of means, such as corporate wellness programs, food service policies, and health benefits programs. Programs should emphasize multi-component approaches targeting diet and physical activity while policies should support behavior changes associated with improving health outcomes such as increasing the availability of healthy foods within the workplace and encouraging more physical activity throughout the workday. Given that approximately 64 percent of adults are employed and spend an average of 34 hours per week at work, the workplace remains an important setting for environmental and behavioral interventions for health promotion and disease prevention.

CHAPTER SUMMARY

Environmental and policy approaches are needed to complement individual-based efforts to improve diet quality and reduce obesity and other diet-related chronic diseases. These approaches have the potential for broad and sustained impact at the population level. The DGAC focused on physical environments (settings) in which foods are available. Our aim was to better understand the impact of the food environment to promote or hinder diet quality healthy eating in these settings and to identify the most effective evidence-based diet-related approaches and policies to improve diet quality and weight status. The DGAC systematically reviewed and graded the scientific evidence in these four settings, community food access, child care, schools and worksites, and their relationships to dietary quality and weight status.

The DGAC found moderate and promising evidence that multi-component obesity prevention approaches implemented in child care settings, schools, and worksites improve weight-related outcomes; strong to moderate evidence that school and worksite policies are associated with improved dietary intake; and moderate evidence that multi-component school-based and worksite approaches increase vegetable and fruit consumption. For the community food access questions addressing the relationship between food retail settings and dietary intake/quality and weight status the evidence was too limited or insufficient to assign grades. To reduce the disparity gaps that currently exist in low resource and underserved communities, more solution-oriented strategies need to be implemented and evaluated on ways to increase access to and procurement of healthy affordable foods, and also to reduce access to energy-dense, nutrient-poor foods.[64, 65] Although several innovative approaches are taking place now throughout the country, they generally lack adequate evaluation efforts.

One striking aspect of the Committee's findings was the power of multi-component interventions over single component interventions. For obesity prevention, effective multi-component interventions incorporated both nutrition and physical activity using a variety of strategies such as environmental policies to improve the availability and provision of healthy foods; increasing opportunities for physical activity, increased parent engagement; and educational approaches, such as a school nutrition curriculum. For multi-component dietary interventions (e.g., to increase consumption of vegetables and fruits) the most effective strategies included nutrition education, parent engagement, and environmental modifications (e.g., policies for nutrition standards, food service changes, point of purchase information).

The evidence reviewed in this chapter will inform and guide new multi-component individual and environmental and policy approaches in settings where people eat and procure their food to successfully target improvements in dietary intake and weight status. Collaborative partnerships and strategic efforts are needed to translate this evidence to action. Further work on restructuring the environment to facilitate healthy eating and physical activity, especially in high risk populations, is needed to advance evidence-based solutions that can be scaled up.

NEEDS FOR FUTURE RESEARCH

1. Develop more valid and reliable methods for measuring all aspects of the food environment, including the total food environment of communities. These methods can then be used to assess the impact of the food environment on community health as well as on economic development and growth.

Rationale: The food environment has become more complex, with more and more retail outlets selling food and beverages. Having valid and reliable methodologies for a variety of food environments and settings (tools and new analytical approaches) will allow more meaningful inquiry into the contributions of various settings in supporting or hindering nutritional health.

2. Identify, implement, evaluate, and scale up best practices (including private-public partnerships) for affordable and sustainable solutions to improving the food environment and increasing food access, especially in those environments of greatest need.

Rationale: The environments in which people live, work, learn, and play greatly influence their food intake. To best guide efforts to improve the food environment, research is needed to identify and evaluate best practices to direct available resources to new programs and scale up.

3. Identify, implement, accelerate, evaluate, and scale up programs that improve access to healthy food and that can be integrated seamlessly with Federal nutrition assistance programs, such as SNAP, WIC and elder nutrition.

Rationale: Federal nutrition assistance programs reach individuals and populations with the greatest health disparities. Identifying and evaluating initiatives that integrate improvements in the food environment with Federal programs will help ensure that Federal nutrition assistance programs have as great an impact as possible.

4. Conduct additional obesity prevention intervention research in child care settings (e.g., child-care centers, family child-care homes) to: 1) Identify the most potent components of the interventions and the optimal combinations for improving diet quality, physical activity, and weight outcomes; 2) Assess implementation and translation costs and benefits of the intervention, including impact, cost-effectiveness, generalizability and reach, sustainability and feasibility; 3) Develop and evaluate culturally appropriate and tailored interventions for preschool children in low-income and racial/ethnic communities, given the disproportionate impact of obesity in these groups; 4) Explore intervention strategies on how to use child care settings as access points to create linkages to parents, caretakers, and health care providers as partners in health promotion; 5) Evaluate the impact of Federal, state, and local policies, regulations, and support (e.g., provider training and technical assistance) for child care programs on the eating and physical activity practices and behaviors, and weight status of young children.

Rationale: Early care and education settings are an important venue for interventions targeting young children. A strong evidence base is essential to identify and support evidence-based practices and policies that can be implemented at Federal, state, and local levels and to mobilize efforts to improve healthy eating and physical activity, leading to healthy weight development in these settings. Interventions found to effectively reduce risk of obesity in one setting need to be appropriately adapted for diverse groups and different settings.

5. Improve intervention research methods by the use of stronger study designs and the development of standardized assessments of body composition, weight status. Develop enhanced validated measures of diet quality, feeding and physical activity practices, and physical activity and eating behaviors and policies. Create standardized measures to assess the nutrition quality of meals and snacks in child care settings, as well as the food and physical activity environments. Create standardized methods for assessing the relationship of child care food, nutrition and physical activity-related measures to similar measures representing non-child care time are needed to provide greater consistency in determining the contributors to the development and progression of childhood overweight and obesity.

   Rationale: Although many of the studies included in these evidence reviews were methodologically strong and were controlled studies, some were limited by small sample size, lack of adequate control for confounding factors, and different outcome measures and different tools used to measure the outcome variables.

6. Examine the effect of the recommended Child and Adult Care Food Program (CACFP) through ongoing periodic evaluations and fill gaps in the knowledge regarding participation, demand, food procurement and practices, nutrient intake, and food security.

   Rationale: Improvements in school meals and the school food environment have been fostered by national data from periodic studies such as the USDA/FNS School Nutrition Dietary Assessment Studies (SNDA), the HHS/CDC School Health Policies and Practices Studies (SHPPS) and the HHS/NIH C.L.A.S.S. In contrast, considerably fewer periodic national studies are conducted of meals and dietary intake in child care settings and their relation to the child care food and physical activity environment.

7. Conduct new research to document the types and quantities of foods and beverages students consume both at school and daily outside of school, before, during, and after school-based healthy eating approaches and policies are implemented.

   Rationale: Effective school-based approaches and policies to improve the availability, accessibility, and consumption of healthy foods and beverages, and reduce competition from unhealthy offerings, are central to improving the weight status and health of children and adolescents. Accurate quantification of the types and quantities of foods and beverages the students consume before, during, and after approaches and policies are implemented is fundamental to assessing effectiveness. However, many of the studies included in the systematic reviews and meta-analyses used by the DGAC to address this issue did not comprehensively measure or report dietary information. Although the USDA/FNS-sponsored

School Nutrition Dietary Assessment (SNDA) series collects student dietary intake data every 10 years, the DGAC recommends more frequent and consistent data collection, especially before and periodically after implementation of school-based nutrition and physical activity policy and program changes.

8. Improve the quality of research studies designed to assess the effects of school-based approaches and policies on dietary behaviors and body weight control to reduce the risk of bias, with an emphasis on randomized controlled trials.

   Rationale: Although the methodological quality of the systematic reviews and meta-analyses used by the DGAC to evaluate school-based approaches and policies on dietary intake and body weight outcomes was high, the authors of these reviews commented that the scientific quality of individual studies was generally poor and the risk of bias high. Many of the studies were done using quasi-experimental (with or without control), pre-post intervention, or cross-sectional designs. Future research should prioritize using prospective, repeated measures, randomized controlled trial experimental designs, with randomization at the individual, classroom, school, or school district level. Pilot feasibility studies also may be helpful to quickly identify promising novel approaches to improve dietary intake and weight control outcomes.

9. Conduct post-program follow-up assessments lasting longer than 1 year to determine the long-term retention of the changed nutrition behaviors as well as the usefulness of continuing to offer the programs while children advance in school grade. Also, conduct research is needed in adolescents (grades 9-12).

   Rationale: Literature supports that eating and physical activity behaviors and body weight status of children predict changes over time as they progress into adolescence and adulthood. Ideally, improvements in dietary intake and weight status achieved due to a given school-based approach or policy would be sustained over time and progressive improvements would occur long-term. The vast majority of published research focuses on children in grades K-8, or ages 4-12 years, and new and improved data are needed on adolescents and the transition from childhood to adolescence.

10. Encourage a wider variety of school-based approaches and policies to develop and evaluate innovative approaches focused on increasing vegetable intakes.

    Rationale: Consumption of non-potato vegetables is below 2010 Dietary Guidelines for Americans recommendations in both children and adolescents. Published research indicates that school-based approaches and policies designed to increase vegetable and fruit intakes are generally more effective at increasing fruit intake, except for –school gardens and economic incentives, which increase vegetable intake among school-aged children. Some past public policies (e.g. the Basic 4) treated fruit and vegetables and as a single food group, which props the need for new research that uses prospective, repeated measures, and randomized controlled trial experimental designs to specifically target increased consumption of healthy vegetables.

11. Conduct assessments of the effectiveness of worksite interventions that emphasize obesity prevention and weight control among workers across racially/ethnically diverse populations, blue and white collar employees, and at-risk populations. Scientifically rigorous studies (especially randomized controlled trials) addressing the long-term health impact of worksite-based approaches and policies that improve employee diet, physical activity, and body weight control would have public health relevance.

    Rationale: In light of the high rates of obesity and overweight, worksite interventions targeting obesity prevention and weight control through enhanced dietary behaviors and increased physical activity among workers is important. The majority of the studies to date have been conducted for relatively short periods of time, and the long-term impact of these approaches and policies may prove beneficial.

REFERENCES

1. Carroll-Scott A, Gilstad-Hayden K, Rosenthal L, Peters SM, McCaslin C, Joyce R, et al. Disentangling neighborhood contextual associations with child body mass index, diet, and physical activity: the role of built, socioeconomic, and social environments. Soc Sci Med. 2013;95:106-14. PMID: 23642646. http://www.ncbi.nlm.nih.gov/pubmed/23642646.
2. Caspi CE, Kawachi I, Subramanian SV, Adamkiewicz G, Sorensen G. The relationship between diet and perceived and objective access to supermarkets among low-income housing residents. Soc Sci Med. 2012;75(7):1254-62. PMID: 22727742. http://www.ncbi.nlm.nih.gov/pubmed/22727742.
3. Dean WR, Sharkey JR. Rural and urban differences in the associations between characteristics of the community food environment and fruit and vegetable intake. J Nutr Educ Behav. 2011;43(6):426-33. PMID: 21616721. http://www.ncbi.nlm.nih.gov/pubmed/21616721.
4. Ding D, Sallis JF, Norman GJ, Saelens BE, Harris SK, Kerr J, et al. Community food environment, home food environment, and fruit and vegetable intake of children and adolescents. J Nutr Educ Behav. 2012;44(6):634-8. PMID: 21531177. http://www.ncbi.nlm.nih.gov/pubmed/21531177.
5. Jago R, Baranowski T, Baranowski JC, Cullen KW, Thompson D. Distance to food stores & adolescent male fruit and vegetable consumption: mediation effects. Int J Behav Nutr Phys Act. 2007;4:35. PMID: 17850673. http://www.ncbi.nlm.nih.gov/pubmed/17850673.

6. Jilcott Pitts SB, Wu Q, McGuirt JT, Crawford TW, Keyserling TC, Ammerman AS. Associations between access to farmers' markets and supermarkets, shopping patterns, fruit and vegetable consumption and health indicators among women of reproductive age in eastern North Carolina, U.S.A. Public Health Nutr. 2013;16(11):1944-52. PMID: 23701901. http://www.ncbi.nlm.nih.gov/pubmed/23701901.
7. Sharkey JR, Johnson CM, Dean WR. Food access and perceptions of the community and household food environment as correlates of fruit and vegetable intake among rural seniors. BMC Geriatr. 2010;10:32. PMID: 20525208. http://www.ncbi.nlm.nih.gov/pubmed/20525208.
8. An R, Sturm R. School and residential neighborhood food environment and diet among California youth. Am J Prev Med. 2012;42(2):129-35. PMID: 22261208. http://www.ncbi.nlm.nih.gov/pubmed/22261208.
9. Jack D, Neckerman K, Schwartz-Soicher O, Lovasi GS, Quinn J, Richards C, et al. Socio-economic status, neighbourhood food environments and consumption of fruits and vegetables in New York City. Public Health Nutr. 2013;16:1197-205. PMID: 23388104. http://www.ncbi.nlm.nih.gov/pubmed/23388104.
10. Moore LV, Diez Roux AV, Nettleton JA, Jacobs DR, Jr. Associations of the local food environment with diet quality--a comparison of assessments based on surveys and geographic information systems: the multi-ethnic study of atherosclerosis. Am J Epidemiol. 2008;167(8):917-24. PMID: 18304960. http://www.ncbi.nlm.nih.gov/pubmed/18304960.
11. Ollberding NJ, Nigg CR, Geller KS, Horwath CC, Motl RW, Dishman RK. Food outlet accessibility and fruit and vegetable consumption. Am J Health Promot. 2012;26(6):366-70. PMID: 22747319. http://www.ncbi.nlm.nih.gov/pubmed/22747319.
12. Powell LM, Zhao Z, Wang Y. Food prices and fruit and vegetable consumption among young American adults. Health Place. 2009;15(4):1064-70. PMID: 19523869. http://www.ncbi.nlm.nih.gov/pubmed/19523869.
13. Zenk SN, Lachance LL, Schulz AJ, Mentz G, Kannan S, Ridella W. Neighborhood retail food environment and fruit and vegetable intake in a multiethnic urban population. Am J Health Promot. 2009;23(4):255-64. PMID: 19288847. http://www.ncbi.nlm.nih.gov/pubmed/19288847.
14. Laska MN, Hearst MO, Forsyth A, Pasch KE, Lytle L. Neighbourhood food environments: are they associated with adolescent dietary intake, food purchases and weight status? Public Health Nutr. 2010;13(11):1757-63. PMID: 20529405. http://www.ncbi.nlm.nih.gov/pubmed/20529405.
15. Gustafson A, Lewis S, Perkins S, Wilson C, Buckner E, Vail A. Neighbourhood and consumer food environment is associated with dietary intake among Supplemental Nutrition Assistance Program (SNAP) participants in Fayette County, Kentucky. Public Health Nutr. 2013;16(7):1229-37. PMID: 23462377. http://www.ncbi.nlm.nih.gov/pubmed/23462377.

16. Boone-Heinonen J, Gordon-Larsen P, Kiefe CI, Shikany JM, Lewis CE, Popkin BM. Fast food restaurants and food stores: longitudinal associations with diet in young to middle-aged adults: the CARDIA study. Arch Intern Med. 2011;171(13):1162-70. PMID: 21747011. http://www.ncbi.nlm.nih.gov/pubmed/21747011.
17. Gittelsohn J, Vijayadeva V, Davison N, Ramirez V, Cheung LW, Murphy S, et al. A food store intervention trial improves caregiver psychosocial factors and children's dietary intake in Hawaii. Obesity (Silver Spring). 2010;18 Suppl 1:S84-90. PMID: 20107467. http://www.ncbi.nlm.nih.gov/pubmed/20107467.
18. Gustafson AA, Sharkey J, Samuel-Hodge CD, Jones-Smith JC, Cai J, Ammerman AS. Food Store Environment Modifies Intervention Effect on Fruit and Vegetable Intake among Low-Income Women in North Carolina. J Nutr Metab. 2012;2012:932653. PMID: 22315676. http://www.ncbi.nlm.nih.gov/pubmed/22315676.
19. Morland K, Diez Roux AV, Wing S. Supermarkets, other food stores, and obesity: the atherosclerosis risk in communities study. Am J Prev Med. 2006;30(4):333-9. PMID: 16530621. http://www.ncbi.nlm.nih.gov/pubmed/16530621.
20. Drewnowski A, Aggarwal A, Hurvitz PM, Monsivais P, Moudon AV. Obesity and supermarket access: proximity or price? Am J Public Health. 2012;102(8):e74-80. PMID: 22698052. http://www.ncbi.nlm.nih.gov/pubmed/22698052.
21. Fiechtner L, Block J, Duncan DT, Gillman MW, Gortmaker SL, Melly SJ, et al. Proximity to supermarkets associated with higher body mass index among overweight and obese preschool-age children. Prev Med. 2013;56(3-4):218-21. PMID: 23219681. http://www.ncbi.nlm.nih.gov/pubmed/23219681.
22. Liu GC, Wilson JS, Qi R, Ying J. Green neighborhoods, food retail and childhood overweight: differences by population density. Am J Health Promot. 2007;21(4 Suppl):317-25. PMID: 17465177. http://www.ncbi.nlm.nih.gov/pubmed/17465177.
23. Ahern M, Brown C, Dukas S. A national study of the association between food environments and county-level health outcomes. J Rural Health. 2011;27(4):367-79. PMID: 21967380. http://www.ncbi.nlm.nih.gov/pubmed/21967380.
24. Bodor JN, Rice JC, Farley TA, Swalm CM, Rose D. The association between obesity and urban food environments. J Urban Health. 2010;87(5):771-81. PMID: 20458548. http://www.ncbi.nlm.nih.gov/pubmed/20458548.
25. Brown AF, Vargas RB, Ang A, Pebley AR. The neighborhood food resource environment and the health of residents with chronic conditions: the food resource environment and the health of residents. J Gen Intern Med. 2008;23(8):1137-44. PMID: 18483833. http://www.ncbi.nlm.nih.gov/pubmed/18483833
26. Ford PB, Dzewaltowski DA. Limited supermarket availability is not associated with obesity risk among participants in the Kansas WIC Program. Obesity (Silver Spring). 2010;18(10):1944-51. PMID: 20057363. http://www.ncbi.nlm.nih.gov/pubmed/20057363.

27. Galvez MP, Hong L, Choi E, Liao L, Godbold J, Brenner B. Childhood obesity and neighborhood food-store availability in an inner-city community. Acad Pediatr. 2009;9:339-43. PMID: 19560992. http://www.ncbi.nlm.nih.gov/pubmed/19560992.
28. Morland KB, Evenson KR. Obesity prevalence and the local food environment. Health Place. 2009;15(2):491-5. PMID: 19022700. http://www.ncbi.nlm.nih.gov/pubmed/19022700.
29. Rundle A, Neckerman KM, Freeman L, Lovasi GS, Purciel M, Quinn J, et al. Neighborhood food environment and walkability predict obesity in New York City. Environ Health Perspect. 2009;117(3):442-7. PMID: 19337520. http://www.ncbi.nlm.nih.gov/pubmed/19337520.
30. Salois MJ. Obesity and diabetes, the built environment, and the 'local' food economy in the United States, 2007. Econ Hum Biol. 2012;10(1):35-42. PMID: 21561816. http://www.ncbi.nlm.nih.gov/pubmed/21561816.
31. Stark JH, Neckerman K, Lovasi GS, Konty K, Quinn J, Arno P, et al. Neighbourhood food environments and body mass index among New York City adults. J Epidemiol Community Health. 2013;67(9):736-42. PMID: 23851151. http://www.ncbi.nlm.nih.gov/pubmed/23851151.
32. Viola D, Arno PS, Maroko AR, Schechter CB, Sohler N, Rundle A, et al. Overweight and obesity: can we reconcile evidence about supermarkets and fast food retailers for public health policy? J Public Health Policy. 2013;34(3):424-38. PMID: 23719294. http://www.ncbi.nlm.nih.gov/pubmed/23719294.
33. Wang MC, Kim S, Gonzalez AA, MacLeod KE, Winkleby MA. Socioeconomic and food-related physical characteristics of the neighbourhood environment are associated with body mass index. J Epidemiol Community Health. 2007;61:491-8. PMID: 17496257. http://www.ncbi.nlm.nih.gov/pubmed/17496257.
34. Block JP, Christakis NA, O'Malley AJ, Subramanian SV. Proximity to food establishments and body mass index in the Framingham Heart Study offspring cohort over 30 years. Am J Epidemiol. 2011;174(10):1108-14. PMID: 21965186. http://www.ncbi.nlm.nih.gov/pubmed/21965186.
35. Gibson DM. The neighborhood food environment and adult weight status: estimates from longitudinal data. Am J Public Health. 2011;101(1):71-8. PMID: 21088263. http://www.ncbi.nlm.nih.gov/pubmed/21088263.
36. Lee H. The role of local food availability in explaining obesity risk among young school-aged children. Soc Sci Med. 2012;74(8):1193-203. PMID: 22381683. http://www.ncbi.nlm.nih.gov/pubmed/22381683.
37. Leung CW, Laraia BA, Kelly M, Nickleach D, Adler NE, Kushi LH, et al. The influence of neighborhood food stores on change in young girls' body mass index. Am J Prev Med. 2011;41(1):43-51. PMID: 21665062. http://www.ncbi.nlm.nih.gov/pubmed/21665062.

38. Rossen LM, Curriero FC, Cooley-Strickland M, Pollack KM. Food availability en route to school and anthropometric change in urban children. J Urban Health. 2013;90(4):653-66. PMID: 23392564. http://www.ncbi.nlm.nih.gov/pubmed/23392564.

39. Shier V, An R, Sturm R. Is there a robust relationship between neighbourhood food environment and childhood obesity in the USA? Public Health. 2012;126(9):723-30. PMID: 22898435. http://www.ncbi.nlm.nih.gov/pubmed/22898435.

40. Sturm R, Datar A. Body mass index in elementary school children, metropolitan area food prices and food outlet density. Public Health. 2005;119(12):1059-68. PMID: 16140349. http://www.ncbi.nlm.nih.gov/pubmed/16140349.

41. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. JAMA. 2014;311(8):806-14. PMID: 24570244. http://www.ncbi.nlm.nih.gov/pubmed/24570244.

42. Shashaj B, Bedogni G, Graziani MP, Tozzi AE, DiCorpo ML, Morano D, et al. Origin of cardiovascular risk in overweight preschool children: a cohort study of cardiometabolic risk factors at the onset of obesity. JAMA Pediatr. 2014;168(10):917-24. PMID: 25111132. http://www.ncbi.nlm.nih.gov/pubmed/25111132.

43. Institute of Medicine. Child and Adult Care Food Program: Aligning Dietary Guidance for All. 2011. Washington, DC: National Academies Press. Available from: http://books.nap.edu/openbook.php?record_id=12959&page=R2.

44. Zhou YE, Emerson JS, Levine RS, Kihlberg CJ, Hull PC. Childhood obesity prevention interventions in childcare settings: systematic review of randomized and nonrandomized controlled trials. Am J Health Promot. 2014;28(4):e92-103. PMID: 24200332. http://www.ncbi.nlm.nih.gov/pubmed/24200332.

45. Alkon A, Crowley AA, Neelon SE, Hill S, Pan Y, Nguyen V, et al. Nutrition and physical activity randomized control trial in child care centers improves knowledge, policies, and children's body mass index. BMC Public Health. 2014;14(1):215. PMID: 24580983. http://www.ncbi.nlm.nih.gov/pubmed/24580983.

46. Cespedes J, Briceno G, Farkouh ME, Vedanthan R, Baxter J, Leal M, et al. Promotion of cardiovascular health in preschool children: 36-month cohort follow-up. Am J Med. 2013;126(12):1122-6. PMID: 24262725. http://www.ncbi.nlm.nih.gov/pubmed/24262725.

47. Cespedes J, Briceno G, Farkouh ME, Vedanthan R, Baxter J, Leal M, et al. Targeting preschool children to promote cardiovascular health: cluster randomized trial. Am J Med. 2013;126(1):27-35.e3. PMID: 23062403. http://www.ncbi.nlm.nih.gov/pubmed/23062403.

48. De Bock F, Breitenstein L, Fischer JE. Positive impact of a pre-school-based nutritional intervention on children's fruit and vegetable intake: results of a cluster-randomized trial. Public Health Nutr. 2012;15(3):466-75. PMID: 21859516. http://www.ncbi.nlm.nih.gov/pubmed/21859516.

49. De Coen V, De Bourdeaudhuij I, Vereecken C, Verbestel V, Haerens L, Huybrechts I, et al. Effects of a 2-year healthy eating and physical activity intervention for 3-6-year-olds in communities of high and low socio-economic status: the POP (Prevention of Overweight among Pre-school and school children) project. Public Health Nutr. 2012;15(9):1737-45. PMID: 22397833. http://www.ncbi.nlm.nih.gov/pubmed/22397833.

50. Herman A, Nelson BB, Teutsch C, Chung PJ. "Eat Healthy, Stay Active!": a coordinated intervention to improve nutrition and physical activity among Head Start parents, staff, and children. Am J Health Promot. 2012;27(1):e27-36. PMID: 22950932. http://www.ncbi.nlm.nih.gov/pubmed/22950932.

51. Natale RA, Lopez-Mitnik G, Uhlhorn SB, Asfour L, Messiah SE. Effect of a Child Care Center-Based Obesity Prevention Program on Body Mass Index and Nutrition Practices Among Preschool-Aged Children. Health Promot Pract. 2014. PMID: 24662896. http://www.ncbi.nlm.nih.gov/pubmed/24662896.

52. Yin Z, Parra-Medina D, Cordova A, He M, Trummer V, Sosa E, et al. Miranos! Look at us, we are healthy! An environmental approach to early childhood obesity prevention. Child Obes. 2012;8(5):429-39. PMID: 23061498. http://www.ncbi.nlm.nih.gov/pubmed/23061498http://online.liebertpub.com/doi/pdfplus/10.1089/chi.2011.0125.

53. Evans CE, Christian MS, Cleghorn CL, Greenwood DC, Cade JE. Systematic review and meta-analysis of school-based interventions to improve daily fruit and vegetable intake in children aged 5 to 12 y. Am J Clin Nutr. 2012;96(4):889-901. PMID: 22952187. http://www.ncbi.nlm.nih.gov/pubmed/22952187.

54. Jensen JD, Hartmann H, de Mul A, Schuit A, Brug J, Consortium E. Economic incentives and nutritional behavior of children in the school setting: a systematic review. Nutr Rev. 2011;69(11):660-74. PMID: 22029832. http://www.ncbi.nlm.nih.gov/pubmed/22029832.

55. Langellotto G GA. Gardening Increases Vegetable Consumption in School-aged Children: A Meta-analytical Synthesis. HortTechnology. 2012;22(4):430-45. http://horttech.ashspublications.org/content/22/4/430.abstract.

56. Chriqui JF, Pickel M, Story M. Influence of school competitive food and beverage policies on obesity, consumption, and availability: a systematic review. JAMA Pediatr. 2014;168(3):279-86. PMID: 24473632. http://www.ncbi.nlm.nih.gov/pubmed/24473632.

57. Waters E, de Silva-Sanigorski A, Hall BJ, Brown T, Campbell KJ, Gao Y, et al. Interventions for preventing obesity in children. Cochrane Database Syst Rev. 2011(12):CD001871. PMID: 22161367. http://www.ncbi.nlm.nih.gov/pubmed/22161367.

58. Wang Y WY, Wilson RF, Bleich S, Cheskin L, Weston C, Showell N, Fawole O, Lau B, Segal J, . Childhood obesity prevention programs: comparative effectiveness review and meta-analysis. Comparative effectiveness review No. 115. AHRQ Publication No. 13-EHC081-EF [Internet]. June 2013. Available from: http://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0057623/pdf/TOC.pdf.

59. Williams AJ, Henley WE, Williams CA, Hurst AJ, Logan S, Wyatt KM. Systematic review and meta-analysis of the association between childhood overweight and obesity and primary school diet and physical activity policies. Int J Behav Nutr Phys Act. 2013;10:101. PMID: 23965018. http://www.ncbi.nlm.nih.gov/pubmed/23965018.
60. Geaney F, Kelly C, Greiner BA, Harrington JM, Perry IJ, Beirne P. The effectiveness of workplace dietary modification interventions: a systematic review. Prev Med. 2013;57(5):438-47. PMID: 23850518. http://www.ncbi.nlm.nih.gov/pubmed/23850518.
61. Aneni EC, Roberson LL, Maziak W, Agatston AS, Feldman T, Rouseff M, et al. A systematic review of internet-based worksite wellness approaches for cardiovascular disease risk management: outcomes, challenges & opportunities. PLoS One. 2014;9(1):e83594. PMID: 24421894. http://www.ncbi.nlm.nih.gov/pubmed/24421894.
62. Kahn-Marshall JL, Gallant MP. Making healthy behaviors the easy choice for employees: a review of the literature on environmental and policy changes in worksite health promotion. Health Educ Behav. 2012;39(6):752-76. PMID: 22872583. http://www.ncbi.nlm.nih.gov/pubmed/22872583.
63. Verweij LM, Coffeng J, van Mechelen W, Proper KI. Meta-analyses of workplace physical activity and dietary behaviour interventions on weight outcomes. Obes Rev. 2011;12(6):406-29. PMID: 20546142. http://www.ncbi.nlm.nih.gov/pubmed/20546142.
64. Bell J, Mora G, Hagan E, Rubin V, Karpyn A. Access to Healthy Food and Why It Matters: a Review of the Research. Oakland CA: PolicyLink. The Food Trust; 2013. Available from: http://thefoodtrust.org/uploads/media_items/access-to-healthy-food.original.pdf.
65. Treuhaft S, Karpyn A. The Grocery Gap: Who Has Access to Healthy Food and Why It Matters. Oakland, CA: PolicyLink. The Food Trust,; 2010. Available from: http://thefoodtrust.org/uploads/media_items/grocerygap.original.pdf.

Part D. Chapter 5: Food Sustainability and Safety

INTRODUCTION

In this chapter, the DGAC addresses food and nutrition issues that will inform public health action and policies to promote the health of the population through sustainable diets and food safety. An important reason for addressing sustainable diets, a new area for the DGAC, is to have alignment and consistency in dietary guidance that promotes both health and sustainability. This also recognizes the significant impact of food and beverages on environmental outcomes, from farm to plate to waste disposal, and, therefore, the need for dietary guidance to include the wider issue of sustainability. Addressing this complex challenge is essential to ensure a healthy food supply will be available for future generations. The availability and acceptability of healthy and sustainable food choices will be necessary to attain food security for the U.S. population over time. Integral to this issue is how dietary guidance and individual food choices influence the nation's capacity to meet the nutritional needs of the U.S. population. Food sustainability and food safety are also interrelated in generating a secure food supply. This chapter focuses on both sustainable diets and food safety.

Food Sustainability

Two definitions are relevant to the material presented in this chapter. These terms were slightly modified from the Food and Agriculture Organization (FAO) definitions to operationalize them for the Committee's work.[1, 2]

<u>Sustainable diets</u>: Sustainable diets are a pattern of eating that promotes health and well-being and provides food security for the present population while sustaining human and natural resources for future generations.

<u>Food security</u>: Food security exists when all people now, and in the future, have access to sufficient, safe, and nutritious food to maintain a healthy and active life.

The topic of *current* food security was addressed in Chapter 3 and to some extent in Chapter 4, where federal food programs were discussed. The topic of *long-term* food security was addressed within this chapter through examination of the evidence on sustainable diets.

The environmental impact of food production is considerable and if natural resources such as land, water and energy are not conserved and managed optimally, they will be strained and potentially lost. The global production of food is responsible for 80 percent of deforestation, more than 70 percent of fresh water use, and up to 30 percent of human-generated greenhouse gas (GHG) emissions.[3] It also is the largest cause of species biodiversity loss.[3] The capacity to produce adequate food in the future is constrained by land use, declining soil fertility, unsustainable water use, and over-fishing of the marine environment.[4] Climate change, shifts in population dietary patterns and demand for food products, energy costs, and population growth will continue to put additional pressures on available natural resources. Meeting current and future food needs will depend on two concurrent approaches: altering individual and population dietary choices and patterns and developing agricultural and production practices that reduce environmental impacts and conserve resources, while still meeting food and nutrition needs. In this chapter, the Committee focuses primarily on the former, examining the effect of population-level dietary choices on sustainability.

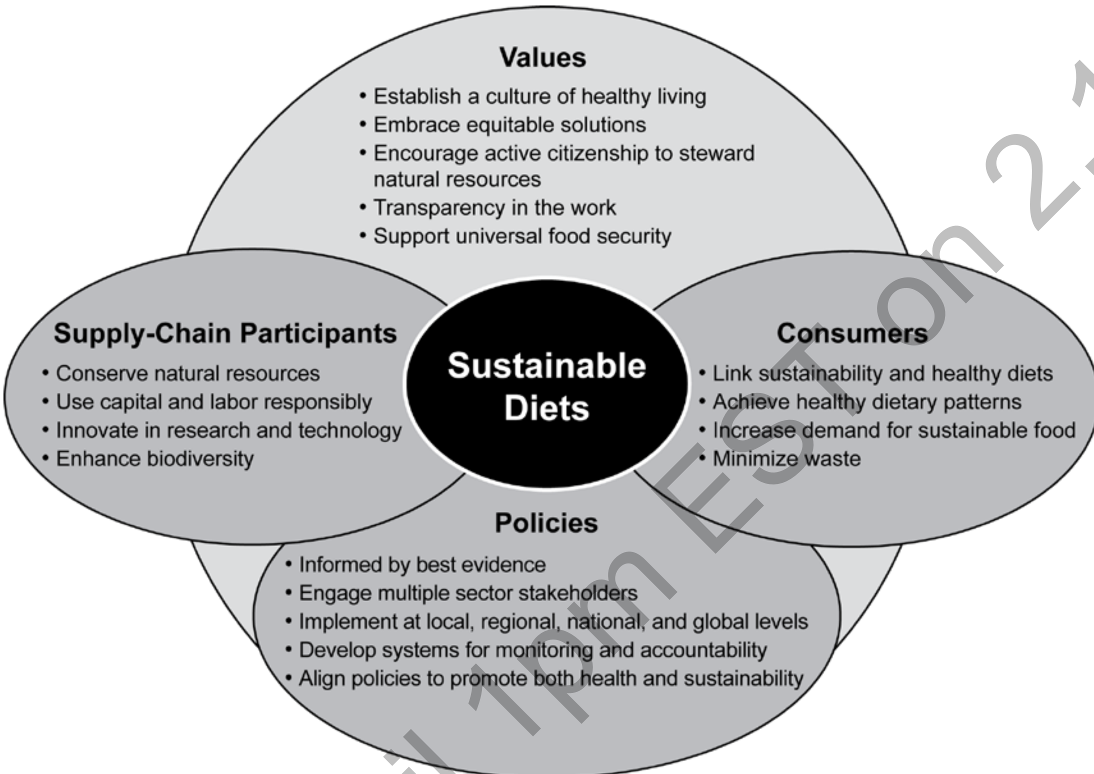
Foods vary widely in the type and amount of resources required for production, so as population-level consumer demand impacts food production (and imports) it will also indirectly influence how and to what extent resources are used.[3] As the focus of the dietary guidelines is to shift consumer eating habits toward healthier alternatives, it is imperative that, in this context, the shift also involve movement toward less resource-intensive diets. Individual and population-level adoption of more sustainable diets can change consumer demand away from more resource-intensive foods to foods that have a lower environmental impact.[3]

In this chapter, the DGAC has used an evidence-based approach to evaluate the foods and food components that improve the sustainability of dietary patterns as a step toward this desirable goal. The approach used was to determine dietary patterns that are nutritionally adequate and promote health, while at the same time are more protective of natural resources. This type of comprehensive strategy also has been used by intergovernmental organizations. For example, the FAO has identified the Mediterranean diet as an example of a sustainable diet due to its emphasis on biodiversity and smaller meat portions,[5] and the European Commission has developed a "2020 Live Well Diet" to reduce GHG emissions through diet change.[6]

It should be noted that research in the area of dietary patterns and sustainability is rapidly evolving and the methodologies for determining dietary patterns in populations and Life Cycle Analysis of foods/food components and environmental outcomes have made significant advances in recent years.[7, 8] This is exemplified by the size of evidence base for this question and the fact that several relevant articles have been published even since the close of the 2015 DGAC Nutrition Evidence Library (NEL) scientific review period for this topic.[9-11]

Figure D5.1 outlines the interconnected elements that the DGAC believes are necessary based on current evidence to develop sustainable diets. Sustainable diets are realized by developing a food system that embraces a core set of values illustrated in the figure. These values need to be implemented through robust private and public sector partnerships, practices and policies across the supply chain, extending from farms to distribution and consumption. New well-coordinated policies that include, but are not limited to, agriculture, economics, transportation, energy, water use, and dietary guidance need to be developed. Behaviors of all participants in the food system are central to creating and supporting sustainable diets.

Figure D5.1: Elements needed for sustainable diets



Although the addition of sustainability topics in the *Scientific Report of the 2015 Dietary Guidelines Advisory Committee* is new in 2015 it was acknowledged as a topic of strong relevance but not addressed by the 2010 DGAC. It has been a widely discussed aspect of nutrition policy for the past decade in countries such as Germany, Sweden and other Nordic countries, the Netherlands, Australia, and Brazil. For example, in the Netherlands, the Advisory report, *Guidelines for a Healthy Diet: The Ecological Perspective* focused on guidelines that inform both health and ecological benefits using an evidence-based strategy.[12] Nordic countries, such as Sweden, have been researching sustainability and dietary choice since the late 1990s with the most recent edition of the Nordic Nutrition Recommendations (NNR) including an emphasis on the environmental impact of dietary recommendations.[13] The German Dietary Guidelines developed a “sustainable shopping basket,” which is a consumer guide for shopping in a more sustainable way.[14] Overall, the environmentally sustainable dietary guidance from these countries includes elements identified in this DGAC report as consistent with the extant data: a focus on decreasing meat consumption, choosing seafood from non-threatened stocks, eating more plants and plant-based products, reducing energy intake, and reducing waste. Non-governmental and international organizations, such as the United Nations, the FAO, the Sustainable Development Commission in the United Kingdom (UK), the Institute of Medicine (IOM), the Academy of Nutrition and Dietetics, and the National Research Council have all convened working groups and commissioned reports on sustainable diets.[2, 15-19] Overall, it is clear that environmental sustainability adds further dimensions to dietary guidance; not just what we eat but where and how food production, processing, and transportation are managed, and waste is decreased.

The DGAC focused on two main topic areas related to sustainability: dietary patterns and seafood. The identification of dietary patterns that are sustainable is a first step toward driving consumer behavior change and demand and supply-chain changes. Furthermore, dietary patterns were an overall focus area of the 2015 DGAC and allow for a more comprehensive approach to total diet and health. This approach is particularly well suited for assessing overall environmental impacts of food consumption, as all food components of a dietary pattern are identified, and keeping within the context of health outcomes that have been documented for different dietary patterns. The topic area of seafood was chosen because consumption has well-established health benefits and the 2010 DGAC report highlighted the concern for seafood sustainability and called for a better understanding of the environmental impact of aquaculture on seafood contaminants. Meeting these recommendations, however, increases demand for seafood production and this, in turn, poses challenges, as certain seafood species are depleted and marine waters are over fished, while most other species are at the limits of sustainable harvesting. To meet these challenges, as world capture fisheries production has leveled off, aquaculture production has increased to meet demand.[20] Therefore, building upon the 2010 DGAC report, the 2015 DGAC addressed the health benefits (nutrients) versus the risks (contaminants) of farm-raised (aquaculture) compared to wild-caught seafood and reviewed the evidence on the worldwide capacity to produce enough seafood to meet dietary guidelines. Overall, promoting sustainable fishing and aquaculture can provide an example for broader ecosystem stewardship.[20]

Food Safety

Food safety was first introduced in the *2000 Dietary Guidelines for Americans*, and the recognition of the importance of food safety continued through the 2010 report. This chapter updates the 2010 DGAC report related to food safety behaviors in the home environment and evaluates new topics of food safety concern with very current and/or updated evidence. The current/updated topics include the safety of beverages, specifically coffee and caffeine, and food additives, specifically aspartame, in the U.S. food supply.

In 2015, the DGAC addressed new topics of concern. For the first time, the DGAC addressed the safety of coffee/caffeine consumption, as well as the safety of consuming higher doses of caffeine in products such as some energy drinks. The food additive, aspartame, has been the only non-nutritive sweetener to be completely re-evaluated in recent years and the results of this reevaluation were deemed important because it includes the most recent science on aspartame and health. These topic areas were chosen for consideration because they are of high public health concern and very recent evidence has been published that significantly updates the knowledge base on health aspects related to caffeine and aspartame in the diet.

For 2015, the DGAC brought forward the updated food safety principles to reduce risk of foodborne illnesses. These principles—Clean, Separate, Cook and Chill—are cornerstones of the Fight BAC! (www.fightbac.org) educational messages developed by the Partnership for Food Safety Education, a collaboration with the Federal government. These messages are reinforced by other USDA educational materials, including the *Be Food Safe* (www.befoodsafe.gov) efforts; Is it Done Yet? (www.isitdoneyet.gov); and *Thermy* (www.fsis.usda.gov/thermy), which outline key elements in thermometer use and placement to ensure proper cooking of meat, poultry, seafood, and egg products. Additional consumer-friendly information on food safety is available at www.foodsafety.gov. The DGAC brought forward the guidance for consumers that has been updated since 2010 on recommended procedures for hand sanitation, washing fresh produce, preventing cross-contamination, and safe meat, poultry, seafood and egg cooking temperatures and thermometer use from the FDA, the Center for Disease Control (CDC) and the Food Safety and Inspection Service (FSIS). The updated food safety tables are located at the end of this chapter.

LIST OF QUESTIONS

Sustainable Diets

Dietary Patterns

1. What is the relationship between population-level dietary patterns and long-term food sustainability?

Seafood

2. What are the comparative nutrient profiles of current farm-raised versus wild caught seafood?
3. What are the comparative contaminant levels of current farm-raised versus wild caught seafood?
4. What is the worldwide capacity to produce farm-raised versus wild-caught seafood that is nutritious and safe for Americans?

Food Safety

5. What is the relationship between usual coffee/caffeine consumption and health?

6. What is the relationship between high-dose caffeine consumption and health?
7. What is the relationship between aspartame consumption and health?
8. What consumer behaviors prevent food safety problems? (Topic update from 2010 DGAC)

METHODOLOGY

Sustainable Diets

The topic of Question 1 is new for a DGAC review and involves an emerging area of scientific investigation that is not readily addressed by traditional study designs such as randomized controlled trials and prospective cohort studies. The literature related to sustainable diets and dietary patterns involves a combination of food pattern modeling, Life Cycle Assessment (LCA) methodology (examines all processes in the life cycle of each food component - from farm to plate to waste), and determination of the environmental outcomes of the full LCA inventory. Because of the unique nature of these studies, a modified NEL systematic review was conducted for Question 1 on dietary patterns and sustainability. Databases included PubMed, Cochrane, Navigator, and Embase and the search covered from January 2000 to March 2014. For this topic and question, it was necessary to use different methods from those described in an original NEL protocol because not all methods in the protocol could be applied. This is sometimes necessary, according to the Cochrane Collaboration, but requires that methods from the original protocol that could not be implemented in the current review be summarized.[21] Due to the nature of the evidence, the NEL 6-step process was tailored for the purposes of this systematic review, with modifications to step 3 – extract data and assess the risk of bias. A description of the NEL systematic review process is provided in ***Part C: Methodology***. A new data extraction grid was developed with emphasis on modeling studies, LCA methodology, and environmental outcomes. The LCA is a standardized methodological framework for assessing the environmental impact (or load) attributable to the life cycle of a food product. The customized grid was then used by NEL abstractors to extract data from the included articles and this informed the evidence synthesis (see ***Appendix E-2.37 Evidence Portfolio***). In addition, NEL abstractors used a different tool to assess individual study quality, not the NEL Bias Assessment Tool (BAT). This alternative tool, the Critical Appraisal Checklist used by the *British Medical Journal*, was appropriate for studies that used a modeling design. This checklist assesses studies that use modeling to extrapolate progression of clinical outcomes, transform final outcomes from intermediate measures, examine relations between inputs and outputs to apportion resource use, and extrapolate findings from one clinical setting or population to another. To attain a high score, studies must report the variables that have been modeled rather than directly observed; what additional variables have been included or excluded; what statistical relations have been assumed; and what evidence supports these assumptions.[22-24] The checklist included key components of the *British Medical Journal* checklist for economic evaluations, together with the

Eddy checklist on mathematical models. This Critical Appraisal Checklist was reviewed and tested for applicability by two sustainability experts who served as consultants to the DGAC.

Question 2 on nutrient profiles in farm-raised versus wild-caught seafood was addressed using data analysis from the USDA-Agricultural Research Service (ARS) National Nutrient Database for Standard Reference, Release 27 (http://www.ars.usda.gov/ba/bhnrc/ndl).[25] The section on finfish and shellfish products included nutrient profiles for both farm-raised and wild-caught seafood for some species. These data were augmented using a USDA-funded report on fatty-acid profiles of commercially available fish[*] in the United States that assessed additional farmed species and compared results with the USDA-ARS NND.[26] Because this question was answered using data analysis, it was not graded (as described in ***Part C: Methodology***). For Question 3 on contaminants in farm-raised versus wild-caught seafood, the DGAC used an expert report, the *Report of the Joint Food and Agriculture Organization of the United Nations (FAO) and the World Health Organization (WHO) Expert Consultation on the Risks and Benefits of Fish Consumption, 2011*.[27] This report was chosen as the most updated and comprehensive source of scientific information on the net health assessment of seafood consumption, including a comparison between wild-caught and farm-raised seafood related to contaminants. Data on levels of chemical contaminants (methyl mercury and dioxins) in a large number of seafood species were reviewed, as well as recent scientific literature covering the risks and benefits of seafood consumption. The sections of the report that were used to address the question were "Data on the composition of fish" and "Risk-benefit comparisons." Lastly, to address Question 4 on the worldwide capacity to produce enough nutritious seafood, the Committee used the FAO's report on the *State of World Fisheries and Aquaculture, 2012*.[20] This was considered the most current and comprehensive source on this topic, specifically the sections on "Selected Issues in Fisheries and Aquaculture" and the "Organization for Economic Cooperation and Development (OECD)-FAO Agricultural Outlook: chapter on fish." The DGAC focused on matters that directly address world production as it affects the supply of seafood for the U.S. population, particularly as the U.S. relies on significant amounts of imported seafood (~90 percent).

Food Safety

For Question 5, the DGAC used an overview of systematic reviews (SRs)/meta-analyses (MA) to address the relationship between usual caffeine/coffee consumption and health. This approach allowed the DGAC to address the broad scope of the evidence on usual caffeine and health, which heretofore had not been addressed by a DGAC. The DGAC used a modification of the method described by the Cochrane Collaboration to conduct the review.[28] The steps included development of analytical framework, determination of inclusion/exclusion criteria, description of search strategy and databases used, determination of methodological quality using the

[*] The term "fish" in this chapter refers to finfish, which includes aquatic species such as salmon, tuna, and trout.

Assessment of Multiple Systematic Reviews (AMSTAR) tool, data extraction, summary of results and key findings, and development of conclusion and grade for each outcome, as well as implications of the evidence and research recommendations. Overlap of studies included across the SRs/MA for the same health outcome was determined and recorded; however, SRs/MA were not excluded for overlap. This approach allowed the Committee to assess and consider whether SRs/MA on the same topic *independently* assessed similar results and arrived at generally similar conclusions. The focus of this review was to summarize the existing SRs/MA on this question, *not* to re-synthesize the evidence or to conduct a new meta-analysis or meta-synthesis.

For the overview on usual caffeine/coffee consumption and health, the target population was healthy adults and adults at risk of chronic disease, as well as youth ages 2 years and older. The intervention or exposure was caffeine/coffee consumption. The outcomes were clinical endpoints: 1) chronic diseases, including cardiovascular, type 2 diabetes, and cancer, and total mortality, 2) neurologic and cognitive diseases, including Alzheimer's and Parkinson's disease, and 3) pregnancy outcomes, including miscarriage and low birth weight. The included studies were SRs/MA and qualitative SRs; the date range was from 2000 to 2014. Data were extracted for all SRs/MA with emphasis on MA results, including categorical and dose-response MA, fixed or random effects models, heterogeneity and sources of heterogeneity, sub-group analysis, and publication bias (see ***Appendix E-2.39b Systematic Review/Meta-Analysis Data Table***). The methodological quality of the included SRs/MA was determined using AMSTAR. Overlap of studies included across the SRs/MA for the same health outcomes was determined and recorded; however, SRs/MA were not excluded for overlap. Rather, the emphasis was to determine consistency across studies.

For Question 6 on high-dose caffeine and health, a duplication assessment found two SRs and these were used in lieu of conducting a full NEL SR. The details of duplication assessment are provided in ***Part C: Methodology***, and the Review of the Evidence for this question provide further detail.

For Question 7 on aspartame and health, the European Food Safety Authority (EFSA) *Scientific Opinion on the Re-evaluation of Aspartame as a Food Additive* was used. This was conducted by the EFSA Panel of Food Additives and Nutrient Sources Added to Food (ANS).[29] The Panel based its evaluation on original study reports and information submitted following public calls for data as well as previous evaluations and additional literature that was available up to February 2013. The 2015 DGAC considered only the human studies and related conclusions from the EFSA report; animal studies and *in vitro* studies were not considered.
Lastly, this chapter provides a topic update from the 2010 DGAC on consumer behaviors and food safety. Tables on this topic were updated to include the most recent recommendations. Federal sources that were used for the update include: 1) Centers for Disease Control and Prevention (CDC) - Hand washing: Clean Hands Save Lives;[30] 2) Food and Drug Administration

(FDA) - Food Facts, Raw Produce: Selecting It and Serving It Safely, 2012; Food Safety for Moms-to-Be: Safe Eats - Meat, Poultry & Seafood;[31] and 3) USDA/Food Safety and Inspection Service (FSIS) – Food Safety Fact Sheets.[32]

SUSTAINABLE DIETS

Evaluating the link between sustainability and dietary guidance will inform policies and practice to ensure food security for present and future generations. The DGAC concentrated its review on the inter-relatedness between human health and food sustainability, with a focus on dietary patterns, a theme of the 2015 DGAC.

Dietary Patterns and Sustainability

Question 1: What is the relationship between population-level dietary patterns and long-term food sustainability?

Source of Evidence: Modified NEL systematic review

Conclusion

Consistent evidence indicates that, in general, a dietary pattern that is higher in plant-based foods, such as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in animal-based foods is more health promoting and is associated with lesser environmental impact (GHG emissions and energy, land, and water use) than is the current average U.S. diet. A diet that is more environmentally sustainable than the average U.S. diet can be achieved without excluding any food groups. The evidence consists primarily of Life Cycle Assessment (LCA) modeling studies or land-use studies from highly developed countries, including the United States.

DGAC Grade: Moderate

Implications

A moderate to strong evidence base supports recommendations that the U.S. population move toward dietary patterns that generally increase consumption of vegetables, fruits, whole grains, legumes, nuts and seeds, while decreasing total calories and some animal-based foods. This can be achieved through a variety of dietary patterns, including the Healthy USDA-style Pattern, the Healthy Vegetarian Pattern, and the Healthy Mediterranean-style Pattern (for more details on the patterns, see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). Each of these patterns provides more plant-based foods and lower amounts of meat than are currently consumed by the U.S. population.

Sustainability considerations provide an additional rationale for following the Dietary Guidelines for Americans and should be incorporated into federal and local nutrition feeding programs when possible. Using sustainability messaging in communication strategies should be encouraged. The application of environmental and sustainability factors to dietary guidelines can be accomplished because of the compatibility and degree of overlap between favorable health and environmental outcomes.

Much has been done by the private and public sectors to improve environmental policies and practices around production, processing, and distribution *within* individual food categories. It will be important that *both* a greater shift toward healthful dietary patterns and an improved environmental profile across food categories are achieved to maximize environmental sustainability now and to ensure greater progress in this direction over time.

Consumer friendly information that facilitates understanding the environmental impact of different foods should be considered for inclusion in food and menu labeling initiatives.

Careful consideration will need to be made to ensure that sustainable diets are affordable for the entire U.S. population.

Promoting healthy diets that also are more environmentally sustainable now will conserve resources for present and future generations, ensuring that the U.S. population has access to a diet that is healthy as well as sustainable and secure in the future.

Review of the Evidence

A total of 15 studies met the inclusion criteria for this systematic review.[33-48] The body of evidence consisted primarily of dietary pattern modeling studies that assessed related environmental outcomes. These studies were conducted between the years 2003 and 2014 in the U.S., the UK, Germany, the Netherlands, France, Spain, Italy, Australia, Brazil, and New Zealand. Dietary patterns that were examined included vegetarian, lacto-ovo vegetarian, and vegan dietary patterns; the average and dietary guidelines-related dietary patterns of respective countries examined; Mediterranean-style dietary patterns; and sustainable diets. The most frequent comparison diet was the average dietary pattern of the country, although numerous studies made additional comparisons across many of the above dietary patterns. Another approach was to examine diet “scenarios” that modeled different percentage replacements of meat and dairy foods with plant-based foods. The modeling studies used cross-sectional assessment of dietary intake from national nutrition surveys of representative adult populations; for example, the British National Diet and Nutrition Survey (NDNS) from studies in the UK,[34, 39] the National Nutrition Surveys (NNS) in Germany,[40] or the Australian National Nutrition Survey[38] were used to determine the observed average dietary patterns. The average dietary patterns were then compared with other modeled dietary patterns, such as vegetarian or Mediterranean- style patterns, as described in detail below. All of the countries were highly developed countries with dietary guidelines and, therefore, generalizable to the U.S. population. The study quality for the body of evidence ranged from scores of 7/12 to 12/12 (indicating the evidence was of high quality) using a modified Critical Appraisal Checklist (see ***Appendix E-2.37 Evidence Portfolio***).

Health outcomes associated with the dietary patterns were most often documented based on adherence to dietary guidelines-related patterns, variations on vegetarian dietary patterns, or Mediterranean-style dietary patterns. Diet quality was assessed in some studies using an a priori index, such as the Healthy Eating Index (HEI) or the WHO Index. In some studies, health outcomes also were modeled. For example Scarborough et al. used the DIETRON model to estimate deaths delayed or averted for each diet pattern.[46] One study assessed the synergy between health and sustainability scores using the WHO Index and the LCA sustainability score to assess combined nutritional and ecological value.[46]

The environmental impacts that were most commonly modeled were GHG emissions and use of resources such as agricultural land, energy, and water. In many studies, the environmental impact for each food/food category was obtained using the LCA method. The LCA is a standardized methodological framework for assessing the environmental impact (or load) attributable to the life cycle of a food product. The life cycle for a food typically includes agricultural production, processing and packaging, transportation, retail, use, and waste disposal.[33, 49-51] An inventory of all stages of the life cycle is determined for each food product and a “weight” or number of points is then attributed to each food or food category, based on environmental impacts such as resource extraction, land use, and relevant emissions. These environmental impact results can be translated into measures of damage done to human health, ecosystem quality, and energy resources using programs such as Eco-Indicator.[52] In addition to the health assessment approaches listed above, some studies used LCA analysis with a standardized approach to determine damages from GHG emissions and use of resources; these damage outcome included human health as an environmental damage component, such as the number and duration of diseases and life years lost due to premature death from environmental causes.

Few studies assessed food security. These studies assessed food security in terms of the cost difference between an average dietary pattern for the country studied and a sustainable dietary pattern for that population.[36, 39, 48] The basic food basket concept was used in some studies, representing household costs for a two-adult/two-child household.

Identified Dietary Patterns and Health and Sustainability Outcomes

Vegetarian and Meat-based Diets

Several studies examined variations on vegetarian diets, or a spectrum from vegan to omnivorous dietary patterns, and associated environmental outcomes.[34, 35, 37, 41] Peters et al. examined 42 different dietary patterns and land use in New York, with patterns ranging from low-fat, lacto-ovo vegetarian diets to high fat, meat-rich omnivorous diets; across this range, the diets met U.S. dietary guidelines when possible.[41] They found that, overall, increasing meat in the diet increased per capita land requirements; however, increasing total dietary fat content of low-meat diets (i.e. vegetarian alternatives) increased the land requirements compared to high-meat diets. In other words, although meat increased land requirements, diets including meat could feed more people than some higher fat vegetarian-style diets. Aston et al. assessed a pattern that was modeled on a feasible UK population in which the proportion of vegetarians in the survey was doubled, and the remainder adopted a diet pattern consistent with the lowest category of red and processed meat (RPM) consumers. They found the combination of low RPM + vegetarian diet had health benefits of lowering the risk of diabetes and colorectal cancer, determined from risk relationships for RPM and CHD, diabetes, and colorectal cancer from published meta-analyses.[53-55] Furthermore, the expected reduction in GHG for this diet was ~3 percent of current total carbon dioxide (CO_2) emissions for agriculture. De Carvalho et al. also examined a high RPM dietary pattern with diet quality assessed using the Brazilian Healthy Eating Index.[37] They found that excessive meat intake was associated not only with poorer diet quality but also with increased projected GHG emissions (~ 4 percent total CO_2 emitted by agriculture). Taken together, the results on RPM intake indicate that reduced consumption is expected to improve some health outcomes and decrease GHG emissions, as well as land use compared to current RPM consumption. Baroni et al. examined vegan, vegetarian, and omnivorous diets, both organically and conventionally grown, and found that the organically grown vegan diet had the most potential health benefits; whereas, the conventionally grown average Italian diet had the least.[37] The organically grown vegan diet also had the lowest estimated impact on resources and ecosystem quality, and the average Italian diet had the greatest projected impact. Beef was the single food with the greatest projected impact on the environment; other foods estimated to have high impact included cheese, milk, and seafood.

Vegetarian diets, dietary guidelines-related diets, and Mediterranean-style diets were variously compared with the average dietary patterns in selected countries.[38, 40, 42, 46] Overall, the estimated greater environmental benefits, including reduced projected GHG emissions and land use, resulted from vegan, lacto-ovo vegetarian, and pesco-vegetarian diets, as well as dietary guidelines-related and Mediterranean-style dietary patterns. These diets had higher overall predicted health scores than the average diet patterns. Moreover, for the most part, the high health scores of these dietary patterns were paralleled by high combined estimated sustainability scores. According to van Doreen et al., the synergy measured across vegetarian, Mediterranean-style, and dietary guidelines-related scores could be explained by a reduction in consumption of meat, dairy, extras (i.e., snacks and sweets), and beverages, as well as a reduction in overall food consumption.[42]

Mediterranean-Style Dietary Patterns

The Mediterranean-style dietary pattern was examined in both Mediterranean and non-Mediterranean countries.[44, 46] In all cases, adherence to a Mediterranean-style dietary pattern—compared to usual intake—reduced the environmental footprint, including improved GHG emissions, agricultural land use, and energy and water consumption. Both studies limited either red and processed meat[40] or meat and poultry[42] to less than 1 serving per week, and increased seafood intake. The authors concluded that adherence to a Mediterranean-style dietary pattern would make a significant contribution to increasing food sustainability, as well as increasing the health benefits that are well-documented for this type of diet (see ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***).

Diet Scenarios

Other studies examined different diet "scenarios" that generally replaced animal foods in various ways with plant foods.[43, 45, 47] Scarborough et al. found that a diet with 50 percent reduced total meat and dairy replaced by fruit, vegetables, and cereals contributed the most to estimated reduced risk of total mortality and also had the largest potential positive environmental impact.[13] This diet scenario increased fruit and vegetable consumption by 63 percent and decreased saturated fat and salt consumption; micronutrient intake was generally similar with the exception of a drop in vitamin B_{12}.

Pradhan et al. examined 16 global dietary patterns that differed by food and energy content, grouped into four categories with per capita intake of low, moderate, high, and very high kcal diets. They assessed the relationship of these patterns to GHG emissions.[43] Low-energy diets had less than 2,100 kcal/cap/day and were composed of more than 50 percent cereals or more than 70 percent starchy roots, cereals, and pulses. Animal products were minor in this group (<10 percent). Moderate, high, and very high energy diets had 2,100-2,400, 2,400-2,800, and greater than 2,800 kcal/cap/day, respectively. Very high calorie diets had high amounts of meat and alcoholic beverages. Overall, very high calorie diets, common in the developed world, exhibited high total per capita CO_{2eq} emissions due to high carbon intensity and high intake of animal products; the low-energy diets, on the other hand, had the lowest total per capita CO_{2eq} emissions.

Lastly, Vieux et al. examined dietary patterns with different indicators of nutritional quality and found that despite containing large amounts of plant foods, not all diets of the highest nutritional quality were those with the lowest GHG emissions.[47] For this study, the diet pattern was assessed by using nutrient-based indicators; high quality diets had energy density below the median, mean adequacy ratio above the median, and a mean excess ratio (percentage of maximum recommended for nutrients that should be limited – saturated fat, sodium, and free sugars) below the median. Four diet patterns were identified based on compliance with these properties to generate one high quality diet, two intermediate quality diets, and one low quality diet. In this study, the high quality diets had higher GHG emissions than did the low quality diets. Regarding the food groups, a higher consumption of starches, sweets and salted snacks, and fats was associated with lower diet-related GHG emissions and an increased intake of fruit and vegetables, was associated with increased diet-related GHG emissions. However, the strongest positive association with GHG emissions was still for the ruminant meat group. Overall, this study used a different approach from the other studies in this review, as nutritional quality determined the formation of dietary pattern categories.

Sustainable Diets and Costs

Three studies examined sustainable diets and related costs.[36, 39, 48] Barosh et al. examined food availability and cost of a health and sustainability (H&S) food basket, developed according to the principles of the Australian dietary guidelines as well as environmental impact.[36] The food basket approach is a commonly used method for assessing and monitoring food availability and cost. The typical food basket was based on average weekly food purchases of a reference household made up of two adults and two children. For the H&S basket, food choices were based on health principles and environmental impact. The H&S basket was compared to the typical Australian basket and it was determined that the cost of the H&S basket was more than the typical basket in five socioeconomic areas; the most disadvantaged spent 30 percent more for the H&S basket. The authors concluded that the most disadvantaged groups at both neighborhood and household levels experienced the greatest inequality in accessing an affordable H&S basket. Macdiarmid et al. examined a sustainable diet (met all energy and nutrient needs and maximally decreased GHG emissions), a “sustainable with acceptability constraints” diet (added foods commonly consumed in the UK; met energy, nutrient, and seafood recommendations as well as recommended minimum intakes for fruits and vegetables and did not exceed the maximum recommended for red and processed meat), and the average UK diet.[7] They found that the sustainable diet that was generated would decrease GHG emissions from primary production (up to distribution) by 90 percent, but consisted of only seven foods. The acceptability constraints diet included 52 foods and was projected to reduce GHG emissions by 36 percent. This diet included meat and dairy but less than the average UK diet. The cost of the sustainable + acceptability diet was comparable to that of the average UK diet. These results showed that a sustainable diet that meets dietary requirements and has lower GHG can be achieved without eliminating meat or dairy products completely, or increasing the cost to the consumer. Lastly, Wilson et al. examined 16 dietary patterns modeled to determine which patterns would minimize estimated risk of chronic disease, cost, and GHG emissions.[48] These patterns included low-cost and low-cost + low GHG diet patterns, as well as healthy patterns with high vegetable intakes including Mediterranean or Asian patterns, as well as the average New Zealand pattern. The authors found that diets that aimed to minimize cost and estimated GHG emissions also had health advantages, such as the simplified low-cost Mediterranean-style and simplified Asian-style diets, both of which would lower cardiovascular disease and cancer risk, compared to the average New Zealand diet. However, dietary variety was limited and further optimization to lower GHG emissions increased cost.

Overall, the studies were consistent in showing that higher consumption of animal-based foods was associated with higher estimated environmental impact, whereas consumption of more plant-based foods as part of a lower meat-based or vegetarian-style dietary pattern was associated with estimated lower environmental impact compared to higher meat or non-plant-based dietary patterns. Related to this, the total energy content of the diet was also associated with estimated environmental impact and higher energy diets had a larger estimated impact. For example, for fossil fuel alone, one calorie from beef or milk requires 40 or 14 calories of fuel, respectively, whereas one calorie from grains can be obtained from 2.2 calories of fuel.[42] Additionally, the evidence showed that dietary patterns that promote health also promote sustainability; dietary patterns that adhered to dietary guidelines were more environmentally sustainable than the population's current average level of intake or pattern. Taken together, the studies agreed on the environmental impact of different dietary patterns, despite varied methods of assessing environmental impact and differences in components of environmental impact assessed (e.g. GHG emissions or land use). The evidence on whether sustainable diets were more or less expensive than typically consumed diets in some locations was limited and inconsistent.

Three additional reports on the relationship between dietary patterns and sustainability were published after this systematic review was completed. Two of these reports were consistent with, and provided more evidence to support the Committee's findings that dietary guidelines-related diets, Mediterranean-style diets, and vegetarian (and variations) diets are associated with improved environmental outcomes. Tilman and Clark showed that following a Mediterranean, vegetarian (lacto-ovo), or pesco-vegetarian dietary pattern would decrease both current and projected GHG emissions and land use.[11] Eshel et al. reported on the five main animal-based categories in the U.S. diet – dairy, beef, poultry, pork, and eggs – and their required feeds including crops, byproducts, and pasture. They found that beef production required more land and irrigation water and produced more GHG emissions than dairy, poultry, pork, or eggs.[9] In addition, as a standard comparator, staple plant foods had lower land use and GHG emissions than did dairy, poultry, pork, or eggs. In contrast, a report from Heller and Keoleian suggests that an isocaloric shift from the average U.S. diet (at current U.S. per capita intake of 2,534 kcals/day from Loss-Adjusted Food Availability (LAFA) data) to a pattern that adheres to the *2010 Dietary Guidelines for Americans* would result in a 12 percent increase in diet-related GHG emissions.[10] This result was modified, however, by their finding that if Americans consumed the recommended pattern within the recommended calorie intake level of 2,000 kcal/day, there would be a 1 percent *decrease* in GHG emissions. This finding reinforces the overriding 2010 DGA recommendation that all of the guidelines need to be followed, including appropriate calorie intake levels for age, gender, and activity level. Furthermore, in contrast to the findings of

Eshel et al. regarding dairy, Heller and Keoleian suggest that increases in dairy to follow 2010 DGA recommendations contribute significantly to increased GHG emissions and counters the modeled benefits of decreased meat consumption.[10]

For additional details on this body of evidence, visit: Appendix E-2.37

Seafood Sustainability

Background

Seafood is recognized as an important source of key macro- and micronutrients. The health benefits of seafood, including support of optimal neurodevelopment and prevention of cardiovascular disease, are likely due in large part to long-chain n-3 polyunsaturated fatty acids (PUFA), docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), although seafood also are good sources of other nutrients including protein, selenium, iodine, vitamin D, and choline.[27] Currently, seafood production is in the midst of rapid expansion to meet growing worldwide demand, but the collapse of some fisheries due to overfishing in past decades raises concerns about the ability to produce safe and affordable seafood to supply the U.S. population and meet current dietary intake recommendations of at least 8 ounces per week.[20, 56] Capture fisheries (wild caught) production has leveled-off as a proportion of fully exploited stocks, and this is due in part to national and international efforts on seafood sustainably (e.g., the U.S. Magnuson-Stevens Fishery Conservation and Management Act (2006) mandating annual catch limits, managed by the U.S. National Oceanographic and Atmospheric Administration). In contrast, the increased productivity of worldwide aquaculture (farm-raised) is expected to continue and will play a major role in expanding the supply of seafood.[20] Expanding farm-raised seafood has the potential to ensure sufficient amounts of seafood to allow the U.S. population to consume levels recommended by dietary guidelines.[57] Productivity gains should be implemented in a sustainable manner with greater attention to maintaining or enhancing the high nutrient density characteristic of captured seafood. Consistent with overall sustainability goals, farm-raised finfish (e.g., salmon and trout) is more sustainable than terrestrial animal production (e.g., beef and pork) in terms of GHG emissions and land/water use.[58, 59] Currently, the United States imports the majority of its seafood (~90 percent), and approximately half of that is farmed.[60] The major groups commonly referred to as finfish, shellfish, and crustaceans include more than 500 species, and thus, generalizations to all seafood must be made with caution.

Question 2: What are the comparative nutrient profiles of current farm-raised versus wild caught seafood?

Source of evidence: USDA Agriculture Research Service (ARS) National Nutrient Database (NND)[25] updated with USDA-funded survey of most commonly consumed species in the United States.[26]

Conclusion

For commonly consumed fish species in the United States, such as bass, cod, trout, and salmon, farmed-raised seafood has as much or more of the omega-3 fatty acids EPA and DHA as the same species captured in the wild. In contrast, farmed low-trophic species, such as catfish and crawfish, have less than half the EPA and DHA per serving than wild caught, and these species have lower EPA and DHA regardless of source than do salmon. Farm-raised seafood has higher total fat than wild caught. Recommended amounts of EPA and DHA can be obtained by consuming a variety of farm-raised seafood, especially high-trophic species, such as salmon and trout.

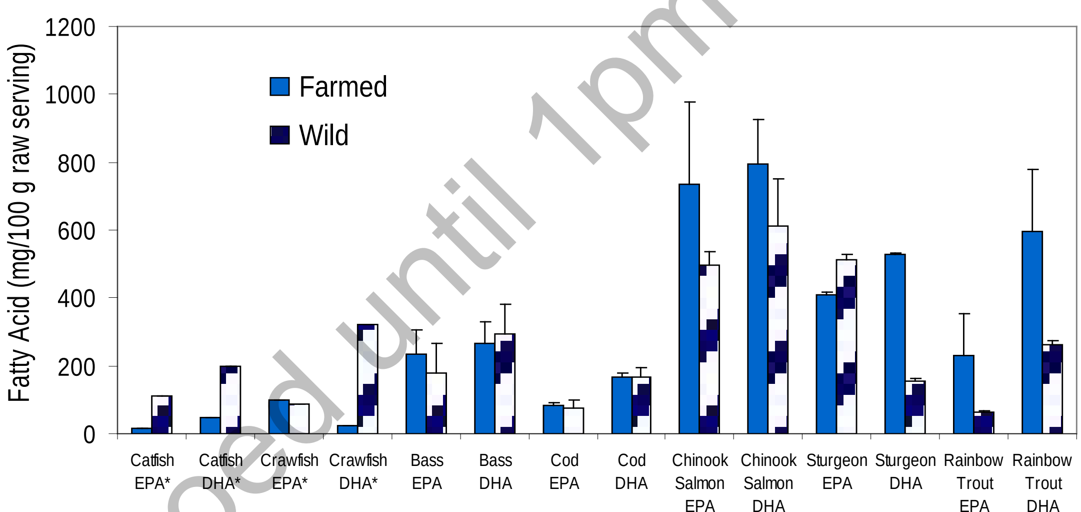
Implications

The U.S. population should be encouraged to eat a wide variety of seafood that can be wild caught or farmed, as they are nutrient-dense foods that are uniquely rich sources of healthy fatty acids. It should be noted that low-trophic farm-raised seafood, such as catfish and crayfish, have lower EPA and DHA levels than do wild-caught. Nutrient profiles in popular low-trophic farmed species should be improved through feeding and processing systems that produce and preserve nutrients similar to those of wild-caught seafood of the same species.

Review of the Evidence

The USDA-Agricultural Research Service (ARS) National Nutrient Database (NND) for Standard Reference, Release 27 was used to address this question (http://www.ars.usda.gov/ba/bhnrc/ndl).[25] The section on finfish and shellfish products included nutrient profiles for both farm-raised and wild-caught seafood for some species. These data were augmented using a USDA-funded report on fatty-acid profiles of commercially available fish in the United States that assessed additional farmed species and compared results[26] with the USDA-ARS NND.[25] The samples collected were from different regions of the United States during different seasons. For wild-caught species, the nutrient profile is determined by changes in environmental conditions, whereas, for farmed species, the nutrient profile is dependent on the amount, timing, and composition of the feed.[26] Because aquaculture diets can be continually modified, updates are important to monitor EPA and DHA in commercial seafood species, to provide consumers with the most accurate information. The NND provided nutrient profiles for six seafood species with data on both wild-caught and farm-raised versions: four fish (rainbow trout, Atlantic and Coho salmon, and catfish), eastern oysters, and mixed species crayfish. The key nutrients EPA and DHA were on average comparable or greater for farmed trout, salmon, and oysters compared to wild capture, reflecting the higher total fat content of these farmed species. On the other hand, low-trophic species, such as catfish and crayfish, when farmed, were lower in EPA and DHA compared to wild capture. Cladis et al. determined EPA and DHA levels for five farmed and wild fish species (rainbow trout, white sturgeon, Chinook salmon, Atlantic cod, striped bass), providing an update and comparison for some of these species (Figure D5.2)[26]. Farmed Atlantic salmon was similar between the NND and the update and most other species compared well; however, Chinook salmon and sturgeon showed differences in EPA and DHA content (although farmed and wild were not distinguished in the NND). Overall, these data showed that existing DGAC recommendations to consume a variety of seafood can be met by consuming a diverse range of species, including farmed species.

Figure D5.2. Comparison of EPA and DHA drawn from data in USDA National Nutrient Database[25] and update from Cladis et al.[26]



For additional details on this body of evidence, visit: Appendix E-2.38 Evidence Portfolio and http://www.ars.usda.gov/ba/bhnrc/ndl

Question 3. What are the comparative contaminant levels of current farm-raised versus wild caught seafood?

Source of evidence: Report of the Joint United Nations Food and Agriculture Organization/World Health Organization Expert Consultation on the Risks and Benefits of Fish Consumption. Rome, 25–29 January 2010. FAO Fisheries and Aquaculture Report No. 978.[27]

Conclusion

The DGAC concurs with the Consultancy that, for the majority of commercial wild and farmed species, neither the risks of mercury nor organic pollutants outweigh the health benefits of seafood consumption, such as decreased cardiovascular disease risk and improved infant neurodevelopment. However, any assessment evaluates evidence within a time frame and contaminant composition can change rapidly based on the contamination conditions at the location of wild catch and altered production practices for farmed seafood. **DGAC Grade: Moderate**

Implications

Based on risk/benefit comparisons, either farmed or wild-caught seafood are appropriate choices to consume to meet current Dietary Guidelines for Americans for increased seafood consumption. The DGAC supports the current FDA and EPA recommendations that women who are pregnant (or those who may become pregnant) and breastfeeding should not eat certain types of seafood—tilefish, shark, swordfish, and king mackerel—because of their high methyl mercury contents. Attention should be paid to local seafood advisories when eating seafood caught from local rivers, streams, and lakes.

Based on the most current evidence on mercury levels in albacore tuna provided in the Report of the Joint United Nations Food and Agriculture Organization/World Health Organization Expert Consultation on the Risks and Benefits of Fish Consumption, 2010,[27] the DGAC recommends that the EPA and FDA re-evaluate their current recommendations[61] for women who are pregnant (or for women who may become pregnant) or breastfeeding to limit white albacore tuna to not more than 6 ounces a week.

Review of the Evidence

The Report of the FAO/WHO Expert Consultation on the Risks and Benefits of Fish Consumption[27] was used to address this question. This report was chosen as the most current and comprehensive source on contaminants in wild-caught and farm-raised seafood, and the DGAC focused on data that addressed the specific comparison between the two. The sections of the report that were used to address the question were "Data on the composition of fish" and "Risk-benefit comparisons." The consultancy took a net effects approach, balancing benefits of seafood, especially benefits associated with EPA and DHA, against the adverse effects of mercury and persistent organic pollutants (POPs), including polychlorinated biphenyls, polychlorinated dibenzo-*p*-dioxins, and polychlorinated dibenzofurans, collectively referred to as dioxins. The Expert Consultancy compiled EPA and DHA, mercury, and dioxins compositional data from national databases of the United States, France, Norway, and Japan, as well as an international database. Together, these provided information on total fat, EPA and DHA, total mercury, and dioxins for a large number of seafood species, including three farmed and wild species (salmon, rainbow trout, and halibut). Two specific outcomes were considered for risk/benefit: 1) prenatal exposure and offspring neurodevelopment, and 2) mortality from cardiovascular diseases and cancer.

Overall, for the species examined, levels of mercury and dioxins were in the same range for farmed and wild seafood. Related to risk/benefit, at the same level of mercury content (lowest [≤ 0.1 μg/g] and 2nd lowest [0.1 - 0.5 μg/g] levels), farmed seafood had the same or higher levels of EPA and DHA as wild-caught. At the same level of dioxin content (2nd lowest [0.5 – 4 pg toxic equivalents (TEQ)/g] level), farmed seafood had the same or higher levels of EPA and DHA as wild-caught. Only wild-caught Pacific salmon had the lowest level of dioxins (<0.5 pg TEQ/g). Overall, the quantitative risk/benefit analysis was not different for farmed compared to wild-caught seafood. For both, using the central estimate for benefits of DHA and for harm from mercury, the neurodevelopmental risks of not eating seafood exceeded the risks of eating seafood. Similarly, for coronary heart disease (CHD) in adults, there were CHD mortality benefits from eating seafood and CHD risks from not eating seafood, except for seafood in the highest dioxin category and lowest EPA and DHA category, which did not include any of the farm-raised species considered.

Albacore tuna, produced only from wild marine fisheries, is a special case of a popular fish highlighted by the 2004 FDA and EPA advisory.[61, 62] For all levels of intake including more than double the 12 ounces per week recommendation, all evidence was in favor of net benefits for infant development and CHD risk reduction.

Limitations in the evidence included the small number of farmed and wild seafood species comparisons considered by the Expert Consultancy, and the possibility of rapid change that may occur in the concentration of contaminants locally. In addition, seafood contaminants are closely linked to levels of contaminants in feed.

For additional details on this body of evidence, visit: *Report of the Joint Food and Agriculture Organization of the United Nations (FAO) and the World Health Organization (WHO) Expert Consultation on the Risks and Benefits of Fish Consumption, 2011*. Available at http://www.fao.org/docrep/014/ba0136e/ba0136e00.pdf

Question 4: What is the worldwide capacity to produce farm-raised versus wild-caught seafood that is nutritious and safe for the U.S. population?

Source of evidence: United National (UN) Food and Agriculture Organization (FAO) report on *The State of World Fisheries and Agriculture.*[20]

Conclusions

The DGAC concurs with the FAO report that consistent evidence demonstrates that capture fisheries increasingly managed in a sustainable way have remained stable over several decades. However, on average, capture fisheries are fully exploited and their continuing productivity relies on careful management to avoid over-exploitation and long-term collapse. **DGAC Grade: Strong**

The DGAC endorses the FAO report that capture fisheries production plateaued around 1990 while aquaculture has increased since that time to meet increasing demand. Evidence suggests that expanded seafood production will rely on the continuation of a rapid increase in aquaculture output worldwide, projected at 33 percent increase by 2021, which will add 15 percent to the total supply of seafood.[20] Distributed evenly to the world's population, this capacity could in principle meet Dietary Guidelines recommendations for consumption of at least 8 ounces of seafood per week. Concern exists that the expanded capacity may be for low-trophic level seafood that has relatively low levels of EPA and DHA compared to other species. Under the current production, Americans who seek to meet U.S. Dietary Guidelines recommendations must rely on significant amounts of imported seafood (~90 percent). **DGAC Grade: Moderate**

Implications

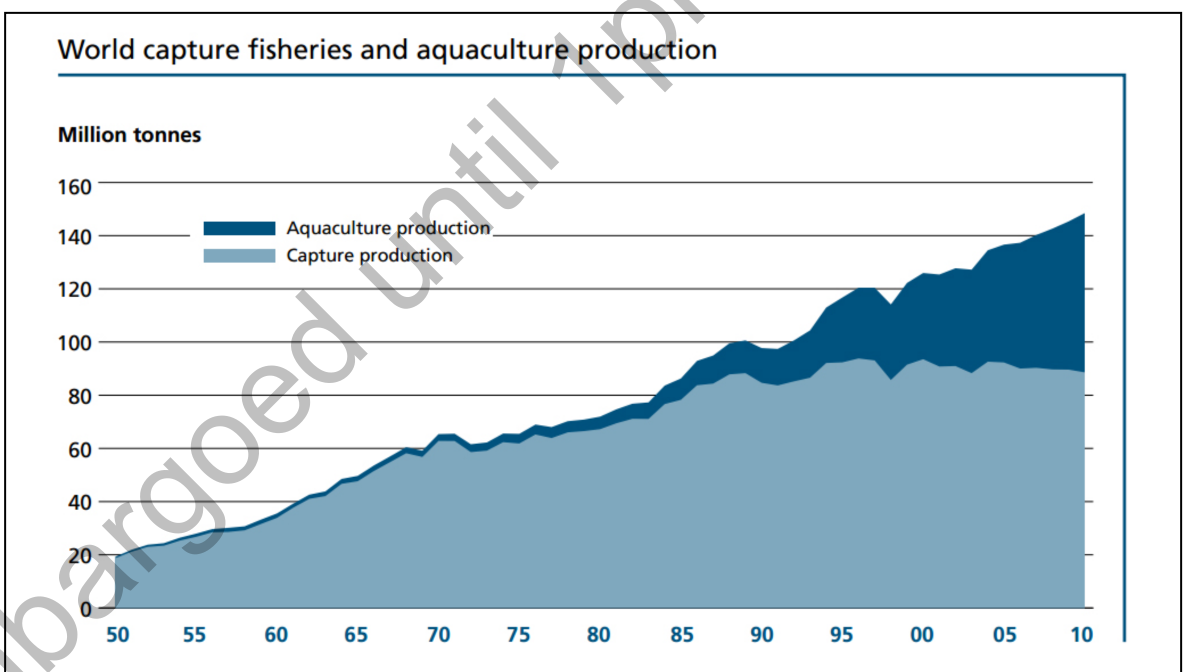
Both wild and farmed seafood are major food sources available to support DGAC recommendations to regularly consume a variety of seafood. Responsible stewardship over environmental impact will be important as farmed seafood production expands. Availability of these important foods is critical for future generations of Americans to meet their needs for a healthy diet. Therefore, strong policy, research, and stewardship support are needed to increasingly improve the environmental sustainability of farmed seafood systems. From the standpoint of the dietary guidelines this expanded production needs to be largely in EPA and DHA rich species and supporting production of low-trophic level species of similar nutrient density as wild-caught.

Review of the Evidence

The UN FAO report on *The State of World Fisheries and Agriculture* issued in 2012 formed the basis of the DGAC's evidence review on this topic.[20] The FAO report addresses a wide variety of issues affecting capture fisheries and aquaculture, including economics, infrastructure, and labor and government policies. The DGAC focused on matters that directly address the world production of one important food—seafood—as a first attempt by a DGAC committee to consider the implications of dietary guidelines for production of a related group of foods.

The production of capture fisheries has remained stable at about 90 million tons from 1990-2011 (Figure D5.3).[20] At the same time, aquaculture production is rising and will continue to increase. FAO model projections indicate that in response to the higher demand for seafood, world fisheries and aquaculture production is projected to grow by 15 percent between 2011 and 2021. This increase will be mainly due to increased aquaculture output, which is projected to increase 33 percent by 2021, compared with only 3 percent growth in wild capture fisheries over the same period. It is predicted that aquaculture will remain one of the fastest growing animal food-producing sectors and will exceed that of beef, pork, or poultry. Aquaculture production is expected to expand on all continents with variations across countries and regions in terms of the seafood species produced. Currently, the United States is the leading importer of seafood products world-wide, with imports making up about 90 percent of seafood consumption. Continuing to meet Americans needs for seafood will require stable importation or substantial expansion of domestic aquaculture.

Figure D5.3. Comparison of fishery production and aquaculture, 1950-2010



For additional details on this body of evidence, visit: UN FAO report on *The State of World Fisheries and Agriculture, 2012.* Available at http://www.fao.org/fishery/sofia/en

FOOD SAFETY

The DGAC reviewed evidence of food safety topics was limited to usual coffee/caffeine consumption, high dose caffeine consumption, and aspartame. Coffee is one of the most widely consumed beverages in the U.S. and represents a major source of caffeine.[63] The effects of coffee/caffeine consumption have not been evaluated by any prior DGAC. The Committee reviewed the evidence on normal and excessive coffee/caffeine intake and health outcomes. In addition, the DGAC reviewed evidence on health outcomes and aspartame; the most widely used nonnutritive sweetener.

Given the importance of food-borne illness prevention, the Committee reviewed the 2010 DGAC report content related to consumer behaviors and updated the key food safety behavior principles.

Question 5: What is the relationship between usual coffee/caffeine consumption and health?

Source of Evidence: Overview of systematic reviews and meta-analyses

<u>Coffee/Caffeine and Chronic Disease</u>

Conclusion

Strong and consistent evidence shows that consumption of coffee within the moderate range (3 to 5 cups/d or up to 400 mg/d caffeine) is not associated with increased risk of major chronic diseases, such as cardiovascular disease (CVD) and cancer and premature death in healthy adults. **DGAC Grade: Strong**

Consistent observational evidence indicates that moderate coffee consumption is associated with reduced risk of type 2 diabetes and cardiovascular disease in healthy adults. In addition, consistent observational evidence indicates that regular consumption of coffee is associated with reduced risk of cancer of the liver and endometrium, and slightly inverse or null associations are observed for other cancer sites. **DGAC Grade: Moderate**

Implications

Moderate coffee consumption can be incorporated into a healthy lifestyle, along with other behaviors, such as refraining from smoking, consuming a nutritionally balanced diet, maintaining a healthy body weight, and being physically active. However, it should be noted that coffee, as it is normally consumed, frequently contains added calories from cream, milk, and added sugars. Care should be taken to minimize these caloric additions. Furthermore, individuals who do not consume caffeinated coffee should not start to consume it for health benefits alone.

Review of the Evidence

Total Mortality

Evidence suggests a significant inverse relationship between coffee consumption of 1 to 4 cups/day with total mortality, especially CVD mortality. This evidence is based on three meta-analyses of more than 20 prospective cohort studies.[64-66] In general, results were similar for men and women. The risk reduction associated with each cup of coffee per day was between 3 to 4 percent. In addition, Je and Giovannucci found a significant inverse association between coffee consumption and CVD mortality.[65] This association was stronger in women (16 percent lower risk) than in men (8 percent lower risk). However, no association was found for cancer mortality. Crippa et al. found that the lowest risk was observed for 4 cups/day for all-cause mortality (16%, 95% CI: 13, 18) and 3 cups/day for CVD mortality (21%, 95% CI: 16, 26).[64]

Cardiovascular Disease

A large and current body of evidence directly addressed the relationship between normal coffee consumption and risk of CVD. The evidence included 12 systematic reviews with meta-analyses, all of which had high quality ratings (AMSTAR scores 8/11 – 11/11). CVD incidence and mortality, as well as CHD, stroke, heart failure, and hypertension were assessed by meta-analyses that consisted primarily of prospective cohort studies. Intermediate outcomes such as blood pressure, blood lipids, and blood glucose were assessed by meta-analyses of randomized controlled trials.

CVD risk was assessed by a current meta-analysis of 36 prospective cohort studies on long-term coffee consumption.[67] This analysis showed a non-linear association, such that the lowest risk of CVD was seen with moderate coffee consumption (3 to 5 cups/day), but higher intakes (>5 cups/day) were neither protective nor harmful. Overall, moderate consumption of caffeinated, but not decaffeinated, coffee was associated with a 12 percent lower risk of CVD.

Results from the assessment of CHD risk in three meta-analyses were not entirely consistent.[67-69] Ding et al. found 10 percent lower CHD risk with moderate coffee consumption (3 to 5 cups/day) in a meta-analysis of 30 prospective cohort studies, whereas Wu et al. and Sofi et al. in meta-analyses of 21 and 10 prospective cohort studies, respectively, found no association between coffee consumption and CHD risk. [67-69] However, in sub-group analysis, Wu et al. found that habitual moderate coffee consumption (1 to 4 cups/day) was associated with an 18 percent lower risk of CHD among women.[69] Overall, the meta-analyses of Sofi et al. and Wu et al. were conducted with smaller bodies of evidence and Ding et al. assessed several more recent studies.[67-69] Of note, coffee brewing methods have changed over time and the filter method has become more widely used, replacing unfiltered forms of coffee such as boiled coffee that were more widely reported by participants in earlier studies. Thus, the findings by Ding et al. are more up to date, reflecting health effects of coffee consumed in recent cohorts.

Risk of stroke was assessed in two systematic reviews with meta-analyses of prospective cohort studies with consistent findings.[70, 71] Kim et al. found that coffee intake of 4 or more cups/day had a protective association on risk of stroke.[70] Larsson et al. documented a non-linear association such that coffee consumption ranging from 1 to 6 cups/day was associated with an 8 percent to 13 percent lower risk of stroke, and higher intakes were not associated with decreased or increased risk.[71] The inverse associations were limited to ischemic stroke and no association was seen with hemorrhagic stroke.

Regarding blood pressure, three meta-analyses evaluated the effect of coffee and caffeine on systolic and diastolic blood pressure using controlled trials.[72-74] The most recent meta-analysis of 10 randomized controlled trials by Steffen et al. showed no effect of coffee on either systolic or diastolic blood pressure. Similarly, in another meta-analysis of 11 coffee trials and 5 caffeine trials, caffeine doses of <410 mg/day had no effect on systolic and diastolic blood pressure, while doses of 410 or more mg/day resulted in a net increase.[73] A third meta-analysis showed that among individuals with hypertension, 200 to 300 mg of caffeine (equivalent to ~2 to 3 cups filtered coffee) resulted in an acute increase of systolic and diastolic blood pressure.[72] Additionally, two meta-analyses quantified the effect of coffee on incidence of hypertension[74, 75] and found no association between habitual coffee consumption and risk of hypertension. However, Zhang et al. documented a slightly elevated risk for light to moderate consumption (1 to 3 cups/day) of coffee compared to less than 1 cup/day.[75]

Regarding blood lipids, meta-analyses of short-term randomized controlled trials revealed that coffee consumption contributed significantly to an increase in total cholesterol and LDL-cholesterol, but cholesterol-raising effects were primarily limited to unfiltered coffee and filtered coffee appeared to have minimal effects on serum cholesterol levels.[76, 77]

In a meta-analysis of observational study data, including prospective, retrospective, and case-control studies, higher amounts of coffee or caffeine had no association with risk of atrial fibrillation, but low doses of caffeine (<350 mg/day) appeared to have a protective association.[78] In addition, coffee consumption of 1 to 5 cups/day was found to be inversely associated with risk of heart failure in a meta-analysis of five prospective studies.[79] A non-linear association was documented and the lowest risk was observed for 4 cups/day.[79]

Type 2 Diabetes

Coffee consumption has consistently been associated with a reduced risk of type 2 diabetes. In four meta-analyses of prospective cohort studies[80-83] and cross-sectional studies,[83] coffee consumption was inversely associated with risk of type 2 diabetes in a dose-response manner. Compared to non-drinkers, risk for type 2 diabetes was 33 percent lower for those consuming 6 cups/day in the analysis by Ding et al. while the risk was 37 percent lower for those consuming 10 cups/day in the analysis by Jiang et al.[67, 82] Using a sub-set of the prospective cohorts in the

Ding et al. and Jiang et al. meta-analyses, Huxley et al. documented that each cup of coffee was associated with a 7 percent lower risk of type 2 diabetes.[81] Similarly, van Dam and Hu noted that consumption of ≥6 or ≥7 cups/day was associated with a 35 percent lower risk of type 2 diabetes.[83] Three meta-analyses[80-82] also found protective associations for decaffeinated coffee. Moderate decaffeinated coffee consumption (3 to 4 cups/day) was associated with a 36 percent lower risk of type 2 diabetes.[81] Each cup of decaffeinated coffee was associated with a 6 percent lower risk[80] while every 2 cups were associated with a 11 percent lower risk.[82] Both reports also documented a dose-response association between caffeine and type 2 diabetes risk such that every 140 mg/day was associated with an 8 percent lower risk in the Ding et al. meta-analysis, while every 200 mg/day was associated with a 14 percent lower risk in the analysis by Jiang et al.[80, 82] However, it remains unclear if this inverse association is independent of coffee consumption, as Ding et al. indicated that none of the studies included in the caffeine dose-response analysis adjusted for total coffee.

Only one systematic review of nine randomized controlled trials examined the effects of caffeine on blood glucose and insulin concentrations among those with type 2 diabetes.[84] Ingestion of 200 to 500 mg of caffeine acutely increased blood glucose concentrations by 16 to 28 percent of the area under the curve and insulin secretions by 19 to 48 percent of the area under the curve when taken before a glucose load. At the same time, these trials also noted a decrease in insulin sensitivity by 14 to 37 percent. Although no study has examined whether the effects of caffeine on blood glucose and insulin persist in the long term, evidence from prospective cohorts indicates that the acute effects of caffeine do not translate into long-term risk of type 2 diabetes. Furthermore, the inverse association between decaffeinated coffee and diabetes risk suggests that the observed benefit is likely to be due to other constituents in coffee rather than caffeine.

Cancer

Several systematic reviews and meta-analyses examined the association between coffee consumption and risk of cancer. Types of cancer examined by the DGAC included total cancer, cancers of the lung, liver, breast, prostate, ovaries, endometrium, bladder, pancreas, upper digestive and respiratory tract, esophagus, stomach, colon, and rectum.

In a quantitative summary of 40 prospective cohort studies with an average follow-up of 14.3 years, Yu et al. found a 13 percent lower risk of total cancer among coffee drinkers compared to non-drinkers or those with lowest intakes.[85] Risk estimates were similar for men and women. In sub-group analyses, the authors noted that coffee drinking was associated with a reduced risk of bladder, breast, buccal and pharyngeal, colorectal, endometrial, esophageal, hepatocellular, leukemic, pancreatic, and prostate cancers.

Tang et al. evaluated five prospective cohorts and eight case-control studies and found that, overall, those with the highest levels of coffee consumption had a 27 percent higher risk for lung cancer compared to never drinkers or those with least consumption.[86] An increase in coffee consumption of 2 cups/day was associated with a 14 percent higher risk of developing lung cancer. However, because smoking is an important confounder, when analyses were stratified by smoking status, coffee consumption was marginally protective in non-smokers and was not associated with lung cancer among smokers. When estimates from two studies that examined decaffeinated coffee were summarized, a protective association with lung cancer was seen. No association was seen with lung cancer when only case-control studies were considered.

Results from two meta-analyses indicate that coffee consumption is associated with a 40 to 50 percent lower risk of liver cancer,[87 88] when considering both cohort and case-control studies. In one meta-analysis, the associations were significant in men but not in women.[87]

Three meta-analyses of observational studies found no association between coffee consumption,[89-91] caffeine consumption, or decaffeinated coffee consumption and risk of breast cancer. In all three reports, each 2 cup/day of coffee was marginally associated with a 2 percent lower risk of breast cancer. However, in sub-group analyses, coffee consumption was protective against breast cancer risk in postmenopausal women,[89] BRCA1 mutation carriers,[89] and women with estrogen receptor negative breast tumors .[90]

The association between coffee consumption and risk of prostate cancer was mixed. Cao et al. and Zhong et al. found that regular or high coffee consumption, compared to non- or lowest levels of consumption, was associated with a 12 percent to 17 percent lower risk of prostate cancer in prospective cohort studies.[92, 93] Further, each 2 cups of coffee per day was associated with a 7 percent lower risk of prostate cancer. However, no associations were seen with case-control data alone or when these studies were examined together with prospective cohort studies. Using a combination of both prospective cohort and case-control data, Discacciati et al. found that each 3 cups/day of coffee was associated with a 3 percent lower risk of localized prostate cancer and an 11 percent lower risk of mortality from prostate cancer.[94] On the other hand, after summarizing data from 12 prospective cohort and case-control studies, Park et al. found a 16 percent higher risk of prostate cancer.[95] However, in sub-group analyses by study design, the higher risk was observed in case-control but not in cohort studies.

Consumption of coffee was not associated with risk of ovarian cancer in a meta-analysis of seven prospective cohort studies with more than 640,000 participants.[96] Two meta-analyses confirmed an inverse association between coffee consumption and risk of endometrial cancer.[97, 98] In the most recent and updated meta-analysis of prospective cohort and case-control studies, compared to those in the lowest category of coffee consumption, those with the highest intakes of coffee had a 29 percent lower risk of endometrial cancer.[98] Each cup of coffee per day was associated with an 8 percent lower risk of endometrial cancer. Similar results were found in the meta-analysis by Bravi et al. that included a sub-set of the studies in Je et al. and documented a 20 percent lower risk of endometrial cancer overall, and a 7 percent decrease for each cup of coffee per day.[97, 98] However, the association was significant only in case-control studies but not in cohort studies, most likely due to lower statistical power.

A recent meta-analysis of 23 case-control studies by Zhou et al. found coffee was a risk factor for bladder cancer. There was a smoking-adjusted increased risk of bladder cancer for those in the highest (45 percent), second highest, (21 percent), and third highest (8 percent) groups of coffee consumption, compared to those in the lowest intake group.[99] No association was, however, seen in cohort studies.

Two meta-analyses of coffee consumption and pancreatic cancer risk provided mixed results.[85, 100] Using both prospective cohort and case-control studies, Turati et al. found that coffee consumption was not associated with risk of pancreatic cancer.[100] However, an increased risk was seen in case-control studies that did not adjust for smoking. Using a sub-set of prospective cohorts included in the Turati et al. meta-analysis, Dong et al. found that coffee drinking was inversely associated with pancreatic cancer risk but did not separate studies based on their adjustment for smoking status.[101] Sub-group analyses revealed a protective association in men, but not in women.

Turati et al. quantified the association between coffee consumption and various upper digestive and respiratory tract cancers using data from observational studies.[102] Coffee consumption was associated with a 36 percent lower risk of oral and pharyngeal cancer but not with risk of laryngeal cancer, esophageal squamous cell carcinoma, or esophageal adenocarcinoma. In a meta-analysis of prospective cohort and case-control studies, Zheng et al. noted that coffee was inversely, but non-significantly, associated with risk of esophageal cancer.[103] Regarding gastric cancer, no association between coffee consumption and risk was seen in a meta-analysis of observational studies by Botelho et al.[104]

Three meta-analyses on the association between coffee consumption and colorectal cancer risk have yielded mixed findings.[105-107] Results from case-control studies suggested coffee consumption was associated with lower risk of colorectal (15 percent lower) and colon cancer (21 percent lower), especially in women. However, this inverse association was non-significant for cohort studies. Using all but one of the case-control studies, Galeone et al. arrived at similar conclusions as a Li et al. analysis, although associations were in general stronger.[105, 107] Galeone et al. also provided suggestive evidence for a dose-response relationship between coffee and colorectal cancer such that each cup of coffee was associated with a 6 percent lower risk of colorectal cancer, 5 percent lower risk of colon cancer, and 3 percent lower risk of rectal cancer.[105] Using several prospective cohort studies, as in the Li et al. meta-analysis, Je et al. found no significant association of coffee consumption with risk of colorectal cancer.[106, 107] Interestingly, no differences were seen by sex but the suggestive inverse associations were slightly stronger in studies that adjusted for smoking and alcohol.

For additional details on this body of evidence, visit: Appendix E-2.39a Evidence Portfolio, Appendix E-2.39b Systematic Review/Meta-Analysis Data Table, and References 64-107

Caffeine and Neurodegenerative Disease

Conclusion

Consistent evidence indicates an inverse association between caffeine intake and risk of Parkinson's disease. **DGAC Grade: Moderate**

Limited evidence indicates that caffeine consumption is associated with a modestly lower risk of cognitive decline or impairment and lower risk of Alzheimer's disease. **DGAC Grade: Limited**

Implications

Moderate coffee consumption can be incorporated into a healthy lifestyle, along with other behaviors, such as refraining from smoking, consuming a nutritionally balanced diet, maintaining a healthy body weight, and being physically active. However, it should be noted that coffee as it is normally consumed can contain added calories from cream, milk, and added sugars. Care should be taken to minimize these caloric additions. Furthermore, individuals who do not consume caffeinated coffee should not start to consume it for health benefits alone.

Review of the Evidence

Parkinson's Disease

Evidence from two systematic reviews[108, 109] and one quantitative meta-analysis[110] confirmed an inverse association between coffee, caffeine, and risk of Parkinson's disease. Qi et al. evaluated six case-control studies and seven prospective articles and documented a non-linear relationship between coffee and risk of Parkinson's disease, overall.[110] The lowest risk was observed at about 3 cups/day (smoking-adjusted risk reduction was 28 percent). For caffeine, a linear dose-response was found and every 200 mg/day increment in caffeine intake was associated with a 17 percent lower risk of Parkinson's disease. Using a combination of cohort, case-control, and cross-sectional data, Costa et al. summarized that the risk of Parkinson's disease was 25 percent lower among those consuming the highest versus lowest amounts of caffeine.[108] Like Qi et al., Costa et al. documented a linear dose-response with caffeine intake such that every 300 mg/day was associated with a 24 percent lower risk of Parkinson's disease. In both reports, associations were weaker among women than in men.

Cognition

Two systematic reviews[111, 112] and one meta-analysis[112] examined the effects of caffeine from various sources, including coffee, tea, and chocolate, on cognitive outcomes. Arab et al. systematically reviewed six longitudinal cohort studies evaluating the effect of caffeine or caffeine-rich beverages on cognitive decline.[111] Most studies in this review used the Mini Mental State Examination Score as a global measure of cognitive decline. The review concluded that estimates of cognitive decline were lower among caffeine consumers, although there was no clear dose-response relationship. Studies also showed stronger associations among women than men. In a meta-analysis of nine cohort and two case-control studies, caffeine intake from various sources was associated with a 16 percent lower risk of various measures of cognitive impairment/decline. Specifically, data from four studies indicate that caffeine is associated with a 38 percent lower risk of Alzheimer’s disease.

For additional details on this body of evidence, visit: Appendix E-2.39a Evidence Portfolio, Appendix E-2.39b Systematic Review/Meta-Analysis Data Table, and References 108-112

<u>Caffeine and Pregnancy Outcomes</u>

Conclusion

Consistent evidence from observational studies indicates that moderate caffeine intake in pregnant women is not associated with risk of preterm delivery. **DGAC Grade: Moderate**

Higher caffeine intake is associated with a small increased risk of miscarriage, stillbirth, low birth weight, and small for gestational age (SGA) births. However, these data should be interpreted cautiously due to potential recall bias in the case-control studies and confounding by smoking and pregnancy signal symptoms. The DGAC recognizes that there is limited data to identify a level of caffeine intake beyond which risk increases. Based on the existing data, the risk of miscarriage, stillbirth, low birth weight, and SGA births is minimal given the average caffeine intake of pregnant women in the United States. **DGAC Grade: Limited**

Implications

Overall, the evidence supports current recommendations to limit caffeine intake during pregnancy as a precaution. Based on existing evidence, women who are pregnant or planning to become pregnant should be cautious and adhere to current recommendations of the American Congress of Obstetricians and Gynecologists regarding caffeine consumption, and not consume more than 200 mg caffeine per day (approximately two cups of coffee per day).

Review of the Evidence

Two SRs/MA assessed observational studies on the association of caffeine intake with adverse pregnancy outcomes.[113, 114] The pregnancy outcomes included miscarriage, pre-term birth, stillbirth, SGA, and low birth weight. The most recent SR/MA by Greenwood et al. quantified the association between caffeine intake and adverse pregnancy outcomes from 60 publications from 53 separate cohort (26) and case-control (27) studies.[113] The evidence covered a variety of countries with caffeine intake categories that ranged from non-consumers to those consuming more than 1,000 mg/day. They found that an increment of 100 mg caffeine was associated with a 14 percent increased risk of miscarriage, 19 percent increased risk of stillbirth, 10 percent increased risk of SGA, and 7 percent increased risk of low birth weight. The risk of pre-term delivery was not increased significantly. The magnitude of these associations was relatively small within the range of caffeine intakes of the majority women in the study populations, and the associations became more pronounced at higher range (≥300 mg/day). The authors also note the substantial heterogeneity observed in the meta-analyses shows that interpretation of the results should be cautious. In addition, the results from prospective cohort studies and case-control studies were mixed together. Because coffee consumption is positively correlated with smoking, residual confounding by smoking may have biased the results toward a positive direction.

The other SR/MA assessed pre-term birth and the results were in agreement with Greenwood et al.[113] Maslova et al. reviewed 22 studies (15 cohort and 7 case-control studies) and found no significant association between caffeine intake and risk of pre-term birth in either case-control or cohort studies.[114] For all of the observational studies assessed across the SRs/MA, most studies did not adequately adjust for the pregnancy signal phenomenon, i.e. that nausea, vomiting, and other adverse symptoms are associated with a healthy pregnancy that results in a live birth, whereas pregnancy signal symptoms occur less frequently when the result is miscarriage. Coffee consumption decreases with increasing pregnancy signal symptoms, typically during the early weeks of pregnancy, and this severely confounds the association.[115] Greenwood et al. state that this potential bias is the most prominent argument against a causal role for caffeine in adverse pregnancy outcomes.[113] Only one randomized controlled trial of caffeine/coffee reduction during pregnancy has been conducted to date.[116] The study found that in pregnant women who consumed at least three cups of coffee a day and were less than 20 weeks pregnant, a reduction of 200 mg of caffeine intake (~ 2 cups) per day did not significantly influence birth weight or length of gestation, compared to those with no decrease in caffeine consumption. The trial did not examine other outcomes.

For additional details on this body of evidence, visit: Appendix E-2.39a Evidence Portfolio, Appendix E-2.39b Systematic Review/Meta-Analysis Data Table, and References 113, 114

Question 6: What is the relationship between high-dose coffee/caffeine consumption and health?

Source of Evidence: Systematic reviews[117, 118]

Conclusion

Evidence on the effects of excessive caffeine intake on the health of adults or children (>400 mg/day for adults; undetermined for children and adolescents) is limited. Some evidence links high caffeine intake in the form of energy drinks to certain adverse outcomes, such as caffeine toxicity and cardiovascular events. Randomized controlled trials (RCTs) on the relationship between high-caffeine energy drinks and cardiovascular risk factors and other health outcomes report mixed results. Evidence also is limited on the health effects of mixing alcohol with energy drinks, but some evidence suggests that energy drinks may mask the effects of alcohol intoxication, so an individual may drink more and increase their risk of alcohol-related adverse events. **DGAC Grade: Limited**

Implications

Early safety signals consisting of case reports of adverse events associated with high-caffeine drink consumption, including increased emergency room visits, indicate a potential public health problem. The DGAC agrees with the American Academy of Pediatrics and the American Medical Association that until safety has been demonstrated, limited or no consumption of high-caffeine drinks, or other products with high amounts of caffeine, is advised for vulnerable populations, including children and adolescents. High-caffeine energy drinks and alcoholic beverages should not be consumed together, either mixed together or consumed at the same sitting. This is especially true for children and adolescents.

Background

According to the FDA, the upper limit of moderate caffeine intake in healthy adult populations (barring pregnant women) is 400 mg/day, with intakes higher than this being considered excessive caffeine consumption. The FDA has not defined moderate and excessive intake levels for children and adolescents. However, according to Health Canada, children should not consume more than 2.5 mg of caffeine per kg bodyweight per day.[119] Although this guideline pertains only to children up to the age of 12 years, in the literature it is usually applied to children and adolescents of all ages. A caffeine threshold of 2.5 mg/kg/day would translate into around 37.5 mg/day for children ages 2 to 5 years with an average weight of 15 kg, 75 mg/day for youth ages 6 to 12 years with an average weight of 30 kg, and 137.5 mg/day for youth ages 13 to 17 years with an average weight of 55 kg.

The main sources of caffeine among both adults and children are coffee, tea, and carbonated soft drinks. Another product, which has received a lot of attention recently as a potential source of excessive caffeine intake, especially among younger populations, is energy drinks.[120] An energy drink is a beverage that contains caffeine as its active ingredient, along with other ingredients such as taurine, herbal supplements, vitamins, and sugar. It is usually marketed as a product that can improve energy, stamina, athletic performance, or concentration.[121] Energy drinks are relatively new to the market and have evaded oversight and regulation by the FDA due to their classification as dietary supplements, or because their components are generally recognized as safe.[121] Overall, these drinks are highly variable in caffeine content and some products have excessively high caffeine content (from 50 to 505 mg per can/bottle, with caffeine concentrations anywhere between 2.5 to 171 mg per fluid ounce).[122]

Health organizations including the American Academy of Pediatrics, the International Society of Sports Nutrition, and the American Medical Association have issued position statements on energy drinks, advising limited or no consumption among children and adolescents. Given the increasing evidence pointing toward harmful effects of excessive caffeine consumption,[105-107] the FDA requested the IOM to convene a workshop examining the science behind safe levels of caffeine intake. A report summarizing this workshop was recently published.[123] Its main conclusions were: 1) Children and adolescents are a potential vulnerable group, in whom caffeine intake could have detrimental health consequences. This is particularly important given insufficient data on caffeine consumption in this demographic, which is increasingly getting exposed to new modes of caffeine intake such as energy drinks, 2) not enough is understood about potential interactions between caffeine and other ingredients commonly found in caffeine-containing foods and beverages, and 3) more research is needed to identify individual differences in reactions to caffeine, especially in vulnerable populations, including children with underlying heart conditions and individuals with genetic predispositions to heart conditions.

The Center for Disease Control (CDC) recently reported on trends in caffeine intake over the past decade (1999-2010) among U.S. children, adolescents, and young adults.[124] The CDC found that although energy drinks were not widely available before 1999, energy drinks made up nearly 6 percent of caffeine intake in 2009-2010, indicating fast growth in U.S. consumption over a short period of time. When energy drink consumption was assessed in a nationally representative sample of U.S. secondary school students,[125] 35 percent of 8th graders, 30 percent of 10th graders, and 31 percent of 12th graders consumed energy drinks or shots, and consumption was higher for adolescent boys than girls. Furthermore, energy drink use was associated with higher prevalence of substance use, as assessed for all grades of U.S. secondary students.

Furthermore, a serious issue of public health concern has been the popular trend of combining energy drinks with alcoholic beverages. In 2010, the FDA determined that caffeine added to alcoholic beverages was not generally recognized as safe (GRAS), leading to withdrawal of premixed, caffeinated alcoholic beverages from the market.[126] Currently, Health Canada caps caffeine levels for energy drinks at 100 mg/250 ml (~1 cup) and has determined that an energy drink container that cannot be resealed be treated as a single-serving container, because the total volume is usually consumed. They also have mandated that manufacturers add a warning to labels that energy drinks should not be combined with alcohol. Recently, the CDC has made public statements on the dangers of mixing alcohol and energy drinks. They indicate that high amounts of caffeine in energy drinks can mask the intoxicating effects of alcohol, while at the same time having no effect on the metabolism of alcohol by the liver. Therefore, high amounts of caffeine in energy drinks may result in an "awake" state of intoxication, thus increasing the risk of alcohol-related harm and injury (http://www.cdc.gov/alcohol/fact-sheets/cab.htm, March 2014).[127]

Review of the Evidence

Several case reports of adverse events related to energy drink use have been published. A recent systematic review of case reports of adverse cardiovascular events related to consumption of energy drinks documented 17 such published case reports.[118] The cardiovascular events documented included atrial fibrillation, ventricular fibrillation, supraventricular tachycardia, prolonged QT, and ST elevation. In 41 percent of the cases, the person had consumed large amounts of energy drinks, and 29 percent of the cases were associated with consumption of energy drinks together with alcohol or other drugs. In 88 percent of the cases, no underlying cardiac condition was found that could potentially explain the cardiovascular event, although other cardiovascular risk factors co-occurred with energy drink consumption before the onset of the event in most cases. Of the cases that presented with serious adverse events, including cardiac arrest, the majority occurred with either acute heavy consumption of energy drinks or consumption in combination with alcohol or other drugs. Overall, the authors concluded that causality cannot be inferred from this case series, but physicians should routinely inquire about energy drink consumption in relevant cases and vulnerable consumers should be cautioned against heavy consumption of energy drinks or concomitant alcohol (or drug) ingestion. This systematic review is consistent with a recent report from the Drug Abuse Warning Network (DAWN) on energy drink-related emergency room visits that showed U.S. emergency room visits temporally related to energy drink consumption doubled between 2007 and 2011.[128] These visits were attributed mainly to adverse reactions to energy drinks, but also to combinations with alcohol or drugs. It is generally agreed that adverse events associated with energy drink consumption are underreported.

Several short-term RCTs have examined the health effects of energy drink consumption. All of these have been carried out in adult populations, probably due to ethical constraints in providing energy drinks to children. Burrows et al. recently published a systematic review of RCTs examining this question.[117] They found 15 such RCTS, examining the effect of variable doses of energy drinks (mean dose: one and a half 250 ml cans per study session) with differing ingredient combinations and concentrations on a number of different health outcomes. The high variability in exposure and outcome definitions made a meta-analysis infeasible. Overall, they found no consistent effects of energy drinks on cardiorespiratory outcomes (heart rate, arrhythmias, blood pressure), pathological outcomes (blood glucose, blood lactate, free fatty acids, clinical safety markers), and body composition, with some studies showing positive, some inverse, and some no associations. For many of these outcomes, consistent results could not be stated due to only one study reporting on them. There was a slight indication of a potential positive effect of energy drinks on physiological outcomes (run time to exhaustion, peak oxygen uptake, resting energy expenditure). However, the authors concluded that more studies were needed before arriving at a definitive conclusion. Two of the studies assessed the simultaneous ingestion of alcohol and energy drinks.[129, 130] One found that when compared with the ingestion of alcohol alone, the addition of an energy drink reduced individuals' perception of impairment from alcohol, while at the same time, objective measures indicated ongoing deficits in motor coordination and visual acuity.[129] Nor did energy drinks reduce breath alcohol concentration, indicating no change or increase in alcohol metabolism by the liver. Another study on energy drinks in combination with alcohol and exercise showed that during post-exercise recovery there was no effect on arrhythmias within 6 hours of energy drink ingestion in healthy young adults.[130]

Many of the these studies have methodological limitations, such as lack of a true control group (water or no drink), a very short follow-up duration of only a few hours, and small sample sizes, which could explain the inconsistent findings. In addition, many of these studies did not report whether they were commercially funded. Several of those that did report funding sources had financial conflicts of interest. Lastly, the doses of energy drinks used in these studies were not too high, resulting in caffeine intake levels that fell within the normal range. It is possible that excessive caffeine intake due to heavy energy drink consumption adversely affects several health outcomes, but this hypothesis was not clearly addressed by these studies. Hence it is difficult to ascertain the impact of excessive caffeine intake on health outcomes on the basis of these RCTs. In addition, very little data are available on the health effects of excessive caffeine consumption in pediatric populations.

For additional details on this body of evidence, visit: Appendix E-2.40 Evidence Portfolio and References 117, 118

Question 7: What is the relationship between consumption of aspartame and health?

Source of Evidence: *Scientific Opinion on the re-evaluation of aspartame (E 951) as a food additive (2013)*, European Food Safety Authority (EFSA) Panel on Food Additives and Nutrient Sources added to Food [29]

Conclusion

The DGAC generally concurs with the European Food Safety Authority (EFSA) Panel on Food Additives that aspartame in amounts commonly consumed is safe and poses minimal health risk for healthy individuals without phenylketonuria (PKU). **DGAC Grade: Moderate**

Limited and inconsistent evidence suggests a possible association between aspartame and risk of some hematopoietic cancers (non-Hodgkin lymphoma and multiple myeloma) in men, indicating the need for more long-term human studies. In addition, limited and inconsistent evidence indicates a potential for risk of preterm delivery. Due to very limited evidence it is not possible to draw any conclusions on the relationship between aspartame consumption and headaches. **DGAC Grade: Limited**

Implications

If individuals choose to drink beverages that are sweetened with aspartame, they should stay below the aspartame Acceptable Daily Intake (ADI) of no more than 50 mg/kg/day (a 12-ounce diet beverage contains approximately 180 mg of aspartame).[131] To be cautious, adults and children should be aware of the amount of aspartame they are consuming, given the need for more long-term human studies. Currently, most Americans are well below the ADI.[132]

Background

Aspartame is the most common low-calorie sweetener used in the United States. It is found in numerous dietary sources. Although most commonly associated with low-calorie/low-sugar versions of carbonated and non-carbonated beverages, it also is found in low-calorie/low-sugar versions of canned fruits and juices; instant cereals; baked goods; ice cream and frozen ices; candy and chocolate products; jams, jellies, syrups, and condiments; yogurt; and beer. Non-nutritive sweeteners are regulated by the FDA. The FDA has concluded that aspartame is safe as a general purpose sweetener in food.[133] Given the high interest of the public in the safety of aspartame, the DGAC reviewed the EFSA report on the sweetener and health outcomes.

Review of the Evidence

The most recent European Food Safety Authority report on the re-evaluation of aspartame as a food additive was used to address this question.[29] The EFSA report based its evaluation on original study reports and information submitted following public calls for data, previous evaluations, and additional literature that became available up until the end of public consultation on November 15, 2013. The DGAC focused on results from human studies, not animal studies or studies conducted *in vitro*. The Mode of Action (MoA) analysis on reproductive and developmental toxicity of aspartame also was included. Although the EFSA report considered both published and unpublished studies, the DGAC considered only published studies.

Cancer

A relatively limited body of evidence on human studies has directly addressed the relationship between aspartame consumption and cancer risk. The most consistent finding in six U.S. and European case-control studies[134-139] was the absence of an adverse relationship between consumption of low-calorie sweeteners, including aspartame, and risk of some cancers. An exception was one study in Argentina that found a positive association between long-term use (≥10 y) of artificial sweeteners and risk of urinary tract tumors (UTT), compared to non-users; although for short-term users, no association was observed.[134]

The findings of two prospective cohort studies[140, 141] were not consistent. Lim et al. examined a large cohort of men and women from the NIH-AARP Diet and Health study and found no association between consumption of aspartame-containing beverages and risk of overall hematopoietic cancers, brain cancers, or their subtypes.[140] A second large prospective cohort study by Shernhammer et al. involved the Nurses' Health Study (NHS) and Health Professionals Follow-up Study (HPFS) cohorts followed over 22 years with dietary intake measured every 4 years.[141] In this study, the highest category of aspartame intake (≥143 mg/day from diet soda and aspartame packets) was associated with significantly elevated risk of non-Hodgkin lymphoma (NHL) and of multiple myeloma in men, but not in women. Both of the prospective cohort studies that addressed cancer risk had limitations regarding generalizability. The NIH-AARP cohort had an age range of 50 to 71 years and was, therefore, not generalizable to the overall adult population. Additionally, the Panel considered the positive findings in Shernhammer et al. to be preliminary and require replication in other populations because the positive association between aspartame consumption and NHL was limited to men and lacked a clear dose-response relationship.[29]

Further investigation should be considered to ensure that no association exists between aspartame consumption and specific cancer risk.

Preterm Delivery

Two European cohort studies were used in this evaluation. A large prospective cohort study by Halldorsson et al.[142] from the Danish National Birth Cohort investigated associations between consumption of artificially sweetened and sugar-sweetened soft drinks during pregnancy and subsequent pre-term delivery. Also, a large prospective cohort study of Norwegian women by Englund-Ögge et al.[143] investigated the relationship between consumption of artificially sweetened and sugar-sweetened soft drinks during the first 4 to 5 months of pregnancy and subsequent pre-term delivery. In addition, La Vecchia combined these two studies in a meta-analysis that the Panel considered.[144]

Regarding the Halldorsson study, significant trends in risk of pre-term delivery with increasing consumption of artificially sweetened drinks (carbonated and non-carbonated) were found, but not for sugar-sweetened drinks.[142] In the highest exposure groups (≥ 4 servings/d) the odds ratios relative to non-consumption were 1.78 (95% CI: 1.19-2.66) and 1.29 (95% CI: 1.05-1.59), respectively, for carbonated and non-carbonated artificially sweetened drinks. Associations with consumption of artificially sweetened carbonated drinks did not differ according to whether delivery was very early (less than 32 weeks) or only moderately or late pre-term.[142] The EFSA Panel noted that the prospective design and large size of the study sample were major strengths, and that the methods used had no important flaws.[29] The Panel agreed with the authors who concluded that replication of their findings in another setting was warranted.

Regarding the Englund-Ögge study, no significant trends were found in risk of pre-term delivery with increasing consumption of artificially sweetened drinks or sugar-sweetened drinks.[143] Small elevations of risk were observed with higher consumption of artificially sweetened soft drinks, but after adjustment for covariates, these reached significance only when categories of consumption were aggregated to four levels, and then the odds ratio for the highest category (≥ 1 serving/day) was 1.11 (95% CI: 1.00-1.24) compared with non-consumption. This was driven by an increase in spontaneous but not medically induced pre-term delivery. Associations with sugar-sweetened soft drinks tended to be stronger, with an adjusted odds ratio of 1.25 (95% CI: 1.08-1.45) for consumption of at least 1 serving per day. The Panel noted that effects may have been underestimated because of inaccuracies in the assessment of dietary exposures, but the method was similar to that used by Halldorsson et al., and the same for sugar-sweetened as for artificially sweetened soft drinks.[29]

Behavior and Cognition

Children

Two RCTs[145, 146] and two non-randomized controlled trials[147, 148] conducted in the United States were included in the evidence on effects of aspartame on behavior and cognition in children. Wolraich et al. compared diets high in sucrose to diets high in aspartame in 25 preschool and 23 primary school-age children and found that even when intake exceeded typical dietary levels, neither dietary sucrose nor aspartame affected children's behavior or cognitive function.[146] Shaywitz et al. examined the effect of large doses of aspartame (10 times usual consumption) on behavioral/cognitive function in children with attention deficit disorder (ages 5 to 13 years) and found no effect of aspartame on cognitive, attentive, or behavioral testing.[146] Roshon and Hagan examined 12 preschool children on alternate experimental days with a challenge of sucrose- or aspartame-containing drinks and found no significant differences in locomotion, task orientation, or learning.[148] Lastly, Kruesi et al. investigated the effect of sugar, aspartame, saccharin, and glucose on disruptive behavior in 30 preschool boys on four separate experimental days.[147] There was no significant difference in scores of aggression or observer's ratings of behavior in response to any of the treatments. The limitations of this evidence were that all of the trials were approximately 20 to 30 years old, all had small sample sizes, and all were conducted over the short-term (1 day to 3 weeks). Overall, the Panel noted that no effects of aspartame on behavior and cognition were observed in children in these studies.[29]

Adults

Seven studies on the effect of aspartame on adult behavior and cognition were included in this body of evidence. Five RCTs, one non-randomized controlled trial, and one case-control study were conducted in the United States. Two of these trials examined a single experimental dose of aspartame on one day.[149, 150] Lapierre et al. examined 15 mg aspartame/kg body weight in 10 healthy adults and found no significant differences between aspartame and placebo in cognition or memory during the study.[149] Ryan-Harshman et al. tested 13 healthy adult men and found no change in any behavioral effects measured.[150] A third randomized crossover trial examined 48 adults over 20 days; half of the participants were given high dose aspartame (45 mg/kg/d) and half were given low dose aspartame (15 mg/kg/d).[151] This study found no neuropsychologic, neurophysiologic, or behavioral effects linked to aspartame consumption. Two trials were conducted with pilots or college students to test cognitive abilities related to aviation tasks.[152, 153] In the first study, 12 pilots were given aspartame (50 mg/kg) or placebo and tested for aviation-related information processing after a single treatment on one day. The authors detected no performance decrements associated with exposure to aspartame. In the follow-up study, college students were given repeated dosing of aspartame (50 mg/kg for 9 days) and tested for aviation-related cognitive tasks. No impaired performance was observed. One non-randomized crossover trial examined the effects of aspartame on mood and well-being in 120 young college women and found no difference in changes in mood after consuming a 12-ounce water or aspartame-sweetened beverage on a single day.[154] Lastly, a case-control study was conducted with 40 adults with unipolar depression and a similar number of subjects without a psychiatric history.[155] Participants were given aspartame (30 mg/kg) or placebo for 7 days and individuals with depression reported an increase in severity of self-scored symptoms between aspartame and placebo; whereas the non-depressed matched subjects reported no difference. This suggested that individuals with mood disorders may be sensitive to aspartame. Overall, the Panel noted the limited number of participants, the short duration of the studies, and the inconsistency of the reporting of the results in all adult studies. However, despite these limitations, the Panel concluded that there was no evidence that aspartame affects behavior or cognitive function in adults.[29]

Other (Headaches, Seizures)

Several studies examined headaches and seizures. A number of RCTs were conducted to assess the incidence of headache after consumption of aspartame. One RCT tested the effects of aspartame within 24 hours of consumption (30 mg/kg) on 40 subjects with a history of headache and found no difference in the incidence rate of headaches.[156] Another RCT looked at the effect of aspartame on frequency and intensity of migraine headaches in 10 subjects with medical diagnosis of migraine headaches over 4 weeks.[157] The authors found an increase in the frequency of migraine headaches with the aspartame treatment. In an RCT of 18 subjects with self-described sensitivity to aspartame, the participants reported headaches on 33 percent of the days, compared with 24 percent with placebo.[158] The authors concluded that a subset of the population may be susceptible to headaches induced by aspartame. Lastly, in a survey study of 171 patients at a headache unit, 8 percent reported that aspartame was a trigger of headaches compared to 2.3 percent for carbohydrates and 50 percent for alcohol.[159] Overall, the Panel concluded the possible effect of aspartame on headaches had been investigated in various studies which reported conflicting results, ranging from no effect to the suggestion that a small subset of the population may be susceptible to aspartame-induced headaches.[29] The number of existing studies was small and not recent and several studies had high dropout rates. The Panel noted that because of the limitations of the studies, it was not possible to draw a conclusion on the relationship between aspartame consumption and headaches.

Several small studies assessed seizures. One RCT in children investigated whether aspartame would induce the occurrence of petit mal seizures.[160] Ten children were given one treatment of aspartame at the ADI of 40 mg/kg and that treatment exacerbated the number of electroencephalogram spike waves per hour for these children without a history of seizures. In a second RCT, aspartame (34 mg/kg) was administered to 10 epileptic children over 2 weeks to examine the induction of seizures.[145] No difference was found in the occurrence of seizures between aspartame and placebo exposure. Another RCT studied 18 subjects who claimed to have experienced epileptic seizures due to aspartame.[161] One treatment (50 mg/kg) was administered on a single day and the authors reported no seizures or other adverse effect from aspartame treatment in this group. Overall, the Panel concluded that the available data do not provide evidence for a relationship between aspartame consumption and seizures.[29]

Pregnancy Outcomes: Mode of Action (MoA) analysis

The EFSA Panel considered that adverse effects on reproduction and development reported for aspartame in animal studies could be attributed to the metabolite phenylalanine.[29] They undertook a formal Mode of Action (MoA) analysis of the putative role of phenylalanine in developmental toxicity (as seen in animal studies).

Risk characterization was based on comparison of plasma phenylalanine levels following aspartame administration with plasma phenylalanine levels associated with developmental effects in children born from mothers with PKU. Current clinical practice guidelines recommend PKU patients restrict dietary intake of phenylalanine to keep plasma levels below 360μM. The EFSA Panel noted that intakes of aspartame as a food additive could occur at the same time as other dietary phenylalanine sources. Therefore, they considered the threshold used for comparisons should be lowered to allow for simultaneous intake of aspartame with meals. So plasma phenylalanine from the diet (120μM) was subtracted from 360μM to determine the maximum safe plasma concentration of phenylalanine that can be derived from aspartame (240µM).

The Panel considered that given these conservative assumptions, realistic dietary intake of aspartame and the confidence intervals provided by the modeling, the peak plasma phenylalanine levels would not exceed the clinical target threshold of 240µM when a normal individual consumed aspartame at or below the current ADI of 40 mg/kg body weight/day. Therefore, the Panel concluded there would not be a risk of adverse effects on pregnancy in the general population at the current ADI.[29]

For additional details on this body of evidence, visit: Appendix E-2.41 Evidence Portfolio and *Scientific Opinion on the re-evaluation of aspartame (E 951) as a food additive (2013),* European Food Safety Authority (EFSA) Panel on Food Additives and Nutrient Sources added to Food. Available at www.efsa.europa.eu/efsajournal

Question 8: What Consumer Behaviors Prevent Food Safety Problems? (Topic update from 2010)

Introduction and Methods

Food safety continues to be an issue of public health importance. Foodborne illness is a preventable, yet common issue affecting the U.S. population. Each year, approximately 1 in 6 people in the U.S. population become ill, 128,000 are hospitalized, and 3,000 die of foodborne illness.[162] It is critical to educate consumers and food producers on good techniques and behaviors for preventing food borne illness.

The 2010 DGAC conducted NEL systematic reviews for the Food Safety and Technology chapter and provided in-depth guidance on foodborne illness prevention. The 2015 DGAC reviewed the content related to consumer behavior and the prevention of food safety problems. The Committee determined that the majority of the 2010 food safety guidance was current and that only minor updates were necessary. For more information on the evidence review on food safety, refer to the DGAC 2010 report, Food Safety and Technology Section: (http://origin.www.cnpp.usda.gov/Publications/DietaryGuidelines/2010/DGAC/Report/D-8-FoodSafety.pdf).

The four food safety principles—Clean, Separate, Cook, and Chill are the foundation of the Fight BAC!® campaign (www.fightbac.org) and are reemphasized in this report. Data from the Centers for Disease Control and Prevention,[30] Food and Drug Administration,[31] and the Food Safety and Inspection Service[32] were used to update the 2010 DGAC tables on the following topics related to consumer behavior and food safety:

CLEAN and SEPARATE (Tables D5.1, D5.2, D5.3)

- Techniques for hand sanitation, washing fresh produce, and preventing cross-contamination.

COOK and CHILL (Table D5.4)

- Temperature control during food preparation and storage.

Table D5.3 includes updated guidance on preventing cross-contamination from shopping to serving foods. Table D5.4 lists recommended internal temperatures for meat, seafood, eggs, and leftovers. Additionally, Tables D5.5 and D5.6 provide recommended techniques for using food and refrigerator/freezer thermometers. Specific changes made to the 2010 tables are detailed in the footnotes of the tables.

Food Safety—Tables

Table D5.1. Recommended procedures for hand sanitation

When washing hands with soap and water:
• **Wet** your hands with clean, running water (warm or cold), turn off the tap, and apply soap.[1]
• **Lather** your hands by rubbing them together with the soap. Be sure to lather the backs of your hands, between your fingers, and under your nails.[2]
• **Scrub** your hands for at least 20 seconds. Need a timer? Hum the "Happy Birthday" song from beginning to end twice.[3]
• **Rinse** your hands well under clean, running water.
• Dry your hands using a clean towel or air dry them.[4]
If soap and clean, running water are not available, use an alcohol-based hand sanitizer that contains at least 60% alcohol[5]. Hand sanitizers are not as effective when hands are visibly dirty or greasy.[6] How do you use hand sanitizer:[7]
• Apply the product to the palm of one hand (read the label to learn the correct amount).
• Rub your hands together.
• Rub the product over all surfaces of your hands and fingers until your hands are dry.

Updates to the 2010 DGAC table

[1] Water temperature "warm or cold" and a conservation recommendation of 'turn off the tap' were added.

[2] The soap is to be help while lathering one's hands, then rub all together. "Scrub all surfaces" was clarified to "the backs of hands, between fingers, and under nails."

[3] "At least" was added to the 20 seconds time frame. To give a time reference, the suggestion to" hum the Happy Birthday song…" was added.

[4] The word 'paper' was removed as a modifier for towel, and instead it was specified to be a 'clean' towel. The option to 'air dry them' was added and the option of using an air dryer was removed from the phrase. Also removed was the direction to use your paper towel to turn off the faucet.

[5] The words 'clean' and 'running' were inserted in the directions for when water is not available. 'Hand sanitizer that contains at least 60% alcohol' replaces 'gel'.

[6] This guidance was added.

[7] The following step was added, "Read the label to learn the correct amount."

Source: Adapted from http://www.cdc.gov/handwashing/when-how-handwashing.html. Accessed June 2, 2014.[30]

Table D5.2. Recommended techniques for washing produce

When preparing any fresh produce, begin with clean hands. Wash your hands for at least 20 seconds with soap and warm water before and after preparation.
Cut away any damaged or bruised areas on fresh fruits and vegetables before preparing and/or eating. Produce that looks rotten should be discarded.
Wash all produce **thoroughly** under running water **before eating, cutting or cooking**. This includes produce grown conventionally or organically at home, or purchased from a grocery store or farmer's market. Washing fruits and vegetables with soap or detergent or using commercial produce washes is not recommended.
Even if you plan to peel the produce before eating, it is still important to wash it first so dirt and bacteria are not transferred from the peel via the knife to the fruit or vegetable [1].
Scrub firm produce, such as melons and cucumbers, with a clean produce brush.
Dry produce with a clean cloth towel or paper towel to further reduce bacteria that may be present.
Many pre-cut, bagged, or packaged produce items like lettuce are pre-washed and ready-to-eat. If so, it will be stated on the package and you can use the product without further washing.
If you do choose to wash a product marked "pre-washed" and "ready-to-eat," be sure to use safe handling practices to avoid any cross-contamination (see Table D5.3).

Updates to the 2010 DGAC table

[1] The following explanation was provided: ". . . so dirt and bacteria aren't transferred from the knife onto fruit or vegetable."

Source: Adapted from http://www.fda.gov/downloads/Food/ResourcesForYou/Consumers/UCM174142.pdf. Accessed June 2, 2014[31]

Table D5.3. Recommended techniques for preventing cross-contamination

When Shopping:
Separate raw meat, poultry, and seafood from other foods in your grocery-shopping cart. Place these foods in plastic bags to prevent their juices from dripping onto other foods. It is also best to separate these foods from other foods at check out and in your grocery bags.
When Refrigerating Food[1]:
Place raw meat, poultry, and seafood in containers or sealed plastic bags to prevent their juices from dripping onto other foods. Raw juices often contain harmful bacteria.
Store eggs in their original carton and refrigerate as soon as possible.
When Preparing Food:
Washing raw poultry, beef, pork, lamb, or veal before cooking it is not recommended. Bacteria in raw meat and poultry juices can be spread to other foods, utensils, and surfaces.
Wash hands and surfaces often. Harmful bacteria can spread throughout the kitchen and get onto cutting boards, utensils, and countertops. To prevent this:
• Wash hands with soap and warm water for 20 seconds before and after handling food, and after using the bathroom, changing diapers; or handling pets.
• Use hot, soapy water and paper towels or clean cloths to wipe up kitchen surfaces or spills. Wash cloths often in the hot cycle of your washing machine.
• Wash cutting boards, dishes, and counter tops with hot, soapy water after preparing each food item and before you go on to the next item.
• A solution of 1 tablespoon of unscented, liquid chlorine bleach per gallon of water may be used to sanitize surfaces and utensils.
Cutting Boards:
Always use a clean cutting board.
If possible, use one cutting board for fresh produce and a separate one for raw meat, poultry, and seafood.
Once cutting boards become excessively worn or develop hard-to-clean grooves, they should be replaced.
Marinating Food:
Always marinate food in the refrigerator, not on the counter.
Sauce that is used to marinate raw meat, poultry, or seafood should not be used on cooked foods, unless it is boiled just before using.
When Serving Food:
Always use a clean plate.
Never place cooked food back on the same plate or cutting board that previously held raw food.

Updates to the 2010 DGAC table

[1]This sentence was deleted, ""When not possible, store raw animal foods below ready-to-eat foods and separate different types of raw animal foods, such as meat, poultry, and seafood from each other so that they do not cross-contaminate each other."

Source: Adapted from http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/safe-food-handling/washing-food-does-it-promote-food-safety/washing-food and http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/safe-food-handling/be-smart-keep-foods-apart/ct_index Accessed June 3, 2014.[32]

Table D5.4. Recommended safe minimum internal temperatures

Cook to the minimum internal temperatures below, as measured with a clean food thermometer before removing meat from the heat source. For safety and quality, allow meat to rest for at least three minutes before carving or consuming. For reasons of personal preference, consumers may choose to cook meat to higher temperatures.[1] [c]

Food	Degrees Fahrenheit
Ground Meat and Meat Mixtures[a]	
Beef, Pork, Veal, Lamb	160
Turkey, Chicken	165
Fresh Beef, Pork, Veal, Lamb[a,2]	
Steaks, roasts, chops[a]	145
Poultry[a]	
Chicken and Turkey, whole	165
Poultry breasts, roasts	165
Poultry thighs, wings	165
Duck and Goose	165
Stuffing (cooked alone or in bird)	165
Fresh Pork[a]	160
Ham[a]	
Fresh (raw)[3]	145
Pre-cooked (to reheat)	140
Eggs and Egg Dishes[a]	
Eggs	Cook until yolk and white are firm.
Egg dishes	160
Fresh Seafood[b]	
Finfish	145
	Cook fish until it is opaque (milky white) and flakes with a fork.
Shellfish	Cook shrimp, lobster, and scallops until they reach their appropriate color. The flesh of shrimp and lobster should be an opaque (milky white) color. Scallops should be opaque (milky white) and firm.
	Cook clams, mussels, and oysters until their shells open. This means that they are done. Throw away the ones that didn't open.
	Shucked clams and shucked oysters are fully cooked when they are opaque (milky white) and firm[4].
Leftovers and Casseroles[a]	165

Updates to the 2010 DGAC table

1 An introductory paragraph was added on the topic of allowing for a three-minute rest period after cooking meat.
2 Pork was added to the list of fresh meats.
3 Fresh (raw) ham was added to the table.
4 Information on cooking status of shucked clams and oysters was added.

Sources:

[a] http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/safe-food-handling/kitchen-companion-your-safe-food-handbook/ct_index. Accessed June 3, 2014.[32]
[b] http://www.fda.gov/Food/ResourcesForYou/HealthEducators/ucm082294.htm. Accessed June 3, 2014.[31]
[c] http://www.fsis.usda.gov/wps/wcm/connect/8e9f95a6-fd35-42d3-b6cb-b07a4b853992/Leftovers_and_Food_Safety.pdf?MOD=AJPERES. Accessed June 3, 2014.[32]

Table D5.5. Recommended techniques for food thermometers

To be safe, meat, poultry, and egg[a] and seafood[b] products must be cooked to a safe minimum internal temperature to destroy any harmful microorganisms that may be in the food.
A food thermometer should also be used to ensure that cooked food is held at safe temperatures until served. Cold foods should be held at 40°F or below. Hot foods should be kept hot at 140°F or above.[a]
Most available food thermometers will give an accurate reading within 2 to 4°F. The reading will only be correct, however, if the thermometer is placed in the proper location in the food.[a]
In general, the food thermometer should be placed in the thickest part of the food, away from bone, fat, or gristle.[a]
When the food being cooked is irregularly shaped, such as with a beef roast, check the temperature in several places. Egg dishes and dishes containing ground meat and poultry should be checked in several places.[a]
When measuring the temperature of a thin food, such as a hamburger patty, pork chop, or chicken breast, a thermistor or thermocouple food thermometer should be used, if possible.[a]
However, if using an "instant-read" dial bimetallic-coil food thermometer, the probe must be inserted in the side of the food so the entire sensing area (usually 2 to 3 inches) is positioned through the center of the food.[a]
To avoid burning fingers, it may be helpful to remove the food from the heat source (if cooking on a grill or in a frying pan) and insert the food thermometer sideways after placing the item on a clean spatula or plate.[a]
Food thermometers should be washed with hot soapy water. Most thermometers should not be immersed in water.[a]

Sources: [a] http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/appliances-and-thermometers/kitchen-thermometers/ct_index., Accessed June 3, 2014.[32]
[b] http://www.fda.gov/Food/ResourcesForYou/HealthEducators/ucm082294.htm, Accessed June 3, 2014.[31]

Table D5.6. Recommended techniques for using refrigerator/freezer thermometers

For safety, it is important to verify the temperature of refrigerators and freezers.
Refrigerators should maintain a temperature no higher than 40°F.
Frozen food will hold its top quality for the longest possible time when the freezer maintains 0°F or below.
To measure the temperature in the refrigerator:
Put the thermometer in a glass of water and place in the middle of the refrigerator. Wait 5 to 8 hours. If the temperature is not 38 to 40°F, adjust the refrigerator temperature control. Check again after 5 to 8 hours.
To measure the temperature in the freezer:
Place the thermometer between frozen food packages. Wait 5 to 8 hours. If the temperature is not 0 to 2°F, adjust the freezer temperature control. Check again after 5 to 8 hours. An appliance thermometer can be kept in the refrigerator and freezer to monitor the temperature at all times. This can be critical in the event of a power outage. When the power goes back on, if the refrigerator is still 40°F and the freezer is 0°F or below, the food is safe[1].

Updates to the 2010 DGAC table
[1] When referring to the correct freezer temperature, 'or below' was added after 'zero degrees Fahrenheit.'

Source: http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/appliances-and-thermometers/appliance-thermometers/appliance-thermometers. , Accessed June 3, 2014.[32]

CHAPTER SUMMARY

Access to sufficient, nutritious, and safe food is an essential element of food security for the U.S. population. A sustainable diet is one that assures this access for both the current population and future generations. This chapter focused on evaluating the evidence around sustainable diets and several topic areas of food safety.

The major findings regarding sustainable diets were that a diet higher in plant-based foods, such as vegetables, fruits, whole grains, legumes, nuts, and seeds, and lower in calories and animal-based foods is more health promoting (as discussed in ***Part B. Chapter 2: 2015 DGAC Themes and Recommendations: Integrating the Evidence***) and is associated with less environmental impact than is the current U.S. diet. This pattern of eating can be achieved through a variety of dietary patterns, including the “Healthy U.S.-style Pattern,” the “Healthy Mediterranean-style Pattern,” and the “Healthy Vegetarian Pattern” (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** for a description of these patterns). All of these dietary patterns are aligned with lower predicted environmental impacts and provide food options that can be adopted by the U.S. population. Current evidence shows that the average U.S. diet has a potentially larger environmental impact in terms of increased GHG emissions, land use, water use, and energy use, compared to the above dietary patterns. This is because the current U.S. population intake of animal-based foods is higher and the plant-based foods are lower, than proposed in these three dietary patterns. Of note is that no food groups need to be eliminated completely to improve food sustainability outcomes.

A moderate amount of seafood is an important component of two of three of these dietary patterns, and has demonstrated health benefits. The seafood industry is in the midst of rapid expansion to meet worldwide demand, although capture fishery production has leveled off while aquaculture is expanding. The collapse of some fisheries due to overfishing in the past decades has raised concern about the ability to produce a safe and affordable supply. In addition, concern has been raised about the safety and nutrient content of farm-raised versus wild-caught seafood. To supply enough seafood to support meeting dietary recommendations, both farm-raised and wild caught seafood will be needed. The review of the evidence demonstrated, in the species evaluated, that farm-raised seafood has as much or more EPA and DHA per serving than wild caught. Low-trophic seafood, such as catfish and crawfish, regardless of whether wild caught or farm-raised seafood, have less than half the EPA and DHA per serving than high-trophic seafood, such as salmon and trout.

Regarding contaminants, for the majority of wild caught and farmed species, neither the risks of mercury nor organic pollutants outweigh the health benefits of seafood consumption. Consistent evidence demonstrated that wild caught fisheries that have been managed sustainably have remained stable over the past several decades; however, wild caught fisheries are fully exploited and their continuing productivity will require careful management nationally and internationally to avoid long-term collapse. Expanded supply of seafood nationally and internationally will be dependent upon the increase of farm-raised seafood worldwide.

The impact of food production, processing, and consumption on environmental sustainability is an area of research that is rapidly evolving. As further research is conducted and best practices evaluated, additional evidence will inform both supply-side participants and consumers on how best to shift behaviors locally, nationally, and globally to support sustainable diets. Linking health, dietary guidance and the environment will promote human health and the sustainability of natural resources and ensure current and long-term food security.

In regards to food safety, updated and previously unexamined areas of food safety were studied. No previous DGACs have reported on coffee/caffeine consumption and health. Currently, strong evidence shows that consumption of coffee within the moderate range (3 to 5 cups per day or up to 400 mg/d caffeine) is not associated with increased long-term health risks among healthy individuals. In fact, consistent evidence indicates that coffee consumption is associated with reduced risk of type 2 diabetes and cardiovascular disease in healthy adults. Moreover, moderate evidence shows a protective association between coffee/caffeine intake and risk of Parkinson's disease. Therefore, moderate coffee consumption can be incorporated into a healthy dietary pattern, along with other healthful behaviors. To meet the growing demand of coffee, there is a need to consider sustainability issues of coffee production in economic and environmental terms. However, it should be noted that coffee as it is normally consumed can contain added calories from cream, milk, and added sugars. Care should be taken to minimize the amount of calories from added sugars and high-fat dairy or dairy substitutes added to coffee.

The marketing and availability of high-caffeine beverages and products is on the rise. Unfortunately, only limited evidence is currently available to ascertain the safety of high caffeine intake (greater than 400 mg/day for adults and undetermined for children and adolescents), that may occur with rapid consumption of large-sized energy drinks. The limited data suggest adverse health outcomes, such as caffeine toxicity and cardiovascular events. Concern is heightened when caffeine is combined with alcoholic beverages. Limited or no consumption of high caffeine drinks, or other products with high amounts of caffeine, is advised for children and adolescents. Energy drinks with high levels of caffeine and alcoholic beverages should not be consumed together, either mixed together or consumed at the same sitting.

The DGAC also examined the food additive aspartame. At the level that the U.S. population consumes aspartame, it appears to be safe. However, some uncertainty continues about increased risk of hematopoietic cancers in men, indicating a need for more research.

Individual behaviors along with sound government policies and responsible private sector practices are all needed to reduce foodborne illnesses. To that end, the DGAC updated the established recommendations for handling foods at home.

NEEDS FOR FUTURE RESEARCH

Dietary Patterns and Sustainability

1. Conduct research to determine whether sustainable diets are affordable and accessible to all sectors of the population and how this can be improved, including how policy strategies could influence the supply chain (all steps from farm to plate) to affect this improvement.

   Rationale: Ensuring that sustainable diets are accessible and affordable to all sectors of the population is important to promote food security.

2. Develop, conduct, and evaluate in-depth analyses of U.S. domestic dietary patterns and determine the degree to which sustainability practices, domestically and internationally, are important to food choice and how to increase public awareness of the impact of food choices on environmental outcomes.

   Rationale: Understanding consumer choice across demographic groups and the degree to which either health and/or sustainability is a significant decisional criterion as well as the degree to which choice theory can be used to improve choices will be important to helping drive change.

3. Develop a robust understanding of how production practices, supply chain decisions, consumer behaviors, and waste disposal affect the environmental sustainability of various practices across the USDA food components of MyPlate.

   Rationale: Developing sustainable production and supply chain practices for all parts of MyPlate, especially meat and dairy products will be important to reduce their environmental impact.

4. Determine the potential economic benefits and challenges to supply chain stakeholders in relationship to findings in Research Recommendation 3.

   Rationale: Experience demonstrates that many practices over the past few decades that improve the environmental footprint of, for example, production practices, also have led to improved profit (e.g., Integrated Pest Management to reduce pesticide use in many fruit and vegetables). It is important to know how changes will affect profit to help enable future policy in both the private and public spheres.

Seafood Sustainability

5. Conduct research on methods to ensure the maintenance of nutrient profiles of high-trophic level farmed seafood and improve nutrient profiles of low-trophic farmed seafood concurrently with research to improve production efficacy.

   Rationale: The evidence supporting healthfulness of seafood consumption is based on consumption of predominantly wild caught species. Many popular low-trophic level farmed seafood have nutrient profiles that depend on feeds. Efficient production of seafood with nutrient profiles that are known to be healthful should be emphasized.

6. Conduct research to develop methods to ensure contaminant levels in all seafood remain at levels similar to or lower than at present. Maintain monitoring of contaminant levels for capture fisheries to ensure that levels caused by pollution do not rise appreciably. This research should include developing effective rapid response approaches if the quality of seafood supply is acutely affected.

   Rationale: Current research findings support the contention that contaminant levels are generally well below those that significantly alter the healthfulness of seafood. As industry naturally improves efficiency, feeds and environmental conditions should be monitored to maintain or reduce priority contaminants and insure significant new contaminants do not enter the seafood supply.

Usual Caffeine/Coffee Intake

7. Evaluate the effects of coffee on health outcomes in vulnerable populations, such as women who are pregnant (premature birth, low birth weight, spontaneous abortion).

   Rationale: Given the limited evidence of the effects of coffee/caffeine consumption on pregnancy outcomes, future studies need to establish safe levels of coffee/caffeine consumption during pregnancy.

8. Examine the effects of coffee on sleep patterns, quality of life, and dependency and addiction.

   Rationale: Because coffee is a known stimulant, future research should examine the effect of coffee/caffeine on sleep quality, dependency, addiction, and overall quality of life measures.

9. Evaluate the prospective association between coffee/caffeine consumption and cancer at different sites.

   Rationale: Large well-conducted prospective cohort studies that adequately control for smoking (status and dosage) and other potential confounders are needed to understand the association of coffee (caffeinated and decaffeinated) with cancer at different sites.

10. Examine prospectively the effects of coffee/caffeine on cognitive decline, neurodegenerative diseases, and depression.

    Rationale: Neurodegenerative diseases affect millions of people worldwide and more than five million Americans are living with Alzheimer's disease. Given the limited evidence of coffee/caffeine on neurodegenerative diseases, well-designed prospective studies should examine the association of coffee/caffeine consumption on cognitive decline, depression, and Alzheimer's disease.

11. Understand the mechanisms underlying the protective effects of coffee on diabetes and CVD.

    Rationale: Evidence for a biological plausibility for coffee on risk of type 2 diabetes and CVD stems primarily from animal studies. Randomized controlled trials in humans should evaluate the effect of coffee/caffeine on measures of glycemia, insulin sensitivity, endothelial dysfunction, and inflammation.

12. Understand the association between coffee and health outcomes in individuals with existing CVD, diabetes, cancer, neurodegenerative diseases, or depressive symptoms.

    Rationale: Strong evidence supports a protective effect of moderate coffee consumption on chronic disease risk in healthy adults, but its association among those with existing diseases has been less studied. Given that a substantial number of people suffer from these chronic diseases, the role of coffee in preventing other health outcomes in such groups remains understudied.

High-dose Caffeine Intake

13. Define excessive caffeine intake and safe levels of consumption for children, adolescents, and young adults.

    Rationale: Current research on caffeine and health outcomes has focused primarily on adults. Given the increasing prevalence of energy drink consumption among children, adolescents, and young adults, research is needed to identify safe levels of consumption in these groups.

14. Determine the prevalence of excessive caffeine intake in children and adults beyond intake of energy drinks.

    Rationale: Data on the sources (other than energy drinks) and doses of caffeine intake in children and adults are limited. Identifying the sources and safe levels of consumption will help in formulating policy and framing recommendations.

15. Examine the effect of excessive consumption of caffeine and energy drinks on health outcomes in both children and adults.

    Rationale: Prospective studies of associations of excessive caffeine and energy drink intake with health outcomes in children and adults are necessary, as randomized controlled trials are not be feasible given ethical constraints.

16. Conduct observational studies to examine the health effects of alcohol mixed with energy drinks.

    Rationale: In recent years, consumption of alcohol energy drinks by adolescents has resulted in emergency room admissions and deaths. No data exist on the prospective association between consumption of alcohol energy drinks and health outcomes in both adolescents and adults.

Aspartame

17. Examine the risks of aspartame related to some cancers, especially hematopoietic ones, and pregnancy outcomes.

    Rationale: Limited and inconsistent evidence suggests a possible association between aspartame and risk of hematopoietic cancers (non-Hodgkin lymphoma and multiple myeloma) in men, indicating the need for long-term human studies. Additionally, limited and inconsistent evidence indicates a potential for risk of preterm delivery, which warrants further research.

REFERENCES

1. Food and Agriculture Organization of the United Nations. Rome Declaration on World Food Security and World Food Summit Plan of Action. Rome, Italy,1996. Available from: http://www.fao.org/docrep/003/w3613e/w3613e00.htm.
2. Food And Agriculture Organization Of The United Nations. International Scientific Symposium Biodiversity And Sustainable Diets United Against Hunger. Rome,2010. Available from: http://www.fao.org/ag/humannutrition/28506-0efe4aed57af34e2dbb8dc578d465df8b.pdf.
3. United Nations Environment Programme. UNEP Year Book: Emerging Issues In Our Global Environment 2012. Available from: www.unep.org/yearbook/2012/.
4. Nellemann C, MacDevette, M., Manders, T., Eickhout, B., , Svihus B, Prins, A. G., Kaltenborn, B. P. (Eds). The environmental food crisis – The environment's role in averting future food crises. A UNEP rapid response assessment United Nations Environment Programme, GRID-Arendal; 2009. Available from: www.grida.no.

5. Burlingame B, Dernini S. Sustainable diets: the Mediterranean diet as an example. Public Health Nutr. 2011;14(12A):2285-7. PMID: 22166185. http://www.ncbi.nlm.nih.gov/pubmed/22166185.
6. World Wildlife Federation. Livewell: a balance of healthy and sustainable food choices 2011. Available from: http://assets.wwf.org.uk/downloads/livewell_report_jan11.pdf.
7. Heller MC, Keoleian GA, Willett WC. Toward a life cycle-based, diet-level framework for food environmental impact and nutritional quality assessment: a critical review. Environ Sci Technol. 2013;47(22):12632-47. PMID: 24152032. http://www.ncbi.nlm.nih.gov/pubmed/24152032.
8. United States Department of Agriculture. A Series of Systematic Reviews on the Relationship Between Dietary Patterns and Health Outcomes 2014. Available from: http://www.nel.gov/vault/2440/web/files/DietaryPatterns/DPRptFullFinal.pdf.
9. Eshel G, Shepon A, Makov T, Milo R. Land, irrigation water, greenhouse gas, and reactive nitrogen burdens of meat, eggs, and dairy production in the United States. Proc Natl Acad Sci U S A. 2014;111(33):11996-2001. PMID: 25049416. http://www.ncbi.nlm.nih.gov/pubmed/25049416.
10. Heller MCaK, G. A. . Greenhouse Gas Emission Estimates of U.S. Dietary Choices and Food Loss. Journal of Industrial Ecology. 2014.
11. Tilman D, Clark M. Global diets link environmental sustainability and human health. Nature. 2014. PMID: 25383533. http://www.ncbi.nlm.nih.gov/pubmed/25383533.
12. Health Council of the Netherlands. Guidelines for a healthy diet: the ecological perspective. 2011. publication no. 2011/08E. ISBN 978-90-5549-845-1:[Available from: http://www.gezondheidsraad.nl/sites/default/files/201108E.pdf.
13. Nordic Council of Ministers. Nordic Council of Ministers Secretariat. Nordic Nutrition Recommendations 2012: Integrating nutrition and physical activity. Available from: http://norden.diva-portal.org/smash/get/diva2:704251/FULLTEXT01.pdf.
14. German Council for Sustainable Development. The Sustainable Shopping Basket: A guide to better shopping. 2013. Available from: http://www.nachhaltigkeitsrat.de/uploads/media/Brochure_Sustainable_Shopping_Basket_01.pdf.
15. Institute of Medicine. Sustainable Diets: Food for Healthy People and a Healthy Planet: Workshop Summary. Washington, DC: The National Academies Press; 2014. Available from: http://www.nap.edu/openbook.php?record_id=18578&page=R2.
16. National Research Council. Toward sustainable agricultural systems in the 21st century Washington, DC: The National Academies Press; 2010. Available from: http://www.nap.edu/openbook.php?record_id=12832.
17. Nordin SM, Boyle M, Kemmer TM, Academy of N, Dietetics. Position of the academy of nutrition and dietetics: nutrition security in developing nations: sustainable food, water, and health. J Acad Nutr Diet. 2013;113(4):581-95. PMID: 23522578. http://www.ncbi.nlm.nih.gov/pubmed/23522578.
18. Sustainable Development Commission, editor. Looking back, Looking Forward: Sustainability and UK food policy 2000 – 20112011.
19. United Nations DfSD. Open Working Group proposal for Sustainable Development Goals 2014. Available from: http://sustainabledevelopment.un.org/content/documents/1579SDGs%20Proposal.pdf.

20. FAO Fisheries and Aquaculture Department Food and Agriculture Organization of the United Nations. The State of World Fisheries and Aquaculture Rome,2012. Available from: http://www.fao.org/docrep/016/i2727e/i2727e.pdf.
21. Higgins JPT GSe. Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0: The Cochrane Collaboration; 2011. Available from: www.cochrane-handbook.org.
22. Drummond MF, Jefferson TO. Guidelines for authors and peer reviewers of economic submissions to the BMJ. The BMJ Economic Evaluation Working Party. BMJ. 1996;313(7052):275-83. PMID: 8704542. http://www.ncbi.nlm.nih.gov/pubmed/8704542.
23. Eddy D. The role of mathematical modelling in Assessing medical technology,. Technology Assessment. 1985:144-54.
24. Stevenson M, Lloyd-Jones M, Morgan MY, Wong R. Non-invasive diagnostic assessment tools for the detection of liver fibrosis in patients with suspected alcohol-related liver disease: a systematic review and economic evaluation. Health Technol Assess. 2012;16(4):1-174. PMID: 22333291. http://www.ncbi.nlm.nih.gov/pubmed/22333291.
25. U.S. Department of Agriculture ARS. USDA National Nutrient Database for Standard Reference, Release 27. Nutrient Data Laboratory Home Page2014.
26. Cladis DP, Kleiner AC, Freiser HH, Santerre CR. Fatty Acid Profiles of Commercially Available Finfish Fillets in the United States. Lipids. 2014. PMID: 25108414. http://www.ncbi.nlm.nih.gov/pubmed/25108414.
27. Food and Agriculture Organization of the United Nations; Geneva WHO. Report of the Joint FAO/WHO Expert Consultation on the Risks and Benefits of Fish Consumption. Rome. 2011.
28. Aromataris E, Pearson A. The systematic review: an overview. Am J Nurs. 2014;114(3):53-8. PMID: 24572533. http://www.ncbi.nlm.nih.gov/pubmed/24572533.
29. European Food Safety Authority (EFSA). Food Additives and Nutrient Sources Added to food Panel. Scientific Opinion on the re-evaluation of aspartame (E 951) as a food additive. Parma, Italy,2013. Available from: http://www.efsa.europa.eu/en/efsajournal/doc/3496.pdf.
30. Centers for Disease Control and Prevention. Handwashing: Clean Hands Save Lives 2013. Available from: http://www.cdc.gov/handwashing/.
31. Food and Drug Administration. Food Safety for Moms-to-Be: Safe Eats - Meat, Poultry & Seafood 2014 [cited 2014 June 3]. Available from: http://www.fda.gov/Food/ResourcesForYou/HealthEducators/ucm082294.htm.
32. United States Department of Agriculture. Food Safety Inspection Service. Food Safety Fact Sheets 2013 [cited 2014 June 3]. Available from: http://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets.
33. International Organisation for Standardisation. Environmental management – Life cycle assessment – principles and framework, Geneva, Switzerland 2006a.
34. Aston LM, Smith JN, Powles JW. Impact of a reduced red and processed meat dietary pattern on disease risks and greenhouse gas emissions in the UK: a modelling study. BMJ Open. 2012;2(5). PMID: 22964113. http://www.ncbi.nlm.nih.gov/pubmed/22964113.
35. Baroni L, Cenci L, Tettamanti M, Berati M. Evaluating the environmental impact of various dietary patterns combined with different food production systems. Eur J Clin

Nutr. 2007;61(2):279-86. PMID: 17035955. http://www.ncbi.nlm.nih.gov/pubmed/17035955.

36. Barosh L, Friel S, Engelhardt K, Chan L. The cost of a healthy and sustainable diet--who can afford it? Aust N Z J Public Health. 2014;38(1):7-12. PMID: 24494938. http://www.ncbi.nlm.nih.gov/pubmed/24494938.

37. de Carvalho AM, Cesar CL, Fisberg RM, Marchioni DM. Excessive meat consumption in Brazil: diet quality and environmental impacts. Public Health Nutr. 2013;16(10):1893-9. PMID: 22894818. http://www.ncbi.nlm.nih.gov/pubmed/22894818.

38. Hendrie GA, Ridoutt BG, Wiedmann TO, Noakes M. Greenhouse gas emissions and the Australian diet--comparing dietary recommendations with average intakes. Nutrients. 2014;6(1):289-303. PMID: 24406846. http://www.ncbi.nlm.nih.gov/pubmed/24406846.

39. Macdiarmid JI, Kyle J, Horgan GW, Loe J, Fyfe C, Johnstone A, et al. Sustainable diets for the future: Can we contribute to reducing greenhouse gas emissions by eating a healthy diet? Am J Clin Nutr. 2012;96(3):632-9. PMID: 22854399. http://www.ncbi.nlm.nih.gov/pubmed/22854399.

40. Meier T, Christen O. Environmental impacts of dietary recommendations and dietary styles: Germany as an example. Environ Sci Technol. 2013;47(2):877-88. PMID: 23189920. http://www.ncbi.nlm.nih.gov/pubmed/23189920.

41. Peters CJ, Wilkins JL, Fick GW. Testing a complete-diet model for estimating the land resource requirements of food consumption and agricultural carrying capacity: The New York State example. Renewable agriculture and food systems. 2007;22(2):145-53. http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=1091328&fileId=S1742170507001767.

42. Pimentel D, Pimentel M. Sustainability of meat-based and plant-based diets and the environment. Am J Clin Nutr. 2003;78(3 Suppl):660S-3S. PMID: 12936963. http://www.ncbi.nlm.nih.gov/pubmed/12936963.

43. Pradhan P, Reusser DE, Kropp JP. Embodied greenhouse gas emissions in diets. PLoS One. 2013;8(5):e62228. PMID: 23700408. http://www.ncbi.nlm.nih.gov/pubmed/23700408.

44. Saez-Almendros S, Obrador B, Bach-Faig A, Serra-Majem L. Environmental footprints of Mediterranean versus Western dietary patterns: beyond the health benefits of the Mediterranean diet. Environ Health. 2013;12:118. PMID: 24378069. http://www.ncbi.nlm.nih.gov/pubmed/24378069.

45. Scarborough P, Allender S, Clarke D, Wickramasinghe K, Rayner M. Modelling the health impact of environmentally sustainable dietary scenarios in the UK. Eur J Clin Nutr. 2012;66(6):710-5. PMID: 22491494. http://www.ncbi.nlm.nih.gov/pubmed/22491494.

46. van Dooren C, Marinussen M, Blonk H, Aiking H, Vellinga P. Exploring dietary guidelines based on ecological and nutritional values: A comparison of six dietary patterns. Food Policy. 2014;44(0):36-46. http://www.sciencedirect.com/science/article/pii/S0306919213001620.

47. Vieux F, Soler LG, Touazi D, Darmon N. High nutritional quality is not associated with low greenhouse gas emissions in self-selected diets of French adults. Am J Clin Nutr. 2013;97(3):569-83. PMID: 23364012. http://www.ncbi.nlm.nih.gov/pubmed/23364012.

48. Wilson N, Nghiem N, Ni Mhurchu C, Eyles H, Baker MG, Blakely T. Foods and dietary patterns that are healthy, low-cost, and environmentally sustainable: a case study of

optimization modeling for New Zealand. PLoS One. 2013;8(3):e59648. PMID: 23544082. http://www.ncbi.nlm.nih.gov/pubmed/23544082.

49. Bellows AC, Alcaraz VG, Hallman WK. Gender and food, a study of attitudes in the USA towards organic, local, U.S. grown, and GM-free foods. Appetite. 2010;55(3):540-50. PMID: 20851727. http://www.ncbi.nlm.nih.gov/pubmed/20851727.

50. Blonk H, Ponsioen, T., Kool, A., Marinussen, M. The Agri-Foorprint Method; Methodological LCA Framework, Assumptions and Applied Data, Version 1.0. 2011.

51. International Organisation for Standardisation. ISO 14044: Environmental management – Life cycle assessment – requirements and guidelines 2006b. Available from: http://www.iso.org/iso/catalogue_detail?csnumber=38498.

52. Goedkoop M SR. The Eco-Indicator 99, A Damage Oriented Method for Life Cycle Assessment Methodology Report 2000. Available from: http://teclim.ufba.br/jsf/indicadores/holan%20ecoindicator%2099.pdf.

53. Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. Circulation. 2010;121(21):2271-83. PMID: 20479151. http://www.ncbi.nlm.nih.gov/pubmed/20479151.

54. Norat T CD, Lau R, Aune D, Vieira R. WCRF/AICR Systematic Literature Review Continuous Update Project Report:The Associations between Food, Nutrition and Physical Activity and the Risk of Colorectal Cancer, 2010. Available from: http://www.dietandcancerreport.org/cancer_resource_center/downloads/cu/Colorectal-Cancer-SLR-2010.pdf.

55. Wiseman M. The second World Cancer Research Fund/American Institute for Cancer Research expert report. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Proc Nutr Soc. 2008;67(3):253-6. PMID: 18452640. http://www.ncbi.nlm.nih.gov/pubmed/18452640.

56. U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2010. 7th ed. Washington, DC: U.S. Government Printing Office; December 2010.

57. National Oceanic and Atmospheric Administration. Fisheries of the United States 2011. Available from: http://www.st.nmfs.noaa.gov/st1/fus/fus11/FUS_2011.pdf.

58. Bouman AF BA, Overbeek CC, D. P. Bureau, M. Pawlowski,, Gilbert. aPM. Hindcasts and future projections of global inland and coastal nitrogen and phosphorus loads due to finfish aquaculture. Reviews in Fisheries Science. 2013;21(2):112-56.

59. Hall S, Delaporte A, Phillips MJ, Beveridge M, O'Keefe, M. Blue frontiers: Managing the Environmental Costs of Aquaculture. WorldFish Center, 2011.

60. National Oceanic and Atmospheric Administration. Fish Watch: US Seafood Facts [cited 2014 June 14]. Available from: http://www.fishwatch.gov/farmed_seafood/outside_the_us.htm.

61. Food and Drug Administration. Fish: What Pregnant Women and Parents Should Know: Draft Updated Advice by FDA and EPA 2014. Available from: http://www.fda.gov/Food/FoodborneIllnessContaminants/Metals/ucm393070.htm.

62. United States Environmental Protection Agency. What You Need to Know about Mercury in Fish and Shellfish 2004. Available from: http://water.epa.gov/scitech/swguidance/fishshellfish/outreach/advice_index.cfm.

63. LaComb RP SR, Wilkinson Enns C, Goldman JD. Beverage Choices of U.S. Adults: What We Eat In America, NHANES 2007-2008. 2011.

64. Crippa A, Discacciati A, Larsson SC, Wolk A, Orsini N. Coffee consumption and mortality from all causes, cardiovascular disease, and cancer: a dose-response meta-analysis. Am J Epidemiol. 2014;180(8):763-75. PMID: 25156996. http://www.ncbi.nlm.nih.gov/pubmed/25156996.

65. Je Y, Giovannucci E. Coffee consumption and total mortality: a meta-analysis of twenty prospective cohort studies. Br J Nutr. 2014;111(7):1162-73. PMID: 24279995. http://www.ncbi.nlm.nih.gov/pubmed/24279995.

66. Malerba S, Turati F, Galeone C, Pelucchi C, Verga F, La Vecchia C, et al. A meta-analysis of prospective studies of coffee consumption and mortality for all causes, cancers and cardiovascular diseases. Eur J Epidemiol. 2013;28(7):527-39. PMID: 23934579. http://www.ncbi.nlm.nih.gov/pubmed/23934579.

67. Ding M, Bhupathiraju SN, Satija A, van Dam RM, Hu FB. Long-term coffee consumption and risk of cardiovascular disease: a systematic review and a dose-response meta-analysis of prospective cohort studies. Circulation. 2014;129(6):643-59. PMID: 24201300. http://www.ncbi.nlm.nih.gov/pubmed/24201300.

68. Sofi F, Conti AA, Gori AM, Eliana Luisi ML, Casini A, Abbate R, et al. Coffee consumption and risk of coronary heart disease: a meta-analysis. Nutr Metab Cardiovasc Dis. 2007;17(3):209-23. PMID: 17156982. http://www.ncbi.nlm.nih.gov/pubmed/17156982.

69. Wu JN, Ho SC, Zhou C, Ling WH, Chen WQ, Wang CL, et al. Coffee consumption and risk of coronary heart diseases: a meta-analysis of 21 prospective cohort studies. Int J Cardiol. 2009;137(3):216-25. PMID: 18707777. http://www.ncbi.nlm.nih.gov/pubmed/18707777.

70. Kim B, Nam Y, Kim J, Choi H, Won C. Coffee Consumption and Stroke Risk: A Meta-analysis of Epidemiologic Studies. Korean J Fam Med. 2012;33(6):356-65. PMID: 23267421. http://www.ncbi.nlm.nih.gov/pubmed/23267421.

71. Larsson SC, Orsini N. Coffee consumption and risk of stroke: a dose-response meta-analysis of prospective studies. Am J Epidemiol. 2011;174(9):993-1001. PMID: 21920945. http://www.ncbi.nlm.nih.gov/pubmed/21920945.

72. Mesas AE, Leon-Munoz LM, Rodriguez-Artalejo F, Lopez-Garcia E. The effect of coffee on blood pressure and cardiovascular disease in hypertensive individuals: a systematic review and meta-analysis. Am J Clin Nutr. 2011;94(4):1113-26. PMID: 21880846. http://www.ncbi.nlm.nih.gov/pubmed/21880846.

73. Noordzij M, Uiterwaal CS, Arends LR, Kok FJ, Grobbee DE, Geleijnse JM. Blood pressure response to chronic intake of coffee and caffeine: a meta-analysis of randomized controlled trials. J Hypertens. 2005;23(5):921-8. PMID: 15834273. http://www.ncbi.nlm.nih.gov/pubmed/15834273.

74. Steffen M, Kuhle C, Hensrud D, Erwin PJ, Murad MH. The effect of coffee consumption on blood pressure and the development of hypertension: a systematic review and meta-analysis. J Hypertens. 2012;30(12):2245-54. PMID: 23032138. http://www.ncbi.nlm.nih.gov/pubmed/23032138.

75. Zhang Z, Hu G, Caballero B, Appel L, Chen L. Habitual coffee consumption and risk of hypertension: a systematic review and meta-analysis of prospective observational studies.

Am J Clin Nutr. 2011;93(6):1212-9. PMID: 21450934. http://www.ncbi.nlm.nih.gov/pubmed/21450934.

76. Cai L, Ma D, Zhang Y, Liu Z, Wang P. The effect of coffee consumption on serum lipids: a meta-analysis of randomized controlled trials. Eur J Clin Nutr. 2012;66(8):872-7. PMID: 22713771. http://www.ncbi.nlm.nih.gov/pubmed/22713771.

77. Jee SH, He J, Appel LJ, Whelton PK, Suh I, Klag MJ. Coffee consumption and serum lipids: a meta-analysis of randomized controlled clinical trials. Am J Epidemiol. 2001;153(4):353-62. PMID: 11207153. http://www.ncbi.nlm.nih.gov/pubmed/11207153.

78. Caldeira D, Martins C, Alves LB, Pereira H, Ferreira JJ, Costa J. Caffeine does not increase the risk of atrial fibrillation: a systematic review and meta-analysis of observational studies. Heart. 2013;99(19):1383-9. PMID: 24009307. http://www.ncbi.nlm.nih.gov/pubmed/24009307.

79. Mostofsky E, Rice MS, Levitan EB, Mittleman MA. Habitual coffee consumption and risk of heart failure: a dose-response meta-analysis. Circ Heart Fail. 2012;5(4):401-5. PMID: 22740040. http://www.ncbi.nlm.nih.gov/pubmed/22740040.

80. Ding M, Bhupathiraju SN, Chen M, van Dam RM, Hu FB. Caffeinated and decaffeinated coffee consumption and risk of type 2 diabetes: a systematic review and a dose-response meta-analysis. Diabetes Care. 2014;37(2):569-86. PMID: 24459154. http://www.ncbi.nlm.nih.gov/pubmed/24459154.

81. Huxley R, Lee CM, Barzi F, Timmermeister L, Czernichow S, Perkovic V, et al. Coffee, decaffeinated coffee, and tea consumption in relation to incident type 2 diabetes mellitus: a systematic review with meta-analysis. Arch Intern Med. 2009;169(22):2053-63. PMID: 20008687. http://www.ncbi.nlm.nih.gov/pubmed/20008687.

82. Jiang X, Zhang D, Jiang W. Coffee and caffeine intake and incidence of type 2 diabetes mellitus: a meta-analysis of prospective studies. Eur J Nutr. 2014;53(1):25-38. PMID: 24150256. http://www.ncbi.nlm.nih.gov/pubmed/24150256.

83. van Dam RM, Hu FB. Coffee consumption and risk of type 2 diabetes: a systematic review. Jama. 2005;294(1):97-104. PMID: 15998896. http://www.ncbi.nlm.nih.gov/pubmed/15998896.

84. Whitehead N, White H. Systematic review of randomised controlled trials of the effects of caffeine or caffeinated drinks on blood glucose concentrations and insulin sensitivity in people with diabetes mellitus. J Hum Nutr Diet. 2013;26(2):111-25. PMID: 23331476. http://www.ncbi.nlm.nih.gov/pubmed/23331476.

85. Yu X, Bao Z, Zou J, Dong J. Coffee consumption and risk of cancers: a meta-analysis of cohort studies. BMC Cancer. 2011;11:96. PMID: 21406107. http://www.ncbi.nlm.nih.gov/pubmed/21406107.

86. Tang N, Wu Y, Ma J, Wang B, Yu R. Coffee consumption and risk of lung cancer: a meta-analysis. Lung Cancer. 2010;67(1):17-22. PMID: 19362749. http://www.ncbi.nlm.nih.gov/pubmed/19362749.

87. Sang LX, Chang B, Li XH, Jiang M. Consumption of coffee associated with reduced risk of liver cancer: a meta-analysis. BMC Gastroenterol. 2013;13:34. PMID: 23433483. http://www.ncbi.nlm.nih.gov/pubmed/23433483.

88. Bravi F, Bosetti C, Tavani A, Gallus S, La Vecchia C. Coffee reduces risk for hepatocellular carcinoma: an updated meta-analysis. Clin Gastroenterol Hepatol. 2013;11(11):1413-21.e1. PMID: 23660416. http://www.ncbi.nlm.nih.gov/pubmed/23660416.

89. Jiang W, Wu Y, Jiang X. Coffee and caffeine intake and breast cancer risk: an updated dose-response meta-analysis of 37 published studies. Gynecol Oncol. 2013;129(3):620-9. PMID: 23535278. http://www.ncbi.nlm.nih.gov/pubmed/23535278.
90. Li XJ, Ren ZJ, Qin JW, Zhao JH, Tang JH, Ji MH, et al. Coffee consumption and risk of breast cancer: an up-to-date meta-analysis. PLoS One. 2013;8(1):e52681. PMID: 23308117. http://www.ncbi.nlm.nih.gov/pubmed/23308117.
91. Tang N, Zhou B, Wang B, Yu R. Coffee consumption and risk of breast cancer: a metaanalysis. Am J Obstet Gynecol. 2009;200(3):290.e1-9. PMID: 19114275. http://www.ncbi.nlm.nih.gov/pubmed/19114275.
92. Cao S, Liu L, Yin X, Wang Y, Liu J, Lu Z. Coffee consumption and risk of prostate cancer: a meta-analysis of prospective cohort studies. Carcinogenesis. 2014;35(2):256-61. PMID: 24343360. http://www.ncbi.nlm.nih.gov/pubmed/24343360.
93. Zhong S, Chen W, Yu X, Chen Z, Hu Q, Zhao J. Coffee consumption and risk of prostate cancer: an up-to-date meta-analysis. Eur J Clin Nutr. 2014;68(3):330-7. PMID: 24300907. http://www.ncbi.nlm.nih.gov/pubmed/24300907.
94. Discacciati A, Orsini N, Wolk A. Coffee consumption and risk of nonaggressive, aggressive and fatal prostate cancer--a dose-response meta-analysis. Ann Oncol. 2014;25(3):584-91. PMID: 24276028. http://www.ncbi.nlm.nih.gov/pubmed/24276028.
95. Park CH, Myung SK, Kim TY, Seo HG, Jeon YJ, Kim Y. Coffee consumption and risk of prostate cancer: a meta-analysis of epidemiological studies. BJU Int. 2010;106(6):762-9. PMID: 20590551. http://www.ncbi.nlm.nih.gov/pubmed/20590551.
96. Braem MG, Onland-Moret NC, Schouten LJ, Tjonneland A, Hansen L, Dahm CC, et al. Coffee and tea consumption and the risk of ovarian cancer: a prospective cohort study and updated meta-analysis. Am J Clin Nutr. 2012;95(5):1172-81. PMID: 22440851. http://www.ncbi.nlm.nih.gov/pubmed/22440851.
97. Bravi F, Scotti L, Bosetti C, Gallus S, Negri E, La Vecchia C, et al. Coffee drinking and endometrial cancer risk: a metaanalysis of observational studies. Am J Obstet Gynecol. 2009;200(2):130-5. PMID: 19110217. http://www.ncbi.nlm.nih.gov/pubmed/19110217.
98. Je Y, Giovannucci E. Coffee consumption and risk of endometrial cancer: findings from a large up-to-date meta-analysis. Int J Cancer. 2012;131(7):1700-10. PMID: 22190017. http://www.ncbi.nlm.nih.gov/pubmed/22190017.
99. Zhou Y, Tian C, Jia C. A dose-response meta-analysis of coffee consumption and bladder cancer. Prev Med. 2012;55(1):14-22. PMID: 22564775. http://www.ncbi.nlm.nih.gov/pubmed/22564775.
100. Turati F, Galeone C, Edefonti V, Ferraroni M, Lagiou P, La Vecchia C, et al. A meta-analysis of coffee consumption and pancreatic cancer. Ann Oncol. 2012;23(2):311-8. PMID: 21746805. http://www.ncbi.nlm.nih.gov/pubmed/21746805.
101. Dong J, Zou J, Yu XF. Coffee drinking and pancreatic cancer risk: a meta-analysis of cohort studies. World J Gastroenterol. 2011;17(9):1204-10. PMID: 21448427. http://www.ncbi.nlm.nih.gov/pubmed/21448427.
102. Turati F, Galeone C, La Vecchia C, Garavello W, Tavani A. Coffee and cancers of the upper digestive and respiratory tracts: meta-analyses of observational studies. Ann Oncol. 2011;22(3):536-44. PMID: 20943597. http://www.ncbi.nlm.nih.gov/pubmed/20943597.
103. Zheng JS, Yang J, Fu YQ, Huang T, Huang YJ, Li D. Effects of green tea, black tea, and coffee consumption on the risk of esophageal cancer: a systematic review and meta-

analysis of observational studies. Nutr Cancer. 2013;65(1):1-16. PMID: 23368908. http://www.ncbi.nlm.nih.gov/pubmed/23368908.

104. Botelho F, Lunet N, Barros H. Coffee and gastric cancer: systematic review and meta-analysis. Cad Saude Publica. 2006;22(5):889-900. PMID: 16680342. http://www.ncbi.nlm.nih.gov/pubmed/16680342.

105. Galeone C, Turati F, La Vecchia C, Tavani A. Coffee consumption and risk of colorectal cancer: a meta-analysis of case-control studies. Cancer Causes Control. 2010;21(11):1949-59. PMID: 20680435. http://www.ncbi.nlm.nih.gov/pubmed/20680435.

106. Je Y, Liu W, Giovannucci E. Coffee consumption and risk of colorectal cancer: a systematic review and meta-analysis of prospective cohort studies. Int J Cancer. 2009;124(7):1662-8. PMID: 19115212. http://www.ncbi.nlm.nih.gov/pubmed/19115212.

107. Li G, Ma D, Zhang Y, Zheng W, Wang P. Coffee consumption and risk of colorectal cancer: a meta-analysis of observational studies. Public Health Nutr. 2013;16(2):346-57. PMID: 22694939. http://www.ncbi.nlm.nih.gov/pubmed/22694939.

108. Costa J, Lunet N, Santos C, Santos J, Vaz-Carneiro A. Caffeine exposure and the risk of Parkinson's disease: a systematic review and meta-analysis of observational studies. J Alzheimers Dis. 2010;20 Suppl 1:S221-38. PMID: 20182023. http://www.ncbi.nlm.nih.gov/pubmed/20182023.

109. Ishihara L, Brayne C. A systematic review of nutritional risk factors of Parkinson's disease. Nutr Res Rev. 2005;18(2):259-82. PMID: 19079910. http://www.ncbi.nlm.nih.gov/pubmed/19079910.

110. Qi H, Li S. Dose-response meta-analysis on coffee, tea and caffeine consumption with risk of Parkinson's disease. Geriatr Gerontol Int. 2014;14(2):430-9. PMID: 23879665. http://www.ncbi.nlm.nih.gov/pubmed/23879665.

111. Arab L, Khan F, Lam H. Epidemiologic evidence of a relationship between tea, coffee, or caffeine consumption and cognitive decline. Adv Nutr. 2013;4(1):115-22. PMID: 23319129. http://www.ncbi.nlm.nih.gov/pubmed/23319129.

112. Santos C, Costa J, Santos J, Vaz-Carneiro A, Lunet N. Caffeine intake and dementia: systematic review and meta-analysis. J Alzheimers Dis. 2010;20 Suppl 1:S187-204. PMID: 20182026. http://www.ncbi.nlm.nih.gov/pubmed/20182026.

113. Greenwood DC, Thatcher NJ, Ye J, Garrard L, Keogh G, King LG, et al. Caffeine intake during pregnancy and adverse birth outcomes: a systematic review and dose-response meta-analysis. Eur J Epidemiol. 2014;29(10):725-34. PMID: 25179792. http://www.ncbi.nlm.nih.gov/pubmed/25179792.

114. Maslova E, Bhattacharya S, Lin SW, Michels KB. Caffeine consumption during pregnancy and risk of preterm birth: a meta-analysis. Am J Clin Nutr. 2010;92(5):1120-32. PMID: 20844077. http://www.ncbi.nlm.nih.gov/pubmed/20844077.

115. Peck JD, Leviton A, Cowan LD. A review of the epidemiologic evidence concerning the reproductive health effects of caffeine consumption: a 2000-2009 update. Food Chem Toxicol. 2010;48(10):2549-76. PMID: 20558227. http://www.ncbi.nlm.nih.gov/pubmed/20558227.

116. Bech BH, Obel C, Henriksen TB, Olsen J. Effect of reducing caffeine intake on birth weight and length of gestation: randomised controlled trial. BMJ. 2007;334(7590):409. PMID: 17259189. http://www.ncbi.nlm.nih.gov/pubmed/17259189.

117. Burrows T, Pursey K, Neve M, Stanwell P. What are the health implications associated with the consumption of energy drinks? A systematic review. Nutr Rev. 2013;71(3):135-48. PMID: 23452281. http://www.ncbi.nlm.nih.gov/pubmed/23452281.
118. Goldfarb M, Tellier C, Thanassoulis G. Review of published cases of adverse cardiovascular events after ingestion of energy drinks. Am J Cardiol. 2014;113(1):168-72. PMID: 24176062. http://www.ncbi.nlm.nih.gov/pubmed/24176062.
119. Government of Canada. Health Canada Health Products. Food Branch. Food Directorate. Caffeine in Foods 2012 [updated 2012-02-16]. Available from: http://www.hc-sc.gc.ca/fn-an/securit/addit/caf/food-caf-aliments-eng.php.
120. Pomeranz JL, Munsell CR, Harris JL. Energy drinks: an emerging public health hazard for youth. J Public Health Policy. 2013;34(2):254-71. PMID: 23486464. http://www.ncbi.nlm.nih.gov/pubmed/23486464.
121. Seifert SM, Schaechter JL, Hershorin ER, Lipshultz SE. Health effects of energy drinks on children, adolescents, and young adults. Pediatrics. 2011;127(3):511-28. PMID: 21321035. http://www.ncbi.nlm.nih.gov/pubmed/21321035.
122. Reissig CJ, Strain EC, Griffiths RR. Caffeinated energy drinks--a growing problem. Drug Alcohol Depend. 2009;99(1-3):1-10. PMID: 18809264. http://www.ncbi.nlm.nih.gov/pubmed/18809264.
123. Institute of Medicine. Caffeine in Food and Dietary Supplements: Examining Safety: Workshop Summary. Washington, DC: The National Academies Press; 2014. Available from: http://www.nap.edu/openbook.php?record_id=18607.
124. Branum AM, Rossen LM, Schoendorf KC. Trends in caffeine intake among U.S. children and adolescents. Pediatrics. 2014;133(3):386-93. PMID: 24515508. http://www.ncbi.nlm.nih.gov/pubmed/24515508.
125. Terry-McElrath YM, O'Malley PM, Johnston LD. Energy drinks, soft drinks, and substance use among United States secondary school students. J Addict Med. 2014;8(1):6-13. PMID: 24481080. http://www.ncbi.nlm.nih.gov/pubmed/24481080.
126. Arria AM, O'Brien MC. The "high" risk of energy drinks. Jama. 2011;305(6):600-1. PMID: 21266673. http://www.ncbi.nlm.nih.gov/pubmed/21266673.
127. Centers for Disease Control and Prevention. Caffeine and Alcohol. 2014. Available from: http://www.cdc.gov/alcohol/fact-sheets/cab.htm.
128. Substance Abuse Mental Health Services Administration. Drug Abuse Warning Network (DAWN), 2011: National Estimates of Drug-Related Emergency Department Visits.HHS. Publication No. (SMA) 13-4760, DAWN Series D-39 Rockville, MD2013. Available from: http://www.samhsa.gov/data/2k13/DAWN2k11ED/DAWN2k11ED.htm.
129. Ferreira SE, de Mello MT, Pompéia S, de Souza-Formigoni ML. Effects of energy drink ingestion on alcohol intoxication. Alcohol Clin Exp Res. 2006;30(4):598-605. PMID: 16573577. http://www.ncbi.nlm.nih.gov/pubmed/16573577.
130. Wiklund U, Karlsson M, Ostrom M, Messner T. Influence of energy drinks and alcohol on post-exercise heart rate recovery and heart rate variability. Clin Physiol Funct Imaging. 2009;29(1):74-80. PMID: 19016812. http://www.ncbi.nlm.nih.gov/pubmed/19016812.
131. Food and Drug Administration. Food additives permitted for direct addition to food for human consumption: aspartame. Federal Register1984.
132. Magnuson BA, Burdock GA, Doull J, Kroes RM, Marsh GM, Pariza MW, et al. Aspartame: a safety evaluation based on current use levels, regulations, and toxicological

and epidemiological studies. Crit Rev Toxicol. 2007;37(8):629-727. PMID: 17828671. http://www.ncbi.nlm.nih.gov/pubmed/17828671.

133. Food And Drug Administration. Food Additives Permitted for Direct Addition to Food for Human Consumption 2014. Available from: http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/cfrsearch.cfm?fr=172.804.

134. Andreatta MM, Munoz SE, Lantieri MJ, Eynard AR, Navarro A. Artificial sweetener consumption and urinary tract tumors in Cordoba, Argentina. Prev Med. 2008;47(1):136-9. PMID: 18495230. http://www.ncbi.nlm.nih.gov/pubmed/18495230.

135. Bosetti C, Gallus S, Talamini R, Montella M, Franceschi S, Negri E, et al. Artificial sweeteners and the risk of gastric, pancreatic, and endometrial cancers in Italy. Cancer Epidemiol Biomarkers Prev. 2009;18(8):2235-8. PMID: 19661082. http://www.ncbi.nlm.nih.gov/pubmed/19661082.

136. Bunin GR, Kushi LH, Gallagher PR, Rorke-Adams LB, McBride ML, Cnaan A. Maternal diet during pregnancy and its association with medulloblastoma in children: a children's oncology group study (United States). Cancer Causes Control. 2005;16(7):877-91. PMID: 16132798. http://www.ncbi.nlm.nih.gov/pubmed/16132798.

137. Cabaniols C, Giorgi R, Chinot O, Ferahta N, Spinelli V, Alla P, et al. Links between private habits, psychological stress and brain cancer: a case-control pilot study in France. J Neurooncol. 2011;103(2):307-16. PMID: 20835749. http://www.ncbi.nlm.nih.gov/pubmed/20835749.

138. Gallus S, Scotti L, Negri E, Talamini R, Franceschi S, Montella M, et al. Artificial sweeteners and cancer risk in a network of case-control studies. Ann Oncol. 2007;18(1):40-4. PMID: 17043096. http://www.ncbi.nlm.nih.gov/pubmed/17043096.

139. Hardell L, Mild KH, Pahlson A, Hallquist A. Ionizing radiation, cellular telephones and the risk for brain tumours. Eur J Cancer Prev. 2001;10(6):523-9. PMID: 11916351. http://www.ncbi.nlm.nih.gov/pubmed/11916351.

140. Lim U, Subar AF, Mouw T, Hartge P, Morton LM, Stolzenberg-Solomon R, et al. Consumption of aspartame-containing beverages and incidence of hematopoietic and brain malignancies. Cancer Epidemiol Biomarkers Prev. 2006;15(9):1654-9. PMID: 16985027. http://www.ncbi.nlm.nih.gov/pubmed/16985027.

141. Schernhammer ES, Bertrand KA, Birmann BM, Sampson L, Willett WC, Feskanich D. Consumption of artificial sweetener- and sugar-containing soda and risk of lymphoma and leukemia in men and women. Am J Clin Nutr. 2012;96(6):1419-28. PMID: 23097267. http://www.ncbi.nlm.nih.gov/pubmed/23097267.

142. Halldorsson TI, Strom M, Petersen SB, Olsen SF. Intake of artificially sweetened soft drinks and risk of preterm delivery: a prospective cohort study in 59,334 Danish pregnant women. Am J Clin Nutr. 2010;92(3):626-33. PMID: 20592133. http://www.ncbi.nlm.nih.gov/pubmed/20592133.

143. Englund-Ogge L, Brantsaeter AL, Haugen M, Sengpiel V, Khatibi A, Myhre R, et al. Association between intake of artificially sweetened and sugar-sweetened beverages and preterm delivery: a large prospective cohort study. Am J Clin Nutr. 2012;96(3):552-9. PMID: 22854404. http://www.ncbi.nlm.nih.gov/pubmed/22854404.

144. La Vecchia C. Low-calorie sweeteners and the risk of preterm delivery: results from two studies and a meta-analysis. J Fam Plann Reprod Health Care. 2013;39(1):12-3. PMID: 23296849. http://www.ncbi.nlm.nih.gov/pubmed/23296849.

145. Shaywitz BA, Anderson GM, Novotny EJ, Ebersole JS, Sullivan CM, Gillespie SM. Aspartame has no effect on seizures or epileptiform discharges in epileptic children. Ann Neurol. 1994;35(1):98-103. PMID: 7506878. http://www.ncbi.nlm.nih.gov/pubmed/7506878.

146. Wolraich ML, Lindgren SD, Stumbo PJ, Stegink LD, Appelbaum MI, Kiritsy MC. Effects of diets high in sucrose or aspartame on the behavior and cognitive performance of children. N Engl J Med. 1994;330(5):301-7. PMID: 8277950. http://www.ncbi.nlm.nih.gov/pubmed/8277950.

147. Kruesi MJ, Rapoport JL, Cummings EM, Berg CJ, Ismond DR, Flament M, et al. Effects of sugar and aspartame on aggression and activity in children. Am J Psychiatry. 1987;144(11):1487-90. PMID: 3674234. http://www.ncbi.nlm.nih.gov/pubmed/3674234.

148. Roshon MS, Hagen RL. Sugar consumption, locomotion, task orientation, and learning in preschool children. J Abnorm Child Psychol. 1989;17(3):349-57. PMID: 2666476. http://www.ncbi.nlm.nih.gov/pubmed/2666476.

149. Lapierre KA, Greenblatt DJ, Goddard JE, Harmatz JS, Shader RI. The neuropsychiatric effects of aspartame in normal volunteers. J Clin Pharmacol. 1990;30(5):454-60. PMID: 2347957. http://www.ncbi.nlm.nih.gov/pubmed/2347957.

150. Ryan-Harshman M, Leiter LA, Anderson GH. Phenylalanine and aspartame fail to alter feeding behavior, mood and arousal in men. Physiol Behav. 1987;39(2):247-53. PMID: 3575461. http://www.ncbi.nlm.nih.gov/pubmed/3575461.

151. Spiers PA, Sabounjian L, Reiner A, Myers DK, Wurtman J, Schomer DL. Aspartame: neuropsychologic and neurophysiologic evaluation of acute and chronic effects. Am J Clin Nutr. 1998;68(3):531-7. PMID: 9734727. http://www.ncbi.nlm.nih.gov/pubmed/9734727.

152. Stokes AF, Belger A, Banich MT, Bernadine E. Effects of alcohol and chronic aspartame ingestion upon performance in aviation relevant cognitive tasks. Aviat Space Environ Med. 1994;65(1):7-15. PMID: 8117231. http://www.ncbi.nlm.nih.gov/pubmed/8117231.

153. Stokes AF, Belger A, Banich MT, Taylor H. Effects of acute aspartame and acute alcohol ingestion upon the cognitive performance of pilots. Aviat Space Environ Med. 1991;62(7):648-53. PMID: 1898300. http://www.ncbi.nlm.nih.gov/pubmed/1898300.

154. Pivonka EE, Grunewald KK. Aspartame- or sugar-sweetened beverages: effects on mood in young women. J Am Diet Assoc. 1990;90(2):250-4. PMID: 2303661. http://www.ncbi.nlm.nih.gov/pubmed/2303661.

155. Walton RG, Hudak R, Green-Waite RJ. Adverse reactions to aspartame: double-blind challenge in patients from a vulnerable population. Biol Psychiatry. 1993;34(1-2):13-7. PMID: 8373935. http://www.ncbi.nlm.nih.gov/pubmed/8373935.

156. Schiffman SS, Buckley CE, 3rd, Sampson HA, Massey EW, Baraniuk JN, Follett JV, et al. Aspartame and susceptibility to headache. N Engl J Med. 1987;317(19):1181-5. PMID: 3657889. http://www.ncbi.nlm.nih.gov/pubmed/3657889.

157. Koehler SM, Glaros A. The effect of aspartame on migraine headache. Headache. 1988;28(1):10-4. PMID: 3277925. http://www.ncbi.nlm.nih.gov/pubmed/3277925.

158. Van den Eeden SK, Koepsell TD, Longstreth WT, Jr., van Belle G, Daling JR, McKnight B. Aspartame ingestion and headaches: a randomized crossover trial. Neurology. 1994;44(10):1787-93. PMID: 7936222. http://www.ncbi.nlm.nih.gov/pubmed/7936222.

159. Lipton RB, Newman LC, Cohen JS, Solomon S. Aspartame as a dietary trigger of headache. Headache. 1989;29(2):90-2. PMID: 2708042. http://www.ncbi.nlm.nih.gov/pubmed/2708042.
160. Camfield PR, Camfield CS, Dooley JM, Gordon K, Jollymore S, Weaver DF. Aspartame exacerbates EEG spike-wave discharge in children with generalized absence epilepsy: a double-blind controlled study. Neurology. 1992;42(5):1000-3. PMID: 1579221. http://www.ncbi.nlm.nih.gov/pubmed/1579221.
161. Rowan AJ, Shaywitz BA, Tuchman L, French JA, Luciano D, Sullivan CM. Aspartame and seizure susceptibility: results of a clinical study in reportedly sensitive individuals. Epilepsia. 1995;36(3):270-5. PMID: 7614911. http://www.ncbi.nlm.nih.gov/pubmed/7614911.
162. Centers for Disease Control and Prevention. CDC Estimates of Foodborne Illness in the United States 2014. Available from: http://www.cdc.gov/foodborneburden/estimates-overview.html.

Part D. Chapter 6: Cross-Cutting Topics of Public Health Importance

INTRODUCTION

The *Dietary Guidelines for Americans, 2010* included guidance on sodium, saturated fat, and added sugars, and the 2015 DGAC determined that a reexamination of the evidence on these topics was necessary to evaluate whether revisions to the guidance were warranted. These topics were considered to be of public health importance because each has been associated with negative health outcomes when over-consumed. As the Committee considered it essential to address these topics across two or more Subcommittees, Working Groups were formed with representatives from the relevant Subcommittees to ensure that the topics were thoroughly addressed in a coordinated way. Additionally, the Committee acknowledged that a potential unintended consequence of a recommendation on added sugars might be that consumers and manufacturers replace added sugars with low-calorie sweeteners. As a result, the Committee also examined evidence on low-calorie sweeteners to inform statements on this topic. The updated findings in this chapter will help inform recommendations on these topics for the *2015 Dietary Guidelines for Americans*.

Although sodium, saturated fat, and added sugars are receiving particular focus here, it is important to consider these aspects of the diet in the context of a healthy dietary pattern. A healthy dietary pattern has little room for sodium, saturated fat, and added sugars. That said, these components of the diet are modifiable, and strategies at various levels of the socio-ecologic model, ranging from policy to consumer education, can promote shifts in intake to support healthy dietary patterns.

The sodium, saturated fat, and added sugars sections of this chapter provide introductory text related to the topic including the rationale and approach for the Committee's review. Because the questions within each topic are so complementary, the DGAC choose to develop only one implications section for each topic.

LIST OF QUESTIONS

Sodium

1. What is the relationship between sodium intake and blood pressure in adults?
2. What is the relationship between sodium intake and blood pressure in children?
3. What is the relationship between sodium intake and cardiovascular disease outcomes?

4. What effect does the interrelationship of sodium and potassium have on blood pressure and cardiovascular disease outcomes?

Saturated Fat

5. What is the relationship between intake of saturated fat and risk of cardiovascular disease?

Added Sugars and Low-Calorie Sweeteners

6. What is the relationship between the intake of added sugars and cardiovascular disease, body weight/obesity, type 2 diabetes, and dental caries?
7. What is the relationship between the intake of low-calorie sweeteners and body weight/obesity and type 2 diabetes?

METHODOLOGY

To answer the questions in this chapter, the Committee relied on existing reports, original Nutrition Evidence Library (NEL) systematic reviews, and NEL updates. The Committee followed the methods described in ***Part C. Methodology*** without modification to answer these questions. Because the DGAC knew strong existing reports, systematic reviews (SRs), and meta-analyses (MA) were available related to most of the cross-cutting questions, to prevent duplication of efforts, the DGAC relied on these reviews in lieu of conducting original NEL systematic reviews. In some cases, existing reviews, SRs, or MA were not available or required updating. In these cases, NEL systematic reviews or updates were conducted. Complete information on the NEL reviews and updates is provided at www.NEL.gov. The reader also is directed to the original existing reports, which are referenced throughout the chapter, for additional information.

Four questions addressed dietary sodium intake. For Question 1, the Committee used the 2013 National Heart, Lung, and Blood Institute (NHLBI) *Lifestyle Interventions to Reduce Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group*[1] and the associated American Heart Association (AHA)/ American College of Cardiology (ACC) *Guideline on Lifestyle Management to Reduce Cardiovascular Risk*.[2] Although new studies examining the relationship between sodium and blood pressure have been published since the completion of the NHLBI review, including findings from the Prospective Urban Rural Epidemiology (PURE) study,[3] the Committee determined the evidence presented in the SR conducted by NHLBI, linking sodium and blood pressure, was strong and that consideration of more recent findings would not change the conclusions. Thus, the Committee did not update the review. For Question 2, the Committee updated the NEL systematic review on sodium and blood pressure in children conducted by the 2010 DGAC. The data reviewed for this question by the 2010 DGAC included children, birth to age 18, and the 2015 DGAC updated the sodium review using the same age range. For Question 3, the Committee relied on the NHLBI systematic review from the Lifestyle Work Group[1] as well as the 2013 Institute of Medicine (IOM) report, *Sodium Intake in Populations*.[4] Additionally, because the quality and quantity of the evidence on sodium and cardiovascular disease (CVD) that was used in the two reports is limited, the Committee updated the sodium and CVD review using a NEL systematic review update from January 2013 to July 2014. The final question in the sodium section, Question 4, also was answered using the recent NHLBI systematic review from the Lifestyle Work Group.[1] The Committee also used the 2010 IOM Report on *Strategies to Reduce Sodium Intake in the United States* to inform the implications statements for these questions.[5]

Regarding saturated fat, Question 5 was answered using the NHLBI systematic review[1] and related AHA/ACC *Guideline on Lifestyle Management to Reduce Cardiovascular Risk*,[2] which focused on randomized controlled trials (RCTs), as well as existing SRs and MA addressing this question published in peer-reviewed literature between January 2009 and August 2014. Particular emphasis was placed on reviews that examined the macronutrient replacement for saturated fat.

The remaining questions in this chapter examined added sugars and low-calorie sweeteners. For Question 6, the DGAC relied on systematic reviews commissioned by the World Health Organization (WHO) to address body weight[6] and dental caries.[7] Additionally, to capture new research, the Committee searched for SRs and MA published since January 2012, the completion of the WHO reviews. Type 2 diabetes was not addressed by the WHO, and therefore, the Committee relied on existing SRs/MA published since January 2010 to address this health outcome. No existing SRs/MA examine added sugars and CVD, so the Committee conducted an original NEL systematic review to address this question (see http://NEL.gov/topic.cfm?cat=3376 for complete information on this review). Question 7 on low-calorie sweeteners was answered using existing SRs/MA published from January 2010 to August 2014. For low-calorie sweeteners, the Committee was initially interested in the health outcomes of body weight, type 2 diabetes, CVD, and dental caries. However, existing reviews were available only for body weight and type 2 diabetes. The Committee did not conduct an original NEL systematic review on CVD or dental caries because of limited time and resources, and because the Committee did not think sufficient evidence was available to address these health outcomes.

SODIUM

Introduction

From its first edition in 1980, the *Dietary Guidelines for Americans* consistently recommended the public reduce dietary sodium intakes in order to prevent and treat hypertension, CVD, and stroke. This recommendation is based on evidence supporting a dose-dependent relationship between sodium intake and blood pressure and observational data identifying associations between sodium intake and blood pressure and cardiovascular outcomes. However, despite many years of accumulating evidence and public health guidelines focused on changing individual behavior to achieve a reduced sodium intake among Americans, consumption continues to far exceed recommendations. The DGAC has identified dietary sodium as a nutrient of public health concern because of overconsumption, with usual intakes for those ages 2 years and older at 3,463 mg/day.[8] Sodium is ubiquitous in the current U.S. food supply and multiple food categories contribute to excessive sodium intake (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends,*** Figure D1.35).

Currently, 30 percent of U.S. adults have high blood pressure (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). Furthermore, the estimated lifetime risk of developing hypertension in the U.S. is 90%. The rate of borderline high blood pressure (defined as a systolic or diastolic blood pressure ≥90th percentile but <95th percentile or blood pressure levels ≥120/80 mm Hg) in youth ages 8 to 17 years is highest in those who are obese (16.2 percent), slightly lower in those who are overweight (11 percent); and this condition is present even in those who are normal weight (5 percent). Dietary sodium reduction can effectively prevent and reduce high blood pressure.[9-11] Given the long-standing awareness of this health concern and scientific foundation for dietary treatment, the DGAC conducted a focused review of dietary sodium and its relationship with blood pressure as well as its relationship with CVD.

Question 1: What is the relationship between sodium intake and blood pressure in adults?

Source of evidence: Existing reports

Conclusions

The DGAC concurs with the three conclusions from the 2013 AHA/ACC Lifestyle Guideline that apply to <u>adults who would benefit from blood pressure lowering</u>.

The DGAC concurs that adults who would benefit from blood pressure lowering should “lower sodium intake.” AHA/ACC Grade: Strong; **DGAC Grade: Strong**

The DGAC concurs that adults who would benefit from blood pressure lowering should “Consume no more than 2,400 mg of sodium/day.” The report also indicates that “Further reduction of sodium intake to 1,500 mg/d can result in even greater reduction in blood pressure”;

and concludes that "Even without achieving these goals, reducing sodium intake by at least 1,000 mg/d lowers blood pressure." AHA/ACC Grade: Moderate; **DGAC Grade: Moderate**

The DGAC concurs that adults who would benefit from blood pressure lowering should "Combine the DASH dietary pattern with lower sodium intake." AHA/ACC Grade: Strong; **DGAC Grade: Strong**

Review of the Evidence

The 2013 AHA/ACC Lifestyle Guideline and associated NHLBI Lifestyle Report summarized strong and consistent evidence that supports dietary sodium reduction as a means to prevent and treat high blood pressure. The studies used to inform the conclusion to lower sodium intake were conducted in older and younger adults, individuals with prehypertension and hypertension, men and women, and African American and non-African American adults. The trials also documented positive effects of sodium reduction that were independent of weight change; and include behavioral interventions where individuals were counseled to reduce sodium, as well as feeding studies.

The recommendation to combine the DASH dietary pattern with lower sodium is based heavily on the results of the DASH sodium trial, which showed clinically significant lowering of blood pressure with sodium intake of 2,400 mg/day and even lower blood pressure with sodium intake of 1,500 mg/day. The goal of 2,400 or less mg/day was selected because it is the estimated average urinary sodium excretion in the DASH sodium trial.

The recommendation to reduce sodium intake by 1,000 mg/day even if goals for 2,400 mg/day or 1,500 mg/day cannot be reached comes from studies where this level of sodium reduction was beneficial for blood pressure lowering.

The differences in the evidence grade for the three conclusions related to sodium and blood pressure in adults results from the differences in the number and power of clinical trials supporting each recommendation. For example, a grade of "moderate" was assigned to the second conclusion because fewer clinical trials informed the goals of 2,400 and 1,500 mg/day than for the overall goal of sodium reduction.

For additional details on this body of evidence, visit: References 1, 2, 4 and 9 and ***Appendix E-2.42***

Question 2: What is the relationship between sodium intake and blood pressure in children?

Source of evidence: Existing systematic review with a NEL systematic review update

Conclusions

The 2015 DGAC concurs with the 2010 DGAC that "a moderate body of evidence has documented that as sodium intake decreases, so does blood pressure in children, birth to age 18 years." **DGAC Grade: Moderate**

Review of the Evidence

The 2010 DGAC conducted a systematic review to examine the relationship between sodium intake and blood pressure in children from birth to age 18 years, examining studies published from January 1970 to May 2009. That systematic review included 19 articles from 15 intervention studies and four prospective cohort studies.

The 2015 DGAC updated this systematic review and identified two additional articles published since May 2009, including one RCT and one prospective cohort study.[12, 13]

The 2015 DGAC considered the evidence reviewed by the 2010 DGAC related to dietary sodium intake and blood pressure in children, and determined that, based on the two new studies identified in the updated search, changes were not warranted to the conclusion statement or grade. In aggregate, the data reviewed by the 2010 DGAC indicated that sodium reduction modestly lowers BP in infants and children. Neither of the two studies identified in the update found a relationship between dietary sodium intake and blood pressure in healthy, normotensive children.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250452

Question 3: What is the relationship between sodium intake and cardiovascular disease outcomes?

Source of evidence: Existing report with a NEL systematic review update

Conclusions

The DGAC concurs with the IOM Report: *Sodium Intake in Populations*, which concluded that "although the reviewed evidence on associations between sodium intake and direct health outcomes has methodological flaws and limitations, when considered collectively, it indicates a positive relationship between higher levels of sodium intake and risk of CVD. This evidence is consistent with existing evidence on blood pressure as a surrogate indicator of CVD risk." IOM Grade: Grade not determined, outside the statement of task; **DGAC Grade: Moderate**

The DGAC concurs with the IOM Report: *Sodium Intake in Populations* that "evidence from studies on direct health outcomes is inconsistent and insufficient to conclude that lowering sodium intakes below 2,300 mg/day either increases or decreases risk of CVD outcomes (including stroke and CVD mortality) or all-cause mortality in the general U.S. population." IOM Grade: Grade not determined, outside the statement of task; **DGAC Grade: Grade not assignable**

The DGAC concurs with the NHLBI Lifestyle Report, which concluded that "a reduction in sodium intake by approximately 1,000 mg/day reduces CVD events by about 30 percent" and that "higher dietary sodium intake is associated with a greater risk for fatal and nonfatal stroke and CVD." NHLBI Strength of Evidence: Low; **DGAC Grade: Limited**

The DGAC concurs with the NHLBI Lifestyle Report that "evidence is not sufficient to determine the association between sodium intake and the development of heart failure." NHLBI Strength of Evidence: Not assigned due to insufficient evidence; **DGAC Grade: Grade not Assignable**

Review of the Evidence

The DGAC updated systematic reviews done in 2013 by the IOM[4] and NHLBI,[1] and identified four additional articles published since 2013, all of which were prospective cohort studies.[14-17]

Of note, the evidence reviewed for the 2013 IOM report was published between 2003 and December 2012. The DGAC concluded that the reviewed evidence on associations between sodium intake and direct health outcomes has methodological flaws and limitations. Specifically, the Committee documented the small number of well-conducted studies evaluating sodium intake and direct health outcomes; the inconsistency in findings across the published literature, possibly due to methodological factors; the lack of comparability in sodium intake levels across studies particularity in international studies; and the absence of strong data related to sodium goals and direct health outcomes, not including hypertension.

The DGAC considered the conclusions reached by the IOM and NHLBI related to dietary sodium intake and risk of CVD, and determined that the findings from the four new studies identified in the updated search did not warrant changes to the conclusion statements. In aggregate, the data indicate a relationship between higher sodium intake and higher risk of CVD.

For additional details on this body of evidence, visit:
http://NEL.gov/conclusion.cfm?conclusion_statement_id=250457

Question 4: What effect does the interrelationship of sodium and potassium have on blood pressure and cardiovascular disease outcomes?

Source of evidence: Existing report

Conclusions

The DGAC concurs with the NHLBI Lifestyle Report that: "Evidence is not sufficient to determine whether increasing dietary potassium intake lowers blood pressure." NHLBI Strength of Evidence: Not assigned due to insufficient evidence; **DGAC Grade: Not Assignable**

The DGAC concurs with the NHLBI Lifestyle Report that: "In observational studies with appropriate adjustments (e.g., blood pressure, sodium intake), higher dietary potassium intake is associated with lower risk for stroke." NHLBI Strength of Evidence: Low; **DGAC Grade: Limited**

The DGAC concurs with the NHLBI Lifestyle Report that: "Evidence is not sufficient to determine an association between dietary potassium intake and coronary heart disease (CHD), heart failure, and cardiovascular mortality." NHLBI Strength of Evidence: Not assigned due to insufficient evidence; **DGAC Grade: Grade not Assignable**

Review of the Evidence

The NHLBI Lifestyle Report summarized limited evidence on the relationship between potassium intake and blood pressure, CHD, heart failure, cardiovascular mortality, or stroke. Although it is postulated that a high ratio of sodium intake to potassium intake is a stronger risk factor for hypertension than either factor alone, the evidence base to support this hypothesis is insufficient for drawing definitive conclusions. Although results of epidemiologic studies suggest that potassium consumption influences the risk of CVD, the strength of the evidence is insufficient to draw conclusions about CHD, heart failure, or cardiovascular mortality. The evidence is limited with regard to stroke, coming from studies with weaker designs in which investigators were able to make appropriate statistical adjustments for potential confounders of the relationship.

For additional details on this body of evidence, visit: References 1 and 2

Implications

The current average sodium intake in the United States is 3,478 mg/d, far exceeding recommendations. Given the well-documented relationship between sodium intake and high blood pressure, sodium intake should be reduced and combined with a healthful dietary pattern (as described in ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***).

The general population, ages 2 years and older, should rely on the recommendations of the IOM Panel on Dietary Reference Intakes for Electrolytes and Water.[9] A tolerable upper limit was set by the Panel at 2,300 mg/day based on evidence showing associations between high sodium intake, high blood pressure, and subsequent risk of heart disease, stroke, and mortality. Of note, the AHA/ACC recommendation of less than 2,400 mg/day (see conclusions for sodium question 1) is slightly different than the less than 2,300 mg/day recommended by the IOM Panel on Dietary Reference Intakes or the 2010 Dietary Guidelines for Americans; less than 2,400 mg/day was selected because it was the estimated average urinary sodium excretion in the DASH-sodium trial.

Individuals who would benefit from blood pressure lowering (i.e., those with prehypertension or hypertension), should rely on the recommendations in the 2013 AHA/ACC Lifestyle Guideline. These include: lowering sodium intake in general; or consuming no more than 2,400 mg of sodium/day; or lowering sodium intake to 1,500 mg per day for even greater reduction in blood pressure; or lowering sodium intake by at least 1,000 mg per day even if the goals of 2,400 or 1,500 mg per day cannot be met.

For decades, sodium intake in the United States has exceeded recommendations in spite of numerous national campaigns, through programs such as the NHLBI's National High Blood Pressure Education Program and the CDC's State Heart Disease and Stroke Prevention Program, focused on individual behavior change for sodium reduction. As described in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***, sodium is ubiquitous in the U.S. food supply and almost all food categories contribute to intake levels. This unique feature of sodium makes it difficult for individuals to achieve recommended intake. As such, we recommend that a <u>primary emphasis</u> be placed on policies and population-based strategies for sodium reduction while at the same time paying attention to consumer education. Local, state, and Federal agencies should consider a comprehensive and coordinated strategy, that includes partnerships with the food industry, to reduce the sodium content of foods in the United States based on the socio-ecological model highlighted in the 2015 DGAC's conceptual model (see ***Part B. Chapter 1: Introduction***).

These strategies should be consistent with the recommendation described in the 2010 IOM report on *Strategies to Reduce Sodium Intake in the United States*.[5] The primary strategy that was recommended is that "The FDA should expeditiously initiate a process to set mandatory national standards for the sodium content of foods". This would include: 1) "a modification of the generally recognized as safe (GRAS) status of salt added to processed foods in order to reduce the salt content of the food supply in a stepwise manner"; 2) "FDA should likewise extend its stepwise application of the GRAS modification, adjusted as necessary, to encompass salt added to menu items offered by restaurant/foodservice operations that are sufficiently standardized so as to allow practical implementation"; and 3) "FDA should revisit the GRAS status of other sodium-containing compounds as well as any food additive provisions for such compounds and make adjustments as appropriate, consistent with changes for salt in processed foods and restaurant/foodservice menu items."

Population sodium reductions efforts should consider: 1) the varied technical and functional roles that sodium plays in foods and the complexity of reducing sodium in foods; 2) the recent accomplishments and voluntary reduction efforts by the food industry; and 3) consumer demand for lower-sodium products. More information about strategies for reducing sodium intake in the United States can be found in the IOM report, at http://www.iom.edu/Reports/2010/Strategies-to-Reduce-Sodium-Intake-in-the-United-States.aspx.

Informative food labels should be used to effectively promote awareness of sodium content in foods. Consumers would benefit from a standardized, easily understood front-of-package (FOP) label on all food and beverage products to give clear guidance about a food's healthfulness. An example is the FOP label recommended by the IOM,[18] which included calories, and 0 to 3 "nutritional" points for added sugars, saturated fat, and sodium. This would be integrated with the Nutrition Facts Panel, allowing consumers to quickly and easily identify nutrients of concern for over-consumption, in order to make healthier choices.

Public-private-community partnerships should be created to reduce sodium levels in commercially processed and restaurant foods.

Strategies that complement policies and support consumers to make dietary behavior changes also are needed. These include (but are not limited to): 1) nutrition services and comprehensive lifestyle interventions by multidisciplinary teams;[2] 2) widely available diet planning tools that include sodium as an area of focus; and 3) educational programs that teach adults simple recipes that emphasize flavoring unsalted foods with spices and herbs.

Although the evidence on potassium and blood pressure is limited, the DGAC recognizes potassium as a nutrient of concern (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***) and encourages increased potassium intake through potassium-rich foods such as vegetables and fruits (see Table D1.7).

Interventions, preferably nonpharmacologic, are needed for children because borderline high blood pressure occurs concomitantly with overweight, obesity, and other cardio-metabolic risk factors (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). Evidence-based strategies in clinical and public health settings need to be implemented and complemented by environmental approaches to reverse these high priority health problems in children.

For blood pressure lowering and hypertension prevention, action is needed at both the individual and population levels.

Sodium reduction in youth will require changes in their food environments and school and community-based education on healthful eating.

School systems should adopt mandatory age-appropriate nutrition and physical activity curricula (K-12) that incorporate the core principles of the future *2015 Dietary Guidelines*.

SATURATED FAT

Introduction

The relationship between different types of dietary fats and risk of CVD has been extensively studied in RCTs and epidemiologic studies. It is now well-established that higher intake of *trans* fat from partially hydrogenated vegetable oils is associated with increased risk of CVD and thus, should be minimized in the diet. Numerous RCTs have demonstrated that saturated fat (SFA) as compared to mono- (MUFA) or polyunsaturated fats (PUFA) or carbohydrates increases total and LDL cholesterol. Thus, limiting saturated fat consumption has been a longstanding dietary recommendation to reduce risk of CVD. In particular, previous DGACs have recommended consuming no more than 10 percent of daily calories from saturated fat.

However, recent meta-analyses of prospective observational studies did not find a significant association between higher saturated fat intake and risk of CVD in large populations. These data have re-ignited the debate regarding the current recommendation to limit saturated fat intake. Therefore, the DGAC chose to conduct a focused review of published systematic reviews and meta-analyses on saturated fat intake and CVD. A central issue in the relationship between saturated fat and CVD is the specific macronutrients that are used to replace it because consuming unsaturated fats versus carbohydrates in place of saturated fat can have different effects on blood lipids and risk of CVD. Thus, the Committee's assessment of the available evidence puts greater emphasis on the replacement macronutrient for saturated fat.

In the United States, the top sources of foods contributing to saturated fat intake are mixed dishes, particularly burgers and sandwiches, and snacks and sweets (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). Although saturated fat intake has declined in the past decades, current intake is still high at a median of 11.1 percent of daily calories (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). Therefore, saturated fat continues to be an area of public health concern and the DGAC deemed it important to re-evaluate and update the knowledge base on saturated fat intake and CVD risk.

Question 5: What is the relationship between intake of saturated fat and risk of cardiovascular disease?

Source of evidence: Existing reports

Conclusions

Strong and consistent evidence from RCTs shows that replacing SFA with unsaturated fats, especially PUFA, significantly reduces total and LDL cholesterol. Replacing SFA with carbohydrates (sources not defined) also reduces total and LDL cholesterol, but significantly increases triglycerides and reduces HDL cholesterol.

Strong and consistent evidence from RCTs and statistical modeling in prospective cohort studies shows that replacing SFA with PUFA reduces the risk of CVD events and coronary mortality. For every 1 percent of energy intake from SFA replaced with PUFA, incidence of CHD is reduced by 2 to 3 percent. However, reducing total fat (replacing total fat with overall carbohydrates) does not lower CVD risk. Consistent evidence from prospective cohort studies shows that higher SFA intake as compared to total carbohydrates is not associated with CVD risk. **DGAC Grade: Strong**

Evidence is limited regarding whether replacing SFA with MUFA confers overall CVD (or CVD endpoint) benefits. One reason is that the main sources of MUFA in a typical American diet are animal fat, and because of the co-occurrence of SFA and MUFA in foods makes it difficult to tease out the independent association of MUFA with CVD. However, evidence from RCTs and prospective studies has demonstrated benefits of plant sources of monounsaturated fats, such as olive oil and nuts on CVD risk. **DGAC Grade: Limited**

Implications

Recommendations on saturated fat intake should specify replacement macronutrients and emphasize replacing saturated fat with unsaturated fats, especially polyunsaturated fats. The Committee recommends retaining the 10 percent upper limit for saturated fat intake. In practice, non-hydrogenated vegetable oils that are high in unsaturated fats and relatively low in SFA (e.g., soybean, corn, olive, and canola oils) instead of animal fats (e.g., butter, cream, beef tallow, and lard) or tropical oils (e.g., palm, palm kernel, and coconut oils) should be recommended as the primary source of dietary fat. Partially hydrogenated oils containing *trans* fat should be avoided.

In low-fat diets, fats are often replaced with refined carbohydrates and this is of particular concern because such diets are generally associated with dyslipidemia (hypertriglyceridemia and low HDL-C concentrations). Therefore, dietary advice should put the emphasis on optimizing types of dietary fat and not reducing total fat.

When individuals reduce consumption of refined carbohydrates and added sugars, they should not replace them with foods high in saturated fat. Instead, refined carbohydrates and added sugars should be replaced by healthy sources of carbohydrates (e.g., whole grains, legumes, vegetables, and fruits), and healthy sources of fats (e.g., non-hydrogenated vegetable oils that are high unsaturated fats, and nuts/seeds). The consumption of “low-fat” or “nonfat” products with high amounts of refined grains and added sugars should be discouraged.

Dietary recommendations on macronutrient composition for reducing CVD risk should be dietary pattern-based emphasizing foods that characterize healthy dietary patterns (see ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***). Individuals are encouraged to consume dietary patterns that emphasize vegetables, fruits, whole grains, legumes, and nuts; include low- and non-fat dairy products, poultry, seafood, non-tropical vegetable oils; limit sodium, saturated fat, refined grains, sugar-sweetened foods and beverages, and are lower in red and processed meats. Multiple dietary patterns can achieve these food and nutrient patterns and are beneficial for cardiovascular health, and they should be tailored to individuals’ biological needs and food preferences.

Review of the Evidence

The DGAC drew evidence from SRs or MA published between January 2009 and August 2014 in English in a peer-reviewed journal, which included RCTs and/or prospective cohort studies. Participants included healthy volunteers as well as individuals at elevated chronic disease risk. The main exposure was SFA, and the main outcomes included LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C), triglycerides (TG), blood pressure (BP), and incidence of CVD and CHD, CVD- and CHD-related death, myocardial infarction, or stroke. All reviews were high-quality, with ratings ranging from 8 to 11 on AMSTAR. The Committee drew evidence on blood lipids and blood pressure outcomes from the AHA/ACC Lifestyle Guideline and the associated NHLBI Lifestyle Report, which included primarily RCTs on intermediate CVD risk factors. The Committee drew evidence on CVD endpoints and effect size estimates from seven published MA that included one or more studies not covered in these reports.[19-25] Little evidence on the contribution of SFA to cardiovascular risk factors in the pediatric populations was available, and that which was published has not been systematically reviewed.

Effects of Replacing SFA on LDL-C, HDL-C, and TG

Macronutrients may affect plasma lipids and lipoproteins, which are strong predictors of CVD risk. The NHLBI Lifestyle Report summarized evidence from three feeding trials examining effects on LDL-C of dietary patterns with varying SFA levels: DASH (Dietary Approaches to Stop Hypertension), DASH-Sodium, and DELTA (Dietary Effects on Lipoproteins and Thrombogenic Activity). The results from these trials indicate that reducing total and saturated fat led to a significant reduction in LDL cholesterol in the context of the DASH dietary pattern and the National Cholesterol Education Program (NCEP) Step 1 diet. To estimate the effects of replacing SFA by specific macronutrients such as carbohydrates, MUFA, or PUFA, the NHLBI Lifestyle Report also included two MA from Mensink and Katan (n=1,672), covering the period from 1970 to 1998 (27 controlled trials in the first MA and 60 controlled trials in the second MA) and using the same inclusion/exclusion criteria to estimate changes in plasma lipids when substituting dietary SFA with carbohydrates or other fat types and holding dietary cholesterol constant.[26, 27] Mensink and Katan found that replacing 1 percent of SFA with an equal amount of carbohydrates, MUFA, or PUFA led to comparable LDL-C reductions: 1.2, 1.3, and 1.8 mg/dL, respectively. Replacing 1 percent of SFA with carbohydrates, MUFA, or PUFA also lowered HDL-C by 0.4, 1.2, and 0.2 mg/dL, respectively. Replacing 1 percent of carbohydrates by an equal amount of MUFA or PUFA raised LDL-C by 0.3 and 0.7 mg/dL, raised HDL-C by 0.3 and 0.2 mg/dL, and lowered TG by 1.7 and 2.3 mg/dL, respectively. The 2003 MA by Mensink and Katan[27] indicated that the ratio of total to HDL-C, a stronger predictor of CVD risk than total or LDL cholesterol alone, did not change when SFA was replaced by carbohydrates, but the ratio significantly decreased when SFA was replaced by unsaturated fats, especially PUFA.

In summary, strong and consistent evidence from RCTs shows that replacing SFA with unsaturated fats, especially PUFA, significantly reduces total and LDL cholesterol. Replacing SFA with carbohydrates also reduces total and LDL cholesterol, but significantly increases TG and reduces HDL cholesterol. However, the evidence of beneficial effects on one risk factor does not rule out neutral or opposite effects on unstudied risk factors. To better assess the overall effects of intervention to reduce or modify SFA intake, studies of clinical endpoints are summarized below.

The Relationship between Consumption of Total Fat and SFA and Risk of CVD

A MA by Skeaff et al. in 2009 included 28 U.S. and European cohorts (6,600 CHD deaths among 280,000 participants) and found no clear relationship between total or SFA intake and CHD events or deaths.[25] Similarly, Siri-Tarino et al., 2010 found that SFA intake was not associated with risk of CHD, stroke or cardiovascular disease.[24] The Siri-Tarino et al., 2010 meta-analysis included data from 347,747 participants (11,006 developed CVD) in 21 unique studies, with 16 studies providing risk estimates for CHD and 8 studies providing data for stroke as an endpoint. In the 2012 MA of trials to reduce or modify intake of SFA, Hooper et al. also found no significant associations of total fat reduction with cardiovascular events or mortality.

Consistent with these prior studies, Chowdhury et al.'s 2014 MA of total SFA also did not specify what macronutrient substituted SFA and again found no association of dietary SFA intake, nor of circulating SFA, with coronary disease.[19] Chowdhury et al. included data from 32 observational studies (530,525 participants) of fatty acids from dietary intake, 17 observational studies (25,721 participants) of fatty acid biomarkers, and 27 RCTs (103,052 participants) of fatty acid supplementation.

The results described above do not explicitly specify the comparison or replacement nutrient, but typically it consists largely of carbohydrates (sources not defined). These results suggest that replacing SFA with carbohydrates is not associated with CVD risk. Taken together, these results suggest that simply reducing SFA or total fat in the diet by replacing it with any type of carbohydrates is not effective in reducing risk of CVD.

Effects of Replacing SFA with Polyunsaturated Fat or Carbohydrates on CVD Events

Hooper et al.'s 2012 Cochrane MA of trials of SFA reduction/modification found that reducing SFA by reducing and/or modifying dietary fat reduced the risk of cardiovascular events by 14 percent (pooled RR = 0.86; 95% CI = 0.77 to 0.96, with 24 comparisons and 65,508 participants of whom 7 percent had a cardiovascular event, I= 50%).[21] Subgroup analyses revealed this protective effect was driven by dietary fat *modification* rather than reduction and was only apparent in longer trials (2 years or more). Despite the reduction in total cardiovascular events, there was no clear evidence of reductions in any individual outcome (total or non-fatal myocardial infarction, stroke, cancer deaths or diagnoses, diabetes diagnoses), nor was there any evidence that trials of reduced or modified SFA reduced cardiovascular mortality. These results suggest that modifying dietary fat by replacing some saturated (animal) fats with plant oils and unsaturated spreads may reduce risk of heart and vascular disease.

Emphasizing the benefits of replacement of saturated with polyunsaturated fats, Mozaffarian et al., 2010 found in a MA of 8 trials (13,614 participants with 1,042 CHD events) that modifying fat reduced the risk of myocardial infarction or coronary heart disease death (combined) by 19 percent (RR = 0.81; 95% CI = 0.70 to 0.95; p = 0.008), corresponding to 10 percent reduced CHD risk (RR = 0.90; 95% CI = 0.83 to 0.97) for each 5 percent energy of increased PUFA.[23] This magnitude of effect is similar to that observed in the Cochrane MA. In secondary analyses restricted to CHD mortality events, the pooled RR was 0.80 (95% CI = 0.65 to 0.98). In subgroup analyses, the RR was greater in magnitude in the four trials in primary prevention populations but non-significant (24 percent reduction in CHD events) compared to a significant reduction of 16 percent in the four trials of secondary prevention populations. Mozaffarian et al. argue that the slightly greater risk reduction in studies of CHD events, compared with predicted effects based on lipid changes alone, is consistent with potential additional benefits of PUFA on other non-lipid pathways of risk, such as insulin resistance. Many of the included trials used vegetable oils containing small amounts of plant-derived n-3 PUFA in addition to omega-6 PUFA.

Consistent with the benefits of replacing SFA with PUFA for prevention of CHD shown in other studies, Farvid et al., 2014 conducted an SR and MA of prospective cohort studies of dietary linoleic acid (LA), which included 13 studies with 310,602 individuals and 12,479 total CHD events (5,882 CHD deaths).[20] Farvid et al. found dietary LA intake is inversely associated with CHD risk in a dose-response manner: when comparing the highest to the lowest category of intake, LA was associated with a 15 percent lower risk of CHD events (pooled RR = 0.85; 95% CI = 0.78 to 0.92; I^2=35.5%) and a 21% lower risk of CHD deaths (pooled RR = 0.79; 95% CI = 0.71 to 0.89; I^2=0.0%). A 5 percent of energy increment in LA intake replacing energy from SFA intake was associated with a 9 percent lower risk of CHD events (RR = 0.91; 95% CI = 0.86 to 0.96) and a 13 percent lower risk of CHD deaths (RR = 0.87; 95% CI = 0.82 to 0.94). In the meta-analysis conducted by Chowdhury et al., there was no significant association between LA intake and CHD risk, but the analysis was based on a limited number of prospective cohort studies.

In Jakobsen et al.'s 2009 pooled analysis of 11 cohorts (344,696 persons with 5,249 coronary events and 2,155 coronary deaths), a 5 percent lower energy intake from SFAs and a concomitant higher energy intake from PUFAs reduced risk of coronary events by 13 percent (hazard ratio [HR] = 0.87; 95% CI = 0.77 to 0.97) and coronary deaths by 16 percent (hazard ratio = 0.74; 95% CI = 0.61 to 0.89).[22] By contrast, a 5 percent lower energy intake from SFAs and a concomitant higher energy intake from carbohydrates, there was a modest significant direct association between carbohydrates and coronary events (hazard ratio = 1.07; 95% CI = 1.01 to 1.14) and no association with coronary deaths (hazard ratio = 0.96; 95% CI = 0.82 to 1.13). Notably, the estimated HRs for carbohydrate intake in this study could reflect high glycemic carbohydrate intake rather than total carbohydrate, as fiber was controlled for in the analyses. MUFA intake was not associated with CHD incidence or death.

Taken together, strong and consistent evidence from RCTs and statistical modeling in prospective cohort studies shows that replacing SFA with PUFA reduces the risk of CVD events and coronary mortality. For every 1 percent of energy intake from SFA replaced with PUFA, incidence of CHD is reduced by 2 to 3 percent. The evidence is not as clear for replacement by MUFA or replacement with carbohydrate, and likely depends on the type and source.

Methodological Issues

When individuals in natural settings reduce calories from SFA, they typically replaced them with other macronutrients, and the type and source of the macronutrients substituting SFA determine effects on CVD. For this reason, studies specifying the macronutrient type replacing SFA are more informative than those examining only total SFA intake, and the strongest and most consistent evidence for CVD reduction is with replacement of SFA with PUFA in both RCTs and observational studies.

The differing effects of the type and source of macronutrient substituted may be one reason for the limited evidence regarding whether replacing SFA with MUFA confers CVD benefits and the lack of benefit from carbohydrate substitution. The main sources of MUFA in a typical American diet are animal fats, which could confound potential benefits of SFA-replacement with plant-source MUFA, such as nuts and olive oil, which have demonstrated benefits on CVD risk. To date, evidence testing replacement of SFA by MUFA from different sources is insufficient to reach a firm conclusion. Similarly, most analyses did not distinguish between substitution of saturated fat by different types of carbohydrates (e.g., refined carbohydrate vs. whole grains).

Of the RCTs included in this evidence summary, the intervention methods used varied from long-term dietary counseling with good generalizability but variable compliance, to providing a whole diet for weeks (e.g., controlled feeding studies) with maximal compliance but limited generalizability. Though the content of the recommended or provided diet is known with greater precision in the RCTs than in observational studies, adherence to the diet is likely variable and could result in lack of compliance and high rates of dropout in long-term trials. Additionally, bias may arise from the lack of blinding in non-supplement dietary intervention trials.

In prospective observational studies, misclassification of dietary fatty acid intake could bias associations towards the null. In addition, residual confounding by other dietary and lifestyle factors cannot be ruled out through statistical adjustment. Despite these methodological issues, there is high consistency of the evidence from prospective cohort studies and RCTs in supporting the benefits of replacing saturated fat with unsaturated fats especially PUFA in reducing CVD risk.

For additional details on this body of evidence, visit: References 1, 2, 19-25 and ***Appendix E-2.43***

ADDED SUGARS AND LOW-CALORIE SWEETENERS

INTRODUCTION

Added sugars are sugars that are either added during the processing of foods, or are packaged as such, and include sugars (free, mono- and disaccharides), syrups, naturally occurring sugars that are isolated from a whole food and concentrated so that sugar is the primary component (e.g., fruit juice concentrates), and other caloric sweeteners.[28] Added sugars have been discussed in previous iterations of the *Dietary Guidelines*, including a key recommendation in the *2010 Dietary Guidelines* to "Reduce the intake of calories from solid fats and added sugars." The *2010 Dietary Guidelines* also included guidance stating that, for most people, no more than about 5 to 15 percent of calories from solid fats and added sugars (combined) can be reasonably accommodated in a healthy eating pattern. However, as discussed in ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***, the current intake of added sugars still remains high at 268 calories, or 13.4 percent of total calories per day among the total population ages 1 year and older.

Similar to the healthy eating patterns modeled for the 2010 DGAC, in the three healthy eating patterns modeled for the 2015 DGAC (Healthy U.S.-style Pattern, Healthy Mediterranean-style Pattern, and Healthy Vegetarian Pattern), a limited number of calories are available to be consumed as added sugars (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends***). As shown in Table D.6.1, the full range of these three patterns at all calorie levels allow for 3 to 9 percent of calories from added sugars, after meeting food group and nutrient recommendations. For the patterns appropriate for most people (1600 to 2400 calories), the range is 4 to 6 percent of calories from added sugars (or 4.5 to 9.4 teaspoons). The total empty calorie allowance in these patterns is 8 to 19 percent of calories, and based on current consumption patterns, 45 percent of empty calories are allocated to limits for added sugars, with the remainder (55 percent) allocated to solid fats.

Table D6.1. Added sugars available in the USDA Food Patterns (Healthy U.S.-Style, Healthy Mediterranean-Style, and Healthy Vegetarian Patterns) in calories, teaspoons, and percent of total calories per day*

CALORIE LEVEL	1000	1200	1400	1600	1800	2000	2200	2400	2600	2800	3000	3200
Empty calorie limits available for **added sugars** (assuming 45% empty calories from added sugars and 55% from solid fat)												
Healthy U.S.-style	68	50	50	54	77	122	126	158	171	180	212	275
Healthy Med-style	63	50	50	81	72	117	126	135	149	158	194	257
Healthy Vegetarian	77	77	81	81	81	131	131	158	158	158	185	234
Average	69	59	60	72	77	123	128	150	159	165	197	255
Average (tsp)	4.3	3.7	3.8	4.5	4.8	7.7	8.0	9.4	9.9	10.3	12.3	15.9
Healthy U.S.-style	7%	4%	4%	3%	4%	6%	6%	7%	7%	6%	7%	9%
Healthy Med-style	6%	4%	4%	5%	4%	6%	6%	6%	6%	6%	6%	8%
Healthy Vegetarian	8%	6%	6%	5%	5%	7%	6%	7%	6%	6%	6%	7%
Average	7%	5%	4%	5%	4%	6%	6%	6%	6%	6%	7%	8%

* See ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** and Appendix E-3.7 for a full discussion of the food pattern modeling.

Although food pattern modeling evaluates the amount of added sugars that can be consumed while meeting food group and nutrient needs, the DGAC also reviewed scientific literature examining the relationship between the intake of added sugars and health to inform recommendations. The Committee focused on the health outcomes most commonly researched related to added sugars, specifically, body weight and risk of type 2 diabetes, CVD, and dental caries.

As noted above, the Committee acknowledged that a potential unintended consequence of a recommendation on added sugars might be that consumers and manufacturers replace added sugars with low-calorie sweeteners. As a result, the Committee also examined evidence on low-calorie sweeteners to inform statements on this topic. The Committee approached this topic broadly, including sweeteners labeled as low-calorie sweeteners, non-caloric sweeteners, non-nutritive sweeteners, artificial sweeteners, and diet beverages. This work is complemented by a food safety evidence review on aspartame (see ***Part D. Chapter 5: Food Sustainability and Safety***). As the evidence on added sugars was considered collectively, the added sugars conclusions are presented together below, and a similar approach was taken for low-calorie sweeteners.

Question 6: What is the relationship between the intake of added sugars and cardiovascular disease, body weight/obesity, type 2 diabetes, and dental caries?

Source of evidence: CVD: NEL systematic review; Body weight/obesity, type 2 diabetes, and dental caries: Existing reports

Conclusions

Strong and consistent evidence shows that intake of added sugars from food and/or sugar-sweetened beverages are associated with excess body weight in children and adults. The reduction of added sugars and sugar-sweetened beverages in the diet reduces body mass index (BMI) in both children and adults. Comparison groups with the highest versus the lowest intakes of added sugars in cohort studies were compatible with a recommendation to keep added sugars intake below 10 percent of total energy intake. **DGAC Grade: Strong**

Strong evidence shows that higher consumption of added sugars, especially sugar-sweetened beverages, increases the risk of type 2 diabetes among adults and this relationship is not fully explained by body weight. **DGAC Grade: Strong**

Moderate evidence from prospective cohort studies indicates that higher intake of added sugars, especially in the form of sugar-sweetened beverages, is consistently associated with increased risk of hypertension, stroke, and CHD in adults. Observational and intervention studies indicate a consistent relationship between higher added sugars intake and higher blood pressure and serum triglycerides. **DGAC Grade: Moderate**

The DGAC concurs with the World Health Organization’s commissioned systematic review that moderate consistent evidence supports a relationship between the amount of free sugars intake and the development of dental caries among children and adults. Moderate evidence also indicates that caries are lower when free sugars intake is less than 10 percent of energy intake. **DGAC Grade: Moderate**

Review of the Evidence

Added Sugars and Body Weight/Obesity

These findings come from three recent reports, all using SRs and MA that examined the relationship between the intake of added sugars and measures of body weight.[6, 29, 30] Te Morenga et al.[6] considered “free sugars,”[*] while Malik[29] and Kaiser et al.[30] focused on sugar-sweetened

[*] Free sugar is defined by WHO as "all monosaccharides and disaccharides added to foods by the manufacturer, cook, or consumer, plus sugars naturally present in honey, syrups, and fruit juices." It is used to distinguish between the sugars that are naturally present in fully unrefined carbohydrates such as brown rice, whole wheat pasta, and fruit and those sugars (or carbohydrates) that have been, to some extent, refined

beverages. All reviews reported on body weight. The Te Morenga report also reported on body fatness. In the Te Morenga et al. study, 30 trials and 38 cohort studies were included in the analyses. In the Malik et al. study, 10 trials and 22 cohort studies were included in the analyses. Kaiser et al. provided an updated meta-analysis to a previous publication (Mattes[31]) and included a total of 18 trials. In total, 92 articles were considered in these reviews, of which 21 were included in two or more reviews. Children and adults were included in the analyses as were females and males. Diverse demographics (race/ethnicity and geographic location) also were represented by the participants in the respective research studies. All three reviews were high-quality, with ratings of 11 out of 11 using the AMSTAR tool, and they specifically addressed the Committee's question of interest.

The reviews by Malik et al. and Te Morenga et al. were very consistent. The findings from both reports provide strong evidence that among free-living people consuming ad libitum diets, the intake of added sugars or sugar-sweetened beverages is associated with unfavorable weight status in children and adults. Increased added sugars intake is associated with weight gain; decreased added sugars intake is associated with decreased body weight. Although a dose response cannot be determined at this time, the data analyzed by Te Morenga et al. support limiting added sugars to no more than 10 percent of daily total energy intake based on lowest versus highest intakes from prospective cohort studies. Te Morenga et al. state that, "despite significant heterogeneity in one meta-analysis and potential bias in some trials, sensitivity analyses showed that the trends were consistent and associations remained after these studies were excluded." Despite these limitations the DGAC gave this evidence a grade of **Strong**, as the limitations are those inherent to the primary research on which they are based, notably inadequacy of dietary intake data and variations in the nature and quality of the dietary interventions.

The Kaiser et al. review concluded that the currently available randomized evidence for the effects of reducing sugar-sweetened beverage intake on obesity is equivocal. However, the DGAC noted methodological issues with this review, particularly the inclusion of both efficacy studies (in more controlled settings) and effectiveness studies (in real world). The outcomes from the effectiveness trials vary substantially, depending how effective the interventions are. As a result, the Committee viewed the reviews by Te Morenga et al. and Malik et al. to be stronger than the Kaiser et al. review.

Added Sugars and Type 2 Diabetes

Evidence for this question and conclusion came from five SRs and MA published between January 2010 and August 2014.[33-37] Four of the reviews focused on sugar-sweetened

(normally by humans but sometimes by animals, such as the free sugars present in honey). They are referred to as "sugars" since they cover multiple chemical forms, including sucrose, glucose, fructose, dextrose, and others.[32]

beverages[33-35, 37] and one review examined sugar intake.[36] Combined, a total of 17 articles were considered in these reviews, of which nine were included in two or more reviews. Increased consumption of sugar-sweetened beverages was consistently associated with increased risk of type 2 diabetes. Pooled estimated relative risks ranged from 1.20 to 1.28, and included 1.20 (95% CI = 1.12 to 1.29)/330 ml/day of sugar-sweetened soft drinks;[33]1.26 (95% CI = 1.12 to 1.41) for sugar-sweetened beverages,[35] and 1.28 (95% CI = 1.04 to 1.59) for sugar-sweetened fruit juices.[37] Comparably, a hazard ratio of 1.29 (1.02, 1.63) was identified for sugar-sweetened beverages.[34] These consistently positive associations between sugar-sweetened beverages and type 2 diabetes were attenuated, but still existed, after adjustment for BMI, suggesting that body weight only partly explains the deleterious effects of sugar-sweetened beverages on type 2 diabetes. Although the studies were highly heterogeneous, findings from the MA by Malik et al. tentatively showed that consumption of more than one 12-ounce serving per day of sugar-sweetened beverage increased the risk of developing type 2 diabetes by 26 percent, compared to consuming less than one serving per month. Insufficient high-quality data are available to determine a dose-response line or curve between sugar-sweetened beverage consumption and type 2 diabetes risk.

The issue of generalizability, whether the participants included in this body of evidence are representative of the general U.S. population, was not specifically addressed in the literature reviewed, but the large sample sizes of the pooled data (several hundred thousand subjects from different populations) are noteworthy.

Added Sugars and Cardiovascular Disease

This NEL systematic review included 23 articles published since 2000 that examined the relationship between added sugars and risk of CVD or CVD risk factors such as blood lipids and blood pressure.[38-60] This literature included 11 intervention studies and 12 prospective cohort studies.

The majority of intervention and observational studies included in this SR provide some evidence among adults in support of an association between higher intake of added sugars, especially in the form of sugar-sweetened beverages, and higher risk of CVD or increased CVD risk factors. More consistent associations were seen between added sugars and elevated serum triglycerides, blood pressure, and increased risk of hypertension, stroke, or CHD. Evidence for associations between added sugars and dyslipidemia (i.e., low HDL, high LDL, and high total cholesterol) was not as consistent, especially among intervention studies.

The body of evidence examined in this SR had a number of limitations. For example, the intervention studies had extensive heterogeneity in terms of the types and forms of sugars used (i.e., fructose, glucose, sucrose, sugar-sweetened beverages, sweetened milk) and the type of control and/or isocaloric condition used. In addition, most intervention studies had a short duration of the intervention and a small sample size. Most of the observational studies assessed dietary intake only at baseline, and did not take assessments during follow-up. Residual confounding by other dietary and lifestyle factors in observational analyses could not be completely ruled out.

Added Sugars and Dental Caries

These findings were extracted from a World Health Organization (WHO)-commissioned SR by Moynihan et al. published in 2014 examining the association between the amount of sugars intake and dental caries.[7] The search for SRs/MA published since completion of the WHO review did not yield any additional reviews that met the DGAC's inclusion criteria.

Moynihan et al. examined total sugars, free sugars, added sugars, sucrose, and non-milk extrinsic (NME) sugars. In the review, eligible studies reported the absolute amount of sugars. Dental caries outcomes included caries prevalence, incidence and/or severity.

Several databases were searched from 1950 through 2011. From 5,990 papers identified, 55 studies (from 65 papers) were eligible, including 3 interventions, 8 cohort studies, 20 population studies, and 24 cross-sectional studies. No RCTs were included. Data variability limited the ability to conduct meta-analysis. Of the 55 studies included in the review, the majority were in children and only four studies were conducted in adults. The terminology used for reporting sugars varied, but most were described as pertaining to free sugars or added sugars.

The findings indicated consistent evidence of moderate quality supporting a relationship between the amount of sugars consumed and dental caries development across age groups. Of the studies, 42 out of 50 studies in children and five out of five in adults reported at least one result for an association between sugars intake with increased caries. Moderate evidence also showed that caries incidence is lower when free sugars intake is less than 10 percent of energy intake. When a less than 5 percent energy intake cutoff was used, a significant relationship between sugars and caries was observed, but the evidence was judged to be of very low quality. Although meta-analysis was limited, analysis of existing data indicated a large effect size (e.g., Standardized Mean Difference for Decayed/Missing/Filled Teeth [DMFT] = 0.82 [CI = 0.67-0.97]) for the relationship of sugars intake and risk of dental caries. A strength of the in-depth SR was the consistency of data, despite methodological weaknesses in many studies, which included unclear definitions of endpoints, questions about outcomes ascertainment, and lack of clarity about the generalizability of individual study results given the study populations used.

For additional details on this body of evidence, visit: References 6, 7, 29, 30, 33-37, and 38-60 and ***Appendices E-2.44*** *(body weight)*, ***E-2.45*** *(type 2 diabetes)*, ***E-2.46*** *(dental caries)*, and http://NEL.gov/topic.cfm?cat=3376 *(CVD)*

Question 7: What is the relationship between the intake of low-calorie sweeteners and body weight/obesity and type 2 diabetes?

Source of evidence: Existing reports

Conclusions

Moderate and generally consistent evidence from short-term RCTs conducted in adults and children supports that replacing sugar-containing sweeteners with low-calorie sweeteners reduces calorie intake, body weight, and adiposity. **DGAC Grade: Moderate**

Long-term observational studies conducted in children and adults provide inconsistent evidence of an association between low-calorie sweeteners and body weight as compared to sugar-containing sweeteners. **DGAC Grade: Limited**

Long-term observational studies conducted in adults provide inconsistent evidence of an association between low-calorie sweeteners and risk of type 2 diabetes. **DGAC Grade: Limited**

Review of the Evidence

Low-Calorie Sweeteners and Body Weight/Obesity

The evidence to support these conclusions comes from three SRs/MA published between January 2010 and August 2014.[61-63] In total, 39 articles were considered in these reviews, of which six were included in two or more reviews. Experimentally, the protocols described in the 39 articles included RCTs and prospective cohort studies. Although results from both experimental designs were carefully assessed, the DCAC deemed evidence from RCTs to be scientifically stronger and used it as the foundation for conclusions pertaining to body weight.

Among prospective cohort studies, low-calorie sweetener intake was not associated with body weight or fat mass, but was significantly associated with slightly higher BMI (0.03; 95% CI = 0.01 to 0.06).[62] These findings should be viewed with caution, however, because of the high risk of reverse causality and the possibility that people with higher body weights would consume more low-calorie sweetener-containing foods and beverages as a weight-control strategy.

Evidence from short-term RCTs consistently indicated that low-calorie sweeteners (vs. sugar-containing foods and beverages) modestly reduce body weight in adults. When evidence from adults and children were combined, low-calorie sweeteners modestly reduced BMI, fat mass, and waist circumference. The primary research articles used by Miller and Perez for the MA contained findings from both adults (n=5 cohorts) and children (n=4 cohorts).[62] The results of interventions lasting 3 to 78 weeks indicated that low-calorie sweeteners reduced body weight in adults (-0.72 kg; 95% CI = -1.15 to -0.30) and children (-1.06 kg; 95% CI = -1.17 to -0.56). Age-specific results were not provided for BMI, fat mass, or waist circumference, but data from both age groups were pooled to show the impact of low-calorie sweeteners vs. sugar-containing foods/beverages on these outcomes.

In contrast, Brown et al. summarized that very limited evidence from three short-term (12 to 25 week) RCTs, which suggested that consumption of low-calorie sweeteners does not influence body weight or BMI in predominantly pre-teenage and teenage youth (ages 10 to 21 years), compared to sugar-sweetened beverage or placebo.[61] The authors cautioned that insufficient data exist to assess causality of low-calorie sweeteners on body weight. The evidence reported in this 2010 publication was obtained from very heterogeneous experimental designs and interventions. One study tested the effects of encapsulated aspartame vs. placebo during weight loss; another allowed subjects to exchange sugar-sweetened beverages with either low-calorie sweetener beverages or water (precluding assessment of low-calorie sweetener beverages specifically); and a third was described as a "pilot study."

Collectively, evidence is mixed on the impact of low-calorie sweeteners vs. sugar-containing foods/beverages on body weight in children. However, the DGAC deemed evidence presented by Miller and Perez[62] to be stronger than from Brown et al.[61] because it culminated from a larger, more recent research base and include both systematic review and meta-analysis assessment and evaluation techniques.

Low-Calorie Sweeteners and Type 2 Diabetes

Evidence to address the impact of low-calorie sweeteners (specifically artificially sweetened soft drinks, ASSD) on risk of type 2 diabetes comes from two SRs/MA published between January 2010 and August 2014.[33, 34] The data from one of the reviews also is represented in the second review.

Greenwood et al. reported that higher consumption of ASSD predicts increased risk of type 2 diabetes.[33] The summary RR for ASSD on type 2 diabetes risk was 1.13 (95% CI = 1.02 to 1.25, $p<0.02$) per 330 ml/day, based on four analyses from three prospective observational studies. Although the finding indicates a positive association between ASSD and type 2 diabetes risk, the trend was not consistent and may indicate an alternative explanation, such as confounding by lifestyle factors or reverse causality (e.g., individuals with higher BMI at baseline may use ASSD as a means to control weight).

Romaguera et al. also reported that higher consumption of ASSD was associated with increased risk of type 2 diabetes.[34] In adjusted models, one 336 g (12 oz) daily increment in ASSD consumption was associated with a hazard ratio for type 2 diabetes of 1.52 (95% CI = 1.26 to 1.83). High consumers of ASSD showed almost twice the hazard ratio of developing type 2 diabetes compared with low consumers (adjusted HR = 1.93; 95% CI = 1.47 to 2.54; p for trend

<0.0001). However, the association was attenuated and became statistically not significant when BMI was included in the model (HR = 1.13, 95% CI = 0.85 to 1.52; p for trend = 0.24). The authors offered these interpretations of the findings: "In light of these findings, we have two possible explanations of the association between artificially sweetened soft drinks and diabetes: (1) the observed association is driven by reverse causality and residual confounding, given that the underlying health of people consuming artificially sweetened soft drinks may be compromised and their risk of type 2 diabetes increased; or (2) the association between artificially sweetened soft drinks and type 2 diabetes is mediated through increased BMI." The authors argued that explanation 1 is more likely correct based on reverse causality, but new research would be needed to clarify the issue.

Collectively, both studies report a positive association between ASSD and type 2 diabetes risk that was confounded by baseline BMI. The experimental designs of the studies included in these reviews analyzed associations, but precluded the assessment of cause and effect relationships, and future experimental studies should examine the relationship between ASSD and biomarkers of insulin resistance and other diabetes biomarkers.

For additional details on this body of evidence, visit: References 33, 34, and 61-63 and ***Appendices E-2.47*** (body weight) and ***E-2.48*** (type 2 diabetes)

Implications

Obesity, type 2 diabetes, CVD, and dental caries are major public health concerns. Added sugars intake negatively impacts all of these conditions, and strong evidence supports reducing added sugars intake to reduce health risks. Added sugars are frequently used in food/beverage processing and provide calories but no other nutrients. Since 39 percent of added sugars are from sugar-sweetened beverages, efforts are needed to reduce these beverages (see Figure D1.36. Food Sources of Added Sugars). Currently, the mean intake of added sugars in the U.S. population is 13%, and from 15% to 17% in children 9 and older, adolescents, and young adults.

The DGAC recommends limiting added sugars to a maximum of 10% of total daily caloric intake. This recommendation is supported by: 1) the food pattern modeling analysis conducted by the 2015 DGAC and 2) the scientific evidence review on added sugars and chronic disease risk conducted by the Committee. The food pattern analysis, based on the Healthy U.S.-Style Pattern, the Healthy Vegetarian Pattern, and the Healthy Mediterranean-Style Pattern (see ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** and ***Appendix E-3.7***), demonstrates that when added sugars in foods and beverages exceeds 3% to 9% of total calories, depending on calorie level, a healthful food pattern may be difficult to achieve and nutrient density may be adversely affected (Table D6.1). The scientific evidence on added sugars and chronic disease risk also supports this limit.

The recommendation to limit added sugars, especially sugar-sweetened beverages, is consistent with recommendations from national and international organizations including the American Academy of Pediatrics, World Health Organization, American Heart Association, Centers for Disease Control and Prevention, and the American Diabetes Association (Table D6.2).

When low-calorie sweeteners are used to replace sugar, the resulting reduction in calories can help to achieve short-term weight loss. However, there is insufficient evidence (due to a paucity of data) to recommend the use of low-calorie sweeteners as a strategy for long-term weight loss and weight maintenance. Since the long-term effects of low-calorie sweeteners are still uncertain, those sweeteners should not be recommended for use as a primary replacement/substitute for added sugars in foods and beverages.

Policies and programs at local, state, and national levels in both the private sector and public sector are necessary to support efforts to lower added sugars in beverages and foods and to limit availability of sugar-sweetened beverages and snacks. Suggested specific approaches for reducing added sugars intake include:

- Water is the preferred beverage choice. Strategies are needed to encourage the US population, especially children and adolescents, to drink water when they are thirsty. Water provides a healthy, low-cost, zero-calorie beverage option. Free, readily accessible, safe water should be available in public settings, as well as child care facilities, schools, worksites and other community places and promoted in all settings where beverages are offered.
- The Nutrition Facts Panel (NFP) should include added sugars (in grams and teaspoons) and include a percent daily value, to assist consumers in making informed dietary decisions by identifying the amount of added sugars in foods and beverages.
- Consumers would benefit from a standardized, easily understood front-of-package (FOP) label on all food and beverage products to give clear guidance about a food's healthfulness. An example is the FOP label recommended by the IOM,[18] which included calories, and 0 to 3 "nutritional" points for added sugars, saturated fat, and sodium. This would be integrated with the NFP, allowing consumers to quickly and easily identify nutrients of concern for over-consumption, in order to make healthier choices.
- Economic and pricing approaches, using incentives and disincentives should be explored to promote the purchase of healthier foods and beverages. For example, higher sugar-sweetened beverage taxes may encourage consumers to reduce sugar-sweetened beverage consumption. Using the revenues from the higher sugar-sweetened beverage taxes for nutrition health promotion efforts or to subsidize fruits and vegetables could have public health benefits.

- Efforts to reduce added sugars in foods and sugar-sweetened beverages in school meals and through the new smart snacks in schools should continue and also be expanded to other settings, including early child care (through the Child and Adult Care Food Program-CACFP), parks, recreation centers, sports leagues, after school programs, work sites and other community settings.
- Policies that limit exposure and marketing of foods and beverages high in added sugars to young children, youth and adolescents are needed as dietary preferences are established early in life.
- Young adults (ages 20-29 years) are among the greatest consumers of sugar-sweetened beverages and are directly targeted in sugar-sweetened beverage marketing campaigns. Health promotion efforts and policies are needed to reduce sugar-sweetened beverages in settings, such as postsecondary institutions and worksites.
- Policy changes within the federal Supplemental Nutrition Assistance Program (SNAP), similar to policies in place for the WIC program, should be considered to encourage purchase of healthier options, including foods and beverages low in added sugars. Pilot studies using incentives and restrictions should be tested and evaluated.
- Public education campaigns are needed to increase the public's awareness of the health effects of added sugars and help consumers reduce added sugars intake and reduce intake of sugar-sweetened beverages through policy, food environment and education initiatives.

Table D6.2. Recommendations or statements related to added sugars or sugar-sweetened beverages from international and national organizations

Organization	Recommendation/Statement Related to Added Sugars and/or Sugar-Sweetened Beverages
World Health Organization (WHO)[64]	• WHO recommends reduced intake of free sugars throughout the life-course (*strong recommendation*). • In both adults and children, WHO recommends that intake of free sugars not to exceed 10% of total energy (*strong recommendation*). • WHO suggests further reduction to below 5% of total energy (*conditional recommendation*).
American Heart Association (AHA)[65]	The AHA recommends reductions in added sugars with an upper limit of half of the discretionary calorie allowance that can be accommodated within the appropriate energy intake level needed for a person to achieve or maintain a healthy weight based on the USDA food intake patterns. Most American women should eat or drink no more than 100 calories per day from added sugars (about 6 teaspoons), and most American men should eat or drink no more than 150 calories per day from added sugars (about 9 teaspoons).
HealthyPeople 2020[66]	Objective NWS-17.2: Reduce consumption of calories from added sugars (Target: 10.8%)
American Academy of Pediatrics (AAP)[67-69]	Limit consumption of sugar-sweetened beverages (consistent evidence) Pediatricians should work to eliminate sweetened drinks in schools *Note: Due to limited studies in children, the American Academy of Pediatrics (AAP) has no official recommendations regarding the use of non-caloric sweeteners.*
American Diabetes Association (ADA)[70, 71]	<u>Prevention</u> Research has shown that drinking sugary drinks is linked to type 2 diabetes, and the American Diabetes Association recommends that people limit their intake of sugar-sweetened beverages to help prevent diabetes. <u>Diabetes Management</u> People with diabetes should limit or avoid intake of sugar-sweetened beverages (from any caloric sweetener including high fructose corn syrup and sucrose) to reduce risk for weight gain and worsening of cardiometabolic risk profile. (Evidence rating B)
NHLBI Expert Panel Guidelines for Cardiovascular Health and Risk Reduction in Childhood[72]	Reduced intake of sugar-sweetened beverages is associated with decreased obesity measures (Grade B).

CHAPTER SUMMARY

The DGAC encourages the consumption of healthy dietary patterns that are low in saturated fat, added sugars, and sodium. The conclusions in this chapter complement the findings from ***Part D. Chapter 1: Food and Nutrient Intakes, and Health: Current Status and Trends*** and ***Part D. Chapter 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes***. The goals for the general population are: less than 2,300 mg dietary sodium per day (or age-appropriate Dietary Reference Intake amount), less than 10 percent of total calories from saturated fat per day, and a maximum of 10 percent of total calories from added sugars per day.

Sodium, saturated fat, and added sugars are not intended to be reduced in isolation, but as a part of a healthy dietary pattern. Rather than focusing purely on reduction, emphasis should be placed on replacement and shifts in food intake and eating patterns. Sources of saturated fat should be replaced with unsaturated fat, particularly polyunsaturated fatty acids. Similarly, added sugars should be reduced in the diet and not replaced with low-calorie sweeteners, but rather with healthy options, such as water in place of sugar-sweetened beverages. For sodium, emphasis should be placed on expanding industry efforts to reduce the sodium content of foods and helping consumers understand how to flavor unsalted foods with spices and herbs.

Achieving reductions in sodium, saturated fat, and added sugars, can all be accomplished and are more attainable by eating a healthy dietary pattern. For all three of these components of the diet, policies and programs at local, state, and national levels in both the private and public sector are necessary to support reduction efforts. Similarly, the Committee supports efforts in labeling and other campaigns to increase consumer awareness and understanding of sodium, saturated fats, and added sugars in foods and beverages. The Committee encourages the food industry to continue reformulating and making changes to certain foods to improve their nutrition profile. Examples of such actions include lowering sodium and added sugars content, achieving better saturated fat to polyunsaturated fat ratio, and reducing portion sizes in retail settings (restaurants, food outlets, and public venues, such as professional sports stadiums and arenas). The Committee also encourages the food industry to market these improved products to consumers.

NEEDS FOR FUTURE RESEARCH

1. Design and conduct studies with sufficient power to define the impact of improving dietary quality, including the lowering of dietary sodium intake, on hypertension and relevant disease outcomes, including cardiovascular disease, stroke, peripheral vascular disease, kidney disease, and others. The interactions with patterns of therapeutic medication use (e.g., diuretics, antihypertensives, and lipid-lowering) should be considered.

Rationale: The current literature is incomplete, limited in power and durations, and often compromised by methodological challenges that must be addressed in well-designed studies with relevant clinical outcomes.

2. Assess the accuracy of 24-hour urine collections for sodium assessment in populations with different health conditions (e.g., diabetes, chronic kidney disease, heart failure, cardiovascular disease) and interactions with different patterns of medication use (e.g., diuretics, antihypertensives).

   Rationale: If there is systematic error in sodium assessment because individuals with various co-morbidities who are taking medications systematically do not provide accurate urine collections, paradoxical findings between sodium and health outcomes may be observed.

3. Examine the effect of behavioral interventions, with novel approaches (e.g., flavorful recipes, cooking techniques) on adherence to dietary sodium recommendations.

   Rationale: For decades, the population has exceeded dietary sodium intake recommendations. A public health approach that results in reformulation of commercially processed foods to lower sodium content should be the primary strategy for decreasing sodium intake in the U.S. population. However, individual support for public health policies will be needed to further document demand for changes in the sodium food environment. To this end, interventions that modify individual knowledge, attitudes, and behaviors around sodium intake should be evaluated.

4. Examine the effect of low sodium intake on taste preferences for sodium and healthy dietary patterns.

   Rationale: It has been argued that populations desire higher levels of sodium intake and will inevitably revert to higher levels of sodium intakes after acute reductions in sodium intake. It has also been argued that after six weeks of reduced sodium intake, taste preferences are modified such that higher sodium is no longer desirable. Studies are needed to elucidate the effects of lowering sodium intake on diet preferences.

5. Document the relationship between portion size and sodium intake.

   Rationale: These data are needed to inform whether dietary recommendations for sodium should be adjusted for caloric intake. It is known that the absolute amount of sodium intake is highly correlated with caloric intake. As a result, the absolute recommended amount of sodium is harder to achieve for a larger, high energy consuming person than for a smaller, low energy consuming person. The science to inform whether sodium density confers different risk than absolute intake of sodium is limited because of methodologic limitations in surveys where both calories and sodium intake can be calculated. Furthermore, the existing correlation between sodium and calories may be an artifact of the current food supply.

6. Determine the effects of replacement of saturated fat with different types of carbohydrates (e.g., refined vs. whole grains) on cardiovascular disease risk.

   Rationale: Most randomized controlled trials and prospective cohort studies compared saturated fat with total carbohydrates. It is important to distinguish different types of carbohydrates (e.g. refined vs. whole grains) in future studies.

7. Examine the effects that replacement of saturated fat with polyunsaturated fat vs. monounsaturated fat has on cardiovascular disease risk.

   Rationale: Most existing studies have examined the effects of substituting PUFA for saturated fat on cardiovascular disease risk. Future studies should also examine the potential benefits of substituting monounsaturated fat from plant sources such as olive oil and nuts/seeds for saturated fat on cardiovascular disease risk.

8. Examine lipid and metabolic effects of specific oils modified to have different fatty acid profiles (e.g. commodity soy oil [high linoleic acid] vs. high oleic soy oil).

   Rationale: As more modified vegetables oils become commercially available, it is important to assess their long-term health effects. In addition, future studies should examine lipid and metabolic effects of plant oils that contain a mix of *n*-9, *n*-6, and *n*-3 fatty acids, as a replacement for animal fat, on cardiovascular disease risk factors.

9. Examine the effects of saturated fat from different sources, including animal products (e.g. butter, lard), plant (e.g., palm vs. coconut oils), and production systems (e.g. refined deodorized bleached vs. virgin coconut oil) on blood lipids and cardiovascular disease risk.

   Rationale: Different sources of saturated fat contain different fatty acid profiles and thus, may result in different lipid and metabolic effects. In addition, virgin and refined coconut oils have different effects in animal models, but human data are lacking.

10. Conduct gene-nutrient interaction studies by measuring genetic variations in relevant genes that will enable evaluation of effects of specific diets for individualized nutrition recommendations.

Rationale: Individuals with different genetic background may respond to the same dietary intervention differently in terms of blood lipids and other cardiovascular disease risk factors. Future studies should explore the potential role of genetic factors in modulating the effects of fat type modification on health outcomes.

11. Identify sources and names of added sugars and low-calorie sweeteners used in the food supply and quantify their consumption levels and trends in the U.S. diet.

Rationale: It is unclear whether all food and nutrient databases capture all added sugars because: 1) added sugars have varied and inconsistent nomenclature and may not be recognized as added sugars in nutrient analyses; and 2) many foods with added sugars have formulations considered proprietary by the manufacturers and for this reason actual added sugars content is difficult to obtain. Accurate assessment of added sugars in the U.S. diet is needed to quantify the population level exposure and subsequent health risks from added sugars. The lack of information on the various added sugars in the food supply hinders efforts to make policy about consumption.

12. Conduct prospective research with strong experimental designs and multiple measurements of the consumption of added sugars and low-calorie sweeteners on health outcomes, such as body weight, adiposity, and clinical markers of type 2 diabetes and cardiovascular disease.

Rationale: High heterogeneity exists among published research with regard to the types and forms of added sugars and low-calorie sweeteners-containing foods/beverages used for interventions, which precludes assessing the effects of specific added sugars and low-calorie sweeteners on body weight, adiposity, and cardio-metabolic health in adults and children. Many studies use single baseline measurements of diet to reflect usual patterns and quantities of intake over time. New research should emphasize assessments within the context of usual dietary intakes and patterns of food and beverage consumption in free-living populations, along with specific added sugars and low-calorie sweeteners, especially those that are currently understudied. Large prospective studies with repeated measurements of low-calorie sweeteners are needed to monitor their long-term effects on cancer and other health outcomes.

13. Design studies that emphasize assessments of relationships between the intakes of added sugars and low-calorie sweeteners and body weight, adiposity, and cardio-metabolic health in diverse sub-populations who are at high risk of obesity and related morbidities.

Rationale: Insufficient evidence exists to assess the impact of added sugars and low-calorie sweeteners contained in foods and beverages on individuals from diverse populations who have high risk for adverse health outcomes. These include (but not limited to) different race/ethnicity groups; low income groups, especially those with food insecurity; groups who live in specific geographic locations with high prevalence of obesity (e.g. inner city, rural, and Southern regions of the United States); and age and sex groups (women, children, and elderly adults).

14. Assess and improve approaches and policies to reduce the amount of added sugars in the food and beverage supply as well as in school and community settings.

    Rationale: Results from this research would assist policy makers and the private sector in establishing sustainable approaches and policies to limit the availability and consumption of added sugars. These approaches and policies would also be important for multi-component strategies to improve weight control and health among people living in the United States.

15. Conduct consumer research to identify and test elements of a standardized, easily understood front-of-package label.

    Rationale: Research is needed to provide an evidence base to support the need and identify critical elements of a front of package label. This is particularly important to support the Food and Drug Administration in implementing a front-of-package labeling system.

REFERENCES

1. National Heart Lung and Blood Institute. Lifestyle Interventions to Reduce Cardiovascular Risk: Systematic Evidence Review from the Lifestyle Work Group. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health; 2013. Available from: http://www.nhlbi.nih.gov/health-pro/guidelines/in-develop/cardiovascular-risk-reduction/lifestyle/index.htm.
2. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63(25 Pt B):2960-84. PMID: 24239922. http://www.ncbi.nlm.nih.gov/pubmed/24239922.
3. Mente A, O'Donnell MJ, Rangarajan S, McQueen MJ, Poirier P, Wielgosz A, et al. Association of urinary sodium and potassium excretion with blood pressure. N Engl J Med. 2014;371(7):601-11. PMID: 25119606. http://www.ncbi.nlm.nih.gov/pubmed/25119606.
4. Institute of Medicine. Sodium Intake in Populations: Assessment of Evidence. Washington, D.C.: The National Academies Press; 2013. Available from: http://www.nap.edu/catalog.php?record_id=18311.

5. Institute of Medicine. Strategies to Reduce Sodium Intake in the United States. Washington D.C.: The National Academies Press; 2010.
6. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2013;346:e7492. PMID: 23321486. http://www.ncbi.nlm.nih.gov/pubmed/23321486.
7. Moynihan PJ, Kelly SA. Effect on caries of restricting sugars intake: systematic review to inform WHO guidelines. J Dent Res. 2014;93(1):8-18. PMID: 24323509. http://www.ncbi.nlm.nih.gov/pubmed/24323509.
8. U.S. Department of Agriculture. Agricultural Research Services. Nutrient Intakes from Food: Mean Amounts Consumed per Individual, by Gender and Age, *What We Eat in America*, NHANES 2009-2010. 2012. Available from: www.ars.usda.gov/ba/bhnrc/fsrg.
9. Institute of Medicine. Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate. Washington, D.C.: The National Academies Press; 2005. Available from: http://books.nap.edu/openbook.php?record_id=10925.
10. Graudal NA, Hubeck-Graudal T, Jürgens G. Effects of low-sodium diet vs. high-sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). Am J Hypertens. 2012;25(1):1-15. PMID: 22068710. http://www.ncbi.nlm.nih.gov/pubmed/22068710.
11. Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010, to the Secretary of Agriculture and the Secretary of Health and Human Services: U.S. Department of Agriculture, Agricultural Research Service, Washington D.C.; 2010. Available from: http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm.
12. Cotter J, Cotter MJ, Oliveira P, Cunha P, Polonia J. Salt intake in children 10-12 years old and its modification by active working practices in a school garden. J Hypertens. 2013;31(10):1966-71. PMID: 24107730. http://www.ncbi.nlm.nih.gov/pubmed/24107730.
13. Shi L, Krupp D, Remer T. Salt, fruit and vegetable consumption and blood pressure development: a longitudinal investigation in healthy children. Br J Nutr. 2014;111(4):662-71. PMID: 24326147. http://www.ncbi.nlm.nih.gov/pubmed/24326147.
14. Cook NR, Appel LJ, Whelton PK. Lower levels of sodium intake and reduced cardiovascular risk. Circulation. 2014;129(9):981-9. PMID: 24415713. http://www.ncbi.nlm.nih.gov/pubmed/24415713.
15. Joosten MM, Gansevoort RT, Mukamal KJ, Lambers Heerspink HJ, Geleijnse JM, Feskens EJ, et al. Sodium excretion and risk of developing coronary heart disease. Circulation. 2014;129(10):1121-8. PMID: 24425751. http://www.ncbi.nlm.nih.gov/pubmed/24425751.
16. O'Donnell M, Mente A, Rangarajan S, McQueen MJ, Wang X, Liu L, et al. Urinary sodium and potassium excretion, mortality, and cardiovascular events. N Engl J Med. 2014;371(7):612-23. PMID: 25119607. http://www.ncbi.nlm.nih.gov/pubmed/25119607.
17. Pfister R, Michels G, Sharp SJ, Luben R, Wareham NJ, Khaw KT. Estimated urinary sodium excretion and risk of heart failure in men and women in the EPIC-Norfolk study. Eur J Heart Fail. 2014. PMID: 24464931. http://www.ncbi.nlm.nih.gov/pubmed/24464931.
18. Institute of Medicine. Front-of-Package Nutrition Rating Systems and Symbols: Promoting Healthier Choices. Washingon D.C.: The National Academies Press; 2012.

19. Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, Johnson L, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. Ann Intern Med. 2014;160(6):398-406. PMID: 24723079. http://www.ncbi.nlm.nih.gov/pubmed/24723079.

20. Farvid MS, Ding M, Pan A, Sun Q, Chiuve SE, Steffen LM, et al. Dietary Linoleic Acid and Risk of Coronary Heart Disease: A Systematic Review and Meta-Analysis of Prospective Cohort Studies. Circulation. 2014. PMID: 25161045. http://www.ncbi.nlm.nih.gov/pubmed/25161045.

21. Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore HJ, et al. Reduced or modified dietary fat for preventing cardiovascular disease. Cochrane Database Syst Rev. 2012;5:CD002137. PMID: 22592684. http://www.ncbi.nlm.nih.gov/pubmed/22592684.

22. Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Bälter K, Fraser GE, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. Am J Clin Nutr. 2009;89(5):1425-32. PMID: 19211817. http://www.ncbi.nlm.nih.gov/pubmed/19211817.

23. Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. PLoS Med. 2010;7(3):e1000252. PMID: 20351774. http://www.ncbi.nlm.nih.gov/pubmed/20351774.

24. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. Am J Clin Nutr. 2010;91(3):535-46. PMID: 20071648. http://www.ncbi.nlm.nih.gov/pubmed/20071648.

25. Skeaff CM, Miller J. Dietary fat and coronary heart disease: summary of evidence from prospective cohort and randomised controlled trials. Ann Nutr Metab. 2009;55(1-3):173-201. PMID: 19752542. http://www.ncbi.nlm.nih.gov/pubmed/19752542.

26. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. Arterioscler Thromb. 1992;12(8):911-9. PMID: 1386252. http://www.ncbi.nlm.nih.gov/pubmed/1386252.

27. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr. 2003;77(5):1146-55. PMID: 12716665. http://www.ncbi.nlm.nih.gov/pubmed/12716665.

28. Department of Health and Human Services. Food and Drug Administration. Proposed definition from FDA in the Proposed Rule to the revision of the Nutrition and Supplement Facts Labels (Docket No. FDA-2012-N-1210). March 2014. Available from: https://www.federalregister.gov/articles/2014/03/03/2014-04387/food-labeling-revision-of-the-nutrition-and-supplement-facts-labels#p-325.

29. Malik VS, Pan A, Willett WC, Hu FB. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. Am J Clin Nutr. 2013;98(4):1084-102. PMID: 23966427. http://www.ncbi.nlm.nih.gov/pubmed/23966427.

30. Kaiser KA, Shikany JM, Keating KD, Allison DB. Will reducing sugar-sweetened beverage consumption reduce obesity? Evidence supporting conjecture is strong, but evidence when testing effect is weak. Obes Rev. 2013;14(8):620-33. PMID: 23742715. http://www.ncbi.nlm.nih.gov/pubmed/23742715.

31. Mattes RD, Shikany JM, Kaiser KA, Allison DB. Nutritively sweetened beverage consumption and body weight: a systematic review and meta-analysis of randomized experiments. Obes Rev. 2011;12(5):346-65. PMID: 20524996. http://www.ncbi.nlm.nih.gov/pubmed/20524996.

32. The science behind the sweetness in our diets. Bull World Health Organ. 2014;92(11):780-1. PMID: 25378738. http://www.ncbi.nlm.nih.gov/pubmed/25378738.

33. Greenwood DC, Threapleton DE, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, et al. Association between sugar-sweetened and artificially sweetened soft drinks and type 2 diabetes: systematic review and dose-response meta-analysis of prospective studies. Br J Nutr. 2014;112(5):725-34. PMID: 24932880. http://www.ncbi.nlm.nih.gov/pubmed/24932880.

34. Romaguera D, Norat T, Wark PA, Vergnaud AC, Schulze MB, van Woudenbergh GJ, et al. Consumption of sweet beverages and type 2 diabetes incidence in European adults: results from EPIC-InterAct. Diabetologia. 2013;56(7):1520-30. PMID: 23620057. http://www.ncbi.nlm.nih.gov/pubmed/23620057.

35. Malik VS, Popkin BM, Bray GA, Després JP, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care. 2010;33(11):2477-83. PMID: 20693348. http://www.ncbi.nlm.nih.gov/pubmed/20693348.

36. Sonestedt E, Overby NC, Laaksonen DE, Birgisdottir BE. Does high sugar consumption exacerbate cardiometabolic risk factors and increase the risk of type 2 diabetes and cardiovascular disease? Food Nutr Res. 2012;56. PMID: 22855643. http://www.ncbi.nlm.nih.gov/pubmed/22855643.

37. Xi B, Li S, Liu Z, Tian H, Yin X, Huai P, et al. Intake of fruit juice and incidence of type 2 diabetes: a systematic review and meta-analysis. PLoS One. 2014;9(3):e93471. PMID: 24682091. http://www.ncbi.nlm.nih.gov/pubmed/24682091.

38. Aeberli I, Gerber PA, Hochuli M, Kohler S, Haile SR, Gouni-Berthold I, et al. Low to moderate sugar-sweetened beverage consumption impairs glucose and lipid metabolism and promotes inflammation in healthy young men: a randomized controlled trial. Am J Clin Nutr. 2011;94(2):479-85. PMID: 21677052. http://www.ncbi.nlm.nih.gov/pubmed/21677052.

39. Aeberli I, Hochuli M, Gerber PA, Sze L, Murer SB, Tappy L, et al. Moderate amounts of fructose consumption impair insulin sensitivity in healthy young men: a randomized controlled trial. Diabetes Care. 2013;36(1):150-6. PMID: 22933433. http://www.ncbi.nlm.nih.gov/pubmed/22933433.

40. Barrio-Lopez MT, Martinez-Gonzalez MA, Fernandez-Montero A, Beunza JJ, Zazpe I, Bes-Rastrollo M. Prospective study of changes in sugar-sweetened beverage consumption and the incidence of the metabolic syndrome and its components: the SUN cohort. Br J Nutr. 2013;110(9):1722-31. PMID: 23534417. http://www.ncbi.nlm.nih.gov/pubmed/23534417.

41. Bernstein AM, de Koning L, Flint AJ, Rexrode KM, Willett WC. Soda consumption and the risk of stroke in men and women. Am J Clin Nutr. 2012;95(5):1190-9. PMID: 22492378. http://www.ncbi.nlm.nih.gov/pubmed/22492378.

42. Black RN, Spence M, McMahon RO, Cuskelly GJ, Ennis CN, McCance DR, et al. Effect of eucaloric high- and low-sucrose diets with identical macronutrient profile on insulin

resistance and vascular risk: a randomized controlled trial. Diabetes. 2006;55(12):3566-72. PMID: 17130505. http://www.ncbi.nlm.nih.gov/pubmed/17130505.

43. Chen L, Caballero B, Mitchell DC, Loria C, Lin PH, Champagne CM, et al. Reducing consumption of sugar-sweetened beverages is associated with reduced blood pressure: a prospective study among United States adults. Circulation. 2010;121(22):2398-406. PMID: 20497980. http://www.ncbi.nlm.nih.gov/pubmed/20497980.

44. Cohen L, Curhan G, Forman J. Association of sweetened beverage intake with incident hypertension. J Gen Intern Med. 2012;27(9):1127-34. PMID: 22539069. http://www.ncbi.nlm.nih.gov/pubmed/22539069.

45. de Koning L, Malik VS, Kellogg MD, Rimm EB, Willett WC, Hu FB. Sweetened beverage consumption, incident coronary heart disease, and biomarkers of risk in men. Circulation. 2012;125(14):1735-41, S1. PMID: 22412070. http://www.ncbi.nlm.nih.gov/pubmed/22412070.

46. Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, et al. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation. 2007;116(5):480-8. PMID: 17646581. http://www.ncbi.nlm.nih.gov/pubmed/17646581.

47. Duffey KJ, Gordon-Larsen P, Steffen LM, Jacobs DR, Popkin BM. Drinking caloric beverages increases the risk of adverse cardiometabolic outcomes in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. Am J Clin Nutr. 2010;92(4):954-9. PMID: 20702604. http://www.ncbi.nlm.nih.gov/pubmed/20702604.

48. Eshak ES, Iso H, Kokubo Y, Saito I, Yamagishi K, Inoue M, et al. Soft drink intake in relation to incident ischemic heart disease, stroke, and stroke subtypes in Japanese men and women: the Japan Public Health Centre-based study cohort I. Am J Clin Nutr. 2012;96(6):1390-7. PMID: 23076619. http://www.ncbi.nlm.nih.gov/pubmed/23076619.

49. Faeh D, Minehira K, Schwarz JM, Periasamy R, Periasami R, Park S, et al. Effect of fructose overfeeding and fish oil administration on hepatic de novo lipogenesis and insulin sensitivity in healthy men. Diabetes. 2005;54(7):1907-13. PMID: 15983189. http://www.ncbi.nlm.nih.gov/pubmed/15983189.

50. Fung TT, Malik V, Rexrode KM, Manson JE, Willett WC, Hu FB. Sweetened beverage consumption and risk of coronary heart disease in women. Am J Clin Nutr. 2009;89(4):1037-42. PMID: 19211821. http://www.ncbi.nlm.nih.gov/pubmed/19211821.

51. Larsson SC, Akesson A, Wolk A. Sweetened beverage consumption is associated with increased risk of stroke in women and men. J Nutr. 2014;144(6):856-60. PMID: 24717367. http://www.ncbi.nlm.nih.gov/pubmed/24717367.

52. Lê KA, Ith M, Kreis R, Faeh D, Bortolotti M, Tran C, et al. Fructose overconsumption causes dyslipidemia and ectopic lipid deposition in healthy subjects with and without a family history of type 2 diabetes. Am J Clin Nutr. 2009;89(6):1760-5. PMID: 19403641. http://www.ncbi.nlm.nih.gov/pubmed/19403641.

53. Lewis AS, McCourt HJ, Ennis CN, Bell PM, Courtney CH, McKinley MC, et al. Comparison of 5% versus 15% sucrose intakes as part of a eucaloric diet in overweight and obese subjects: effects on insulin sensitivity, glucose metabolism, vascular compliance, body composition and lipid profile. A randomised controlled trial. Metabolism. 2013;62(5):694-702. PMID: 23363580. http://www.ncbi.nlm.nih.gov/pubmed/23363580.

54. Lowndes J, Sinnett S, Pardo S, Nguyen VT, Melanson KJ, Yu Z, et al. The effect of normally consumed amounts of sucrose or high fructose corn syrup on lipid profiles, body composition and related parameters in overweight/obese subjects. Nutrients. 2014;6(3):1128-44. PMID: 24642950. http://www.ncbi.nlm.nih.gov/pubmed/24642950.
55. Lowndes J, Sinnett S, Yu Z, Rippe J. The effects of fructose-containing sugars on weight, body composition and cardiometabolic risk factors when consumed at up to the 90th percentile population consumption level for fructose. Nutrients. 2014;6(8):3153-68. PMID: 25111121. http://www.ncbi.nlm.nih.gov/pubmed/25111121.
56. Maersk M, Belza A, Stødkilde-Jørgensen H, Ringgaard S, Chabanova E, Thomsen H, et al. Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat depot: a 6-mo randomized intervention study. Am J Clin Nutr. 2012;95(2):283-9. PMID: 22205311. http://www.ncbi.nlm.nih.gov/pubmed/22205311.
57. Raben A, Vasilaras TH, Møller AC, Astrup A. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. Am J Clin Nutr. 2002;76(4):721-9. PMID: 12324283. http://www.ncbi.nlm.nih.gov/pubmed/12324283.
58. Tasevska N, Park Y, Jiao L, Hollenbeck A, Subar AF, Potischman N. Sugars and risk of mortality in the NIH-AARP Diet and Health Study. Am J Clin Nutr. 2014;99(5):1077-88. PMID: 24552754. http://www.ncbi.nlm.nih.gov/pubmed/24552754.
59. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added sugar intake and cardiovascular diseases mortality among US adults. JAMA Intern Med. 2014;174(4):516-24. PMID: 24493081. http://www.ncbi.nlm.nih.gov/pubmed/24493081.
60. Yu Z, Lowndes J, Rippe J. High-fructose corn syrup and sucrose have equivalent effects on energy-regulating hormones at normal human consumption levels. Nutr Res. 2013;33(12):1043-52. PMID: 24267044. http://www.ncbi.nlm.nih.gov/pubmed/24267044.
61. Brown RJ, de Banate MA, Rother KI. Artificial sweeteners: a systematic review of metabolic effects in youth. Int J Pediatr Obes. 2010;5(4):305-12. PMID: 20078374. http://www.ncbi.nlm.nih.gov/pubmed/20078374.
62. Miller PE, Perez V. Low-calorie sweeteners and body weight and composition: a meta-analysis of randomized controlled trials and prospective cohort studies. Am J Clin Nutr. 2014;100(3):765-77. PMID: 24944060. http://www.ncbi.nlm.nih.gov/pubmed/24944060.
63. Wiebe N, Padwal R, Field C, Marks S, Jacobs R, Tonelli M. A systematic review on the effect of sweeteners on glycemic response and clinically relevant outcomes. BMC Med. 2011;9:123. PMID: 22093544. http://www.ncbi.nlm.nih.gov/pubmed/22093544.
64. World Health Organization. Draft guidelines on free sugars released for public consultation, 5 March 2014. http://www.who.int/nutrition/sugars_public_consultation/en/.
65. Van Horn L, Johnson RK, Flickinger BD, Vafiadis DK, Yin-Piazza S, Group ASCP. Translation and implementation of added sugars consumption recommendations: a conference report from the American Heart Association Added Sugars Conference 2010. Circulation. 2010;122(23):2470-90. PMID: 21060079. http://www.ncbi.nlm.nih.gov/pubmed/21060079.
66. U.S. Department of Health and Human Services. Office of Disease Prevention and Health Promotion. Healthy People 2020. Objective NWS-17.2. Available from:

https://www.healthypeople.gov/2020/topics-objectives/topic/nutrition-and-weight-status/objectives.

67. Barlow SE, Committee E. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;120 Suppl 4:S164-92. PMID: 18055651. http://www.ncbi.nlm.nih.gov/pubmed/18055651.
68. American Academy of Pediatrics Committee on School Health. Soft drinks in schools. Pediatrics. 2004;113(1 Pt 1):152-4. PMID: 14702469. http://www.ncbi.nlm.nih.gov/pubmed/14702469.
69. American Academy of Pediatrics. Sweeteners and Sugar Substitutes. Available from: http://www.healthychildren.org/English/healthy-living/nutrition/Pages/Sweeteners-and-Sugar-Substitutes.aspx.
70. American Diabetes Association. Sugar and Desserts [November 2014]. Available from: http://www.diabetes.org/food-and-fitness/food/what-can-i-eat/understanding-carbohydrates/sugar-and-desserts.html.
71. Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care. 2013;36(11):3821-42. PMID: 24107659. http://www.ncbi.nlm.nih.gov/pubmed/24107659.
72. National Heart Lung and Blood Institute. Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health; 2013.

Part D. Chapter 7: Physical Activity

INTRODUCTION

The combination of a healthy diet and regular physical activity is central to promoting overall health and preventing many chronic diseases. The *Dietary Guidelines for Americans* first emphasized the importance of physical activity in 1990 and has included the topic in every edition in the two decades since. Although the 1990 and 1995 *Dietary Guidelines for Americans* discussed physical activity as a tool for managing and maintaining a healthy body weight, it broadened this perspective with the 2000 edition. Beginning in 2000, the *Dietary Guidelines for Americans'* physical activity content reflected the growing evidence base on the relationship between physical activity and various health outcomes. This evidence, from a wide range of well-conducted studies, clearly demonstrates that physically active people have improved growth and development, higher levels of fitness, a lower risk profile for developing a number of disabling medical conditions, and lower rates of various chronic diseases than do people who are less active or sedentary.[1]

In 2008, the U.S. Department of Health and Human Services issued the first *Physical Activity Guidelines for Americans* (PAG).[2] The PAG serves as the benchmark and single, authoritative voice for science-based guidance on physical activity, fitness, and health for Americans 6 years and older (Table D7.1). The content of the PAG complements the *Dietary Guidelines for Americans*. Recognizing the dual importance of being physically active and eating a healthy diet to promote good health and reduce the risk of chronic diseases, therefore, the 2015 DGAC included a number of physical activity questions, including several related to body weight.

Table D7.1. ***2008 Physical Activity Guidelines for Americans:*** **Key Recommendations**

Recommendations for Children and Adolescents Ages 6 to 17 Years

Children and adolescents should do 60 minutes (1 hour) or more of physical activity daily.

- **Aerobic:** Most of the 60 or more minutes a day should be either moderate- or vigorous-intensity aerobic physical activity, and should include vigorous-intensity physical activity at least 3 days a week.
- **Muscle-strengthening:** As part of their 60 or more minutes of daily physical activity, children and adolescents should include muscle-strengthening physical activity on at least 3 days of the week.
- **Bone-strengthening:** As part of their 60 or more minutes of daily physical activity, children and adolescents should include bone-strengthening physical activity on at least 3 days of the week.
- It is important to encourage young people to participate in physical activities that are appropriate for their age, that are enjoyable, and that offer variety.

Recommendations for Adults Ages 18 Years and Older

- All adults should avoid inactivity. Some physical activity is better than none, and adults who participate in any amount of physical activity gain some health benefits.
- For substantial health benefits, adults should do at least 150 minutes (2 hours and 30 minutes) a week of moderate-intensity, or 75 minutes (1 hour and 15 minutes) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, it should be spread throughout the week.
- For additional and more extensive health benefits, adults should increase their aerobic physical activity to 300 minutes (5 hours) a week of moderate intensity, or 150 minutes a week of vigorous intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity activity. Additional health benefits are gained by engaging in physical activity beyond this amount.
- Adults should also do muscle-strengthening activities that are moderate or high intensity and involve all major muscle groups on 2 or more days a week, as these activities provide additional health benefits.

Recommendations for Older Adults

The PAG recommendations for adults also apply to older adults. In addition, the following Guidelines are just for older adults (ages 65 years and older):

- When older adults cannot do 150 minutes of moderate-intensity aerobic activity a week because of chronic conditions, they should be as physically active as their abilities and conditions allow.
- Older adults should do exercises that maintain or improve balance if they are at risk of falling.
- Older adults should determine their level of effort for physical activity relative to their level of fitness.
- Older adults with chronic conditions should understand whether and how their conditions affect their ability to do regular physical activity safely.

Despite the consistent public health advice and encouragement to engage in regular physical activity, the majority of the U.S. population does not meet PAG recommendations. Using self-reported measures, in 2012 fewer than 21 percent of adults met the PAG recommendations for aerobic and muscle-strengthening physical activity, with fewer women than men meeting recommendations.[3] As reported in the National Health Interview Survey, physical activity participation rates are lower in Blacks or African Americans and Hispanic or Latinos than in White populations. Older adults had the lowest participation rates across all adult age groups.[3] In

2013, only 27 percent of adolescents met PAG recommendations; again, fewer girls than boys achieved recommended levels of physical activity.[4]

It is important to note that self-reported data on physical activity participation rates are likely to have significant over-reporting bias.[5] Using objective accelerometer data on a nationally representative sample, Troiano et al. demonstrated that the percentage of the population meeting PAG recommendations was much lower than with self-report. For example, when considering bouts of moderate- to vigorous-intensity aerobic physical activity lasting 8 to 10 minutes or longer, less than 5 percent of adults met 2008 PAG recommendations.[5] Nonetheless, some data indicate that Americans may be increasing their level of physical activity. Over the past six years, consistent data show a minimal, but positive, trend (Tables D7.2a and D7.2b).[3,6-8]

Table D7.2a. Proportion of adults who self-report meeting the Physical Activity Guidelines for Americans recommendations for aerobic and muscle-strengthening physical activity

Population	2008	2009	2012	2013
Adult Total:	18.2%	19.0%	20.6%	*
Adult Male	21.7%	22.0%	24.3%	
Adult Female	14.9%	16.2%	17.1%	

Table D7.2b. Proportion of adolescents who self-report meeting the Physical Activity Guidelines for Americans recommendations for aerobic physical activity

Population	2008	2009	2012	2013
Adolescent Total:	**	18.4%	**	27.1%
Adolescent Boys		24.8%		36.6%
Adolescent Girls		11.4%		17.7%

* National Health Interview Survey, 2013 data unavailable at time of publication.
** Youth Risk Behavior Surveillance was not conducted in 2008 or 2012.
Sources: Pleis, 2008; Pleis, 2009; Blackwell et al., 2014; CDC, 2010; CDC, 2014

To ensure sufficient discussion of physical activity for the population across the life cycle, as well as its relationship with a range of health outcomes, the DGAC reviewed the three major Federal reports on physical activity and health outcomes and selected specific questions for inclusion in this chapter. The Committee did not conduct independent formal systematic reviews of the evidence. This chapter summarizes the key evidence contained in these reports of the benefits of physical activity on health. Due to the extensive nature and number of evidence reviews within the three reports, the Committee refers readers to specific information using hyperlinks in each review of evidence found in this chapter.

LIST OF QUESTIONS

Physical Activity and Health Outcomes in Children and Adolescents

1. What is the relationship between physical activity, body weight, and health outcomes in children and adolescents?

Physical Activity and Health Outcomes in Adults

2. What is the relationship between physical activity and body weight?
3. What is the relationship between physical activity and cardiorespiratory health?
4. What is the relationship between physical activity and metabolic health and risk of type 2 diabetes?
5. What is the relationship between physical activity and musculoskeletal health?
6. What is the relationship between physical activity and incidence of breast and colon cancer?
7. What is the relationship between physical activity and mental health?

Physical Activity and Health Outcomes in People with Disabilities

8. What is the relationship between physical activity and health outcomes in people with disabilities?

Physical Activity and Health Outcomes During Pregnancy and the Postpartum Period

9. Does being physically active during pregnancy and the postpartum period provide health benefits?

Physical Activity and Adverse Events

10. What is the relationship between the amount and type of physical activity and the risk of adverse events?

Physical Activity Dose

11. What dose of physical activity is most likely to provide health benefits in children and adolescents?
12. What dose of physical activity is most likely to provide health benefits in adults?
13. Are there any special considerations for dose of physical activity for older adults?

Physical Activity Interventions in Children and Adolescents

14. What is the relationship between physical activity participation and interventions in school-based settings?
15. What is the relationship between physical activity participation and interventions to change the built environment?
16. What is the relationship between physical activity participation and interventions based in home settings?
17. What is the relationship between physical activity participation and interventions based in early care and education centers?
18. What is the relationship between physical activity participation and interventions based in primary health care settings?

METHODOLOGY

The DGAC agreed to use existing systematic reviews and reports to address the physical activity topic area. The Committee used the PAG and two related reports—the *Physical Activity Guidelines Advisory Committee Report, 2008* (PAGAC) and the *Physical Activity Guidelines for Americans Midcourse Report*—as primary sources of evidence[1,2,9] and discussed at its public meetings questions that could be developed to frame the reports' key findings. The DGAC reviewed and extracted information on the methodological approaches from each report and identified key findings. The DGAC then carried forward verbatim conclusion statements from the PAGAC Report and PAG Midcourse Report and concurred with 2008 PAG recommendations to answer the questions. The DGAC subsequently assigned strength of evidence grades and, based on the various report findings and conclusions, developed an overall physical activity implications statement. Below is a brief description of each of the three reports.

Physical Activity Guidelines Advisory Committee Report, 2008. In 2007, the Secretary of HHS appointed a 13-member Physical Activity Guidelines Advisory Committee and charged them with reviewing existing scientific literature to identify areas where sufficient evidence existed to develop a comprehensive set of specific physical activity recommendations and highlight areas where further scientific research was needed.[1] The PAGAC conducted systematic searches of the scientific literature on physical activity and selected health outcomes in people ages 5 years and older. Similar to the 2010 and 2015 DGAC, the PAGAC developed analytic frameworks for each question and examined a diverse array of literature representing a number of study designs, including randomized controlled trials (RCTs), non-randomized trials, prospective cohort studies, case-control studies, and other observational studies. For each topic area, the PAGAC used the best available and most appropriate body of evidence to answer specific questions. One of the PAGAC's major goals was to integrate the scientific information on the relationship between physical activity and health and to summarize it in a manner that could be used effectively by HHS to develop the *Physical Activity Guidelines for Americans* and related policy statements.

Physical Activity Guidelines for Americans, 2008. In 2008, HHS issued the PAG, which provides science-based guidance to help Americans ages 6 years and older improve their health through appropriate physical activity.[2] The 2008 PAG is designed to provide information and guidance on the types and amounts of physical activity that provide substantial health benefits. The primary audiences for the PAG are policymakers, health professionals, and interested members of the public.

Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among Youth. In spring 2012, HHS convened a subcommittee of the President's Council on Fitness, Sports & Nutrition to review the evidence on strategies to increase youth physical activity and make recommendations. The *Physical Activity Guidelines for Americans Midcourse Report*, released in 2013, is intended to identify interventions that can help increase physical activity in youth across a variety of settings.[9] The subcommittee used a review-of-reviews approach to assess the current literature on interventions to increase physical activity in youth across five selected settings: schools, preschool and childcare centers, community, family and home, and primary health care. A total of 31 reviews covering 910 studies were examined. In its report, the subcommittee expanded the PAG's age focus on those ages 6 years and older to include children ages 3 to 5 years.

Overall, the DGAC concurs with the findings and evidence grades of the *Physical Activity Guidelines Advisory Committee Report, 2008*; the *2008 Physical Activity Guidelines for Americans*; and the *Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among Youth*.[1,2,9] These reports state that being physically active is one of the most important steps that people of all ages can take to improve and maintain their health.

PHYSICAL ACTIVITY AND HEALTH OUTCOMES IN CHILDREN AND ADOLESCENTS

Question 1: What is the relationship between physical activity, body weight, and health outcomes in children and adolescents?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that strong evidence demonstrates that the physical fitness and health status of children and adolescents is substantially enhanced by frequent physical activity. Compared to inactive young people, physically active children and adolescents have higher levels of cardiorespiratory endurance and muscular strength, and well documented health benefits include lower body fatness, more favorable cardiovascular and metabolic disease risk profiles, enhanced bone health, and reduced symptoms of anxiety and depression. These conclusions are based on the results of prospective observational studies in which higher levels of physical activity were found to be associated with favorable health parameters as well as intervention studies in which exercise treatments caused improvements in physical fitness and various health-related factors. **DGAC Grade: Strong**

Review of Evidence

A body of RCTs, non-randomized trials, prospective cohort studies, case-control studies, other observational studies, and meta-analyses support the relationship between physical activity and physical fitness (i.e., cardiorespiratory fitness and muscular strength), healthy body weight and composition, cardio-metabolic health, bone health, and mental health (i.e., anxiety and depression).

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Physical fitness, see Part G. Section 9: Youth
- Body weight and composition, see Part G. Section 9: Youth
- Cardio-metabolic health, see Part G. Section 9: Youth
- Bone health, see Part G. Section 9: Youth
- Mental health, see Part G. Section 9: Youth

PHYSICAL ACTIVITY AND HEALTH OUTCOMES IN ADULTS

Question 2: What is the relationship between physical activity and body weight?

Question 3: What is the relationship between physical activity and cardiorespiratory health?

Question 4: What is the relationship between physical activity and metabolic health and risk of type 2 diabetes?

Question 5: What is the relationship between physical activity and musculoskeletal health?

Question 6: What is the relationship between physical activity and incidence of breast and colon cancer?

Question 7: What is the relationship between physical activity and mental health?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that compared to less active people, physically active adults and older adults exhibit a higher level of cardiorespiratory and muscular fitness, healthier body weight and body composition, and a biomarker profile that is more favorable for preventing cardiovascular disease (CVD) and type 2 diabetes and enhancing bone health. In addition, there is an association between higher levels of physically activity in adults and older adults and lower rates of all-cause mortality, coronary heart disease, high blood pressure, stroke, type 2 diabetes, metabolic syndrome, colon cancer, breast cancer, and depression. High-intensity muscle-strengthening activity enhances skeletal muscle mass, strength, power, and intrinsic neuromuscular activation. Physically active adults who are overweight or obese experience a variety of health benefits that are generally similar to those observed in physically active people of ideal body weight. Physical activity reduces risk of depression and is associated with lower risk of cognitive decline in adults and older adults. Physical activity is associated with higher levels of functional health and a lower risk of falling in older adults. **DGAC Grade: Strong**

In older adults with existing functional limitations, fairly consistent evidence indicates that regular physical activity is safe and has a beneficial effect on functional ability. Consistent evidence indicates that physically active adults and older adults have better quality sleep and health-related quality of life. **DGAC Grade: Moderate**

Review of Evidence

A body of well-designed prospective cohort studies, case-control studies, and other observational studies exists for the relationship between regular physical activity and lower risk of all-cause mortality; coronary heart disease (CHD), CVD, and stroke; type 2 diabetes; metabolic syndrome, body weight, and body composition; bone health; functional health; cancer; and mental health. A body of RCTs and meta-analyses provides evidence for a positive effect of physical activity on blood pressure, atherogenic dyslipidemia, and cardiorespiratory fitness; body weight and body composition; bone health and muscular strength; falls risk; mental health; and type 2 diabetes.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- All-cause mortality, see Part G, Section 1: All-cause Mortality
- Coronary heart disease (CHD), CVD, and stroke; blood pressure, atherogenic dyslipidemia, and cardiorespiratory fitness, see Part G, Section 2: Cardiorespiratory Health
- Type 2 diabetes, see Part G, Section 3: Metabolic Health
- Metabolic syndrome, see Part G, Section 3: Metabolic Health
- Body weight and body composition, see Part G, Section 4: Energy Balance
- Bone health and muscular strength, see Part G, Section 5: Musculoskeletal Health
- Functional health and falls risk, see Part G, Section 6
- Cancer, see Part G, Section 7
- Mental Health, see Part G, Section 8

PHYSICAL ACTIVITY AND HEALTH OUTCOMES IN PEOPLE WITH DISABILITIES

Question 8: What is the relationship between physical activity and health outcomes in people with disabilities?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that for people with physical disabilities, strong evidence shows that exercise can increase cardiorespiratory, musculoskeletal, and mental health outcomes; and for people with cognitive disabilities, strong evidence shows that exercise can improve musculoskeletal health and select functional health and mental health outcomes. **DGAC Grade: Strong**

For people with physical disabilities, moderate evidence indicates that physical activity improves a variety of functional health outcomes and reduces the effects of certain types of secondary conditions (i.e., pain and fatigue associated with the primary disability); and for people with cognitive disabilities, moderate evidence indicates that physical activity improves cardiorespiratory health outcomes, musculoskeletal fitness, and metabolic health, and helps maintain healthy weight. **DGAC Grade: Moderate**

For people with physical disabilities, limited evidence suggests physical activity may promote a healthy weight and improve metabolic health, and for people with cognitive disabilities, limited evidence suggests that physical activity may reduce secondary conditions. **DGAC Grade: Limited**

Based on these conclusions from the 2008 PAGAC, the PAG provided recommendations on physical activity for people with disabilities (Table D7.3). The DGAC concurs with these recommendations.

Table D7.3. PAG Recommendations for Adults with Disabilities
• Adults with disabilities, who are able to, should get at least 150 minutes a week of moderate-intensity, or 75 minutes a week of vigorous-intensity aerobic activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, it should be spread throughout the week.
• Adults with disabilities, who are able to, should also do muscle-strengthening activities of moderate or high intensity that involve all major muscle groups on 2 or more days a week, as these activities provide additional health benefits.
• When adults with disabilities are not able to meet the Guidelines, they should engage in regular physical activity according to their abilities and should avoid inactivity.
• Adults with disabilities should consult their health-care provider about the amounts and types of physical activity that are appropriate for their abilities.

Review of Evidence

A body of RCTs, meta-analyses, and non-randomized trials provides evidence on physical activity in people with physical and cognitive disabilities. Non-randomized trials were included in the review of evidence for this question due to the high variability of physical and cognitive disabilities considered.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Physical and cognitive disabilities, see Part G, Section 11: Understudied Populations. Review of the Science: Health Outcomes Associated with Physical Activity in People With Disabilities (pages G11-2 to G11-35)

For additional details about the PAG recommendations, visit: http://www.health.gov/paguidelines/pdf/paguide.pdf.

PHYSICAL ACTIVITY AND HEALTH OUTCOMES DURING PREGANCY AND THE POSTPARTUM PERIOD

Question 9: Does being physically active during pregnancy and the postpartum period provide health benefits?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that while the benefits of maternal physical activity have clearly been demonstrated, there is a lack of prospective, randomized intervention studies in diverse populations. Based on current evidence, unless there are medical reasons to the contrary, a pregnant woman can begin or continue a regular physical activity program throughout gestation, adjusting the frequency, intensity, and time as her condition warrants. Very little evidence exists for the dose of activity that confers the greatest health benefits to women during pregnancy and the postpartum period. In the absence of data, it is reasonable for women during pregnancy and the postpartum period to follow the moderate-intensity physical activity recommendations set for adults unless specific medical concerns warrant a reduction in activity. **DGAC Grade: Limited**

Based on these conclusions from the 2008 PAGAC, the PAG provided recommendations on physical activity for women who are pregnant or in the postpartum period (Table D7.4). The DGAC concurs with these recommendations.

Table D7.4. PAG Recommendations for Women During Pregnancy and the Postpartum Period
• Healthy women who are not already highly active or doing vigorous-intensity activity should get at least 150 minutes of moderate-intensity aerobic activity a week during pregnancy and the postpartum period. Preferably, this activity should be spread throughout the week. • Pregnant women who habitually engage in vigorous-intensity aerobic activity or who are highly active can continue physical activity during pregnancy and the postpartum period, provided that they remain healthy and discuss with their health care provider how and when activity should be adjusted over time.

Review of Evidence

Laboratory investigations and observational studies provide evidence on physical activity during pregnancy and the postpartum period.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Pregnancy and the postpartum period, see Part G, Section 11: Understudied Populations. Review of the Science: Physical Activity During Pregnancy and the Postpartum Period (pages G11-35 to G11-38)

For additional details about the PAG recommendations, visit: http://www.health.gov/paguidelines/pdf/paguide.pdf.

PHYSICAL ACTIVITY AND ADVERSE EVENTS

Question 10: What is the relationship between the amount and type of physical activity and the risk of adverse events?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that the benefits of regular physical activity outweigh the inherent risk of adverse events. Risk of musculoskeletal injuries is lower for non-contact (e.g., walking) and limited contact (e.g., baseball) activities than for contact (e.g., basketball) and collision (e.g., football) activities. The usual dose of regular physical activity is directly related to the risk of musculoskeletal injury and inversely related to the risk of sudden adverse cardiac events. The risk of musculoskeletal injuries and sudden cardiac adverse events is directly related to the size of the difference between the usual dose of activity and the new or momentary dose of activity. The most consistently reported risk factor for musculoskeletal injuries and sudden cardiac adverse events is inactivity and low fitness. **DGAC Grade: Strong**

Based on these conclusions from the 2008 PAGAC, the PAG provided recommendations on physical activity and reducing the risk of adverse events (Table D7.5). The DGAC concurs with these recommendations.

Table D7.5. PAG Recommendations for Reducing the Risk of Adverse Events

To do physical activity safely and to reduce risk of injuries and other adverse events, people should:

- Understand the risks and yet be confident that physical activity is safe for almost everyone.
- Choose to do types of physical activity that are appropriate for their current fitness level and health goals, because some activities are safer than others.
- Increase physical activity gradually over time whenever more activity is necessary to meet the guidelines or health goals. Inactive people should "start low and go slow" by gradually increasing how often and how long activities are done.
- Protect themselves by using appropriate gear and sports equipment, looking for safe environments, following rules and policies, and making sensible choices about when, where, and how to be active.
- Be under the care of a health care provider if they have chronic conditions or symptoms. People with chronic conditions and symptoms should consult their health care provider about the types and amounts of activity appropriate for them.

Review of Evidence

A body of RCTs, meta-analyses, well-designed prospective cohort studies, and case control studies provides evidence on physical activity and risk of adverse events.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Adverse events, see Part G, Section 10: Adverse Events

For additional details about the PAG recommendations, visit: http://www.health.gov/paguidelines/pdf/paguide.pdf.

PHYSICAL ACTIVITY DOSE

Question 11: What dose of physical activity is most likely to provide health benefits in children and adolescents?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that substantial evidence indicates important health and fitness benefits can be expected to accrue to most children and adolescents who participate daily in 60 or more minutes of moderate to vigorous physical activity. Also, certain specific types of physical activity should be included in an overall physical activity pattern in order for children and adolescents to gain comprehensive health benefits. These include regular participation in each of the following types of physical activity on 3 or more days per week: resistance exercise to enhance muscular strength in the large muscle groups of the trunk and limbs, vigorous aerobic exercise to improve cardiorespiratory fitness and cardiovascular and metabolic disease risk factors, and weight-loading activities to promote bone health. **DGAC Grade: Strong**

Based on these conclusions from the 2008 PAGAC, the PAG provides recommendations on physical activity for children and adolescents (Table D7.1). The DGAC concurs with these recommendations.

Review of Evidence

A body of RCTs, meta-analyses, non-randomized trials, well-designed prospective cohort studies, case-control studies, and other observational studies supports the dose of physical activity most likely to provide health benefits in children and adolescents.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Children and adolescents, see Part G, Section 9: Youth

For additional details about the PAG recommendations, visit: http://www.health.gov/paguidelines/pdf/paguide.pdf.

Question 12: What dose of physical activity is most likely to provide health benefits in adults?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that for overall public health benefit, data from a large number of studies evaluating a wide variety of benefits in diverse populations generally support 30 to 60 minutes per day of moderate- to vigorous-intensity physical activity on 5 or more days of the week. For a number of benefits, including all-cause mortality, coronary heart disease, stroke, hypertension, and type 2 diabetes in adults and older adults, lower risk is consistently observed at 2.5 hours per week of moderate- to vigorous-intensity activity. The amount of moderate- to vigorous-intensity activity most consistently associated with significantly lower rates of colon and breast cancer and the prevention of unhealthy weight gain or significant weight loss by physical activity alone is in the range of 3 to 5 hours per week. The available evidence suggests that the major health benefits of physical activity and the dose needed for major health benefits are similar for all adults, regardless of race or ethnicity. For a variety of health and fitness outcomes, including chronic disease prevention, improvement of various disease biomarkers and the maintenance of a healthy weight, reasonably strong evidence demonstrates that amounts of moderate- to vigorous-intensity activity that exceed 150 minutes per week are associated with greater health benefits. **DGAC Grade: Strong**

Based on these conclusions from the 2008 PAGAC, the PAG provides recommendations on physical activity for adults ages 18 years and older (Table D7.1). The DGAC concurs with these recommendations.

Review of Evidence

A body of well-designed prospective cohort studies and case control studies provides evidence on physical activity dose most likely to provide health benefits in adults.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Adults, see Part E: Integration and Summary of the Science (pages E-23 to E-24)

For additional details about the PAG recommendations, visit: http://www.health.gov/paguidelines/pdf/paguide.pdf.

Question 13: Are there any special considerations for dose of physical activity for older adults?

Source of Evidence: *Physical Activity Guidelines Advisory Committee Report, 2008*

Conclusion

The DGAC concurs with the 2008 PAGAC, which found that, because the exercise capacity of adults tends to decrease as they age, older adults generally have lower exercise capacities than younger persons. Thus, they may need a physical activity plan that is of lower absolute intensity and amount (but similar in self-perceived relative intensity and amount) than is appropriate for more fit people, especially when they have been sedentary and are starting an activity program.

For older adults at risk of falling, strong evidence exists that regular physical activity is safe and reduces falls by about 30 percent. Most evidence supports a program of exercise with the following characteristics: 3 times per week of balance training and moderate-intensity muscle-strengthening activities for 30 minutes per session and with additional encouragement to participate in moderate-intensity walking activities 2 or more times per week for 30 minutes per session. Some evidence, albeit less consistent, suggests that tai chi exercises also reduce falls. Successful reduction in falls by tai chi interventions resulted from programs conducted from 1 to 3 hours or more per week. No evidence indicates that planned physical activity reduces falls in adults and older adults who are not at risk of falls. **DGAC Grade: Strong**

Based on these conclusions from the 2008 PAGAC, the PAG provides recommendations on physical activity for adults ages 65 years and older (Table D7.1). The DGAC concurs with these recommendations.

Review of Evidence

A body of RCTs, meta-analyses, and non-randomized trials provides evidence on physical activity dose in older adults.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines Advisory Committee Report, 2008* at http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf.

For evidence reviews on:

- Older adults, see Part E: Integration and Summary of the Science (pages E-23 to E-24)

For additional details about the PAG recommendations, visit: http://www.health.gov/paguidelines/pdf/paguide.pdf.

PHYSICAL ACTIVITY INTERVENTIONS FOR CHILDREN AND ADOLESCENTS

Question 14: What is the relationship between physical activity participation and interventions in school-based settings?

Question 15: What is the relationship between physical activity participation and interventions to change the built environment?

Question 16: What is the relationship between physical activity participation and interventions based in home settings?

Question 17: What is the relationship between physical activity participation and interventions based in early care and education centers?

Question 18: What is the relationship between physical activity participation and interventions based in primary health care settings?

Source of Evidence: *Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among Youth*

Conclusion

The DGAC concurs with the *Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among Youth*, which found that multi-component school-based interventions that include strategies such as physical education, active transportation, and activity breaks can increase physical activity in children and adolescents during school hours. **DGAC Grade: Strong**

Reasonably consistent evidence suggests that changing the built environment as well as interventions in early care and education centers can increase physical activity in children and adolescents. **DGAC Grade: Moderate**

Evidence to date is insufficient to conclude that intervention strategies in home or primary health care settings increase physical activity in children and adolescents. **DGAC Grade: Grade Not Assignable**

Review of Evidence

A body of systematic reviews and meta-analyses supports interventions to increase physical activity in children and adolescents.

For additional details on this body of evidence, visit: Appendix E-2.49 and *Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among Youth* at http://www.health.gov/paguidelines/midcourse/pag-mid-course-report-final.pdf.

For evidence reviews on:

- School-based interventions, see School Setting (pages 9 to 14)
- Early care and education interventions, see Preschool and Childcare Center Setting (page 15)
- Built environment interventions, see Community Setting (pages 16 to 18)
- Home-based interventions, see Family and Home Setting (page 19)
- Primary care interventions, see Primary Health Care Setting (pages 20 to 21)

IMPLICATIONS

Given the strong evidence for health benefits of regular physical activity as well as the low levels of adherence to national recommendations, every effort should be made to encourage and facilitate programs at multiple levels so that children, adults, and older adults can meet the 2008 PAG in combination with the *Dietary Guidelines for Americans.* This can be achieved if programs, policies, and communication strategies are developed across sectors to increase opportunities for engaging in physical activity and to improve the built environment. Ultimately, these actions can create a culture of health that facilitates participation in regular physical activity. Individuals, communities, schools, health care, and the private and public sectors should:

- Ensure that all individuals have access to safe, affordable, and enjoyable modes of physical activity throughout the day in the environments where they live, learn, work, and play. These opportunities must include structured programming and informal modes of transportation and play.
- Focus particular attention on people with the greatest health disparities, as these individuals have the lowest physical activity participation rates but can gain the most health benefits by being physically active.
- Support policies and promote programs for children, adolescents, adults, and older adults that help set and reinforce a personal value system that instills a lifetime of physical activity.
- Enact effective policies and strengthen existing policies within schools, communities, health care settings, housing, and worksites that promote opportunities for regular physical activity.
- Enact effective policies and strengthen existing policies that promote active transport (e.g., walking and bicycling) within and between communities.
- Develop and promote programs to create or enhance access to safe and enjoyable places to be physically active, including public spaces and local, state, and national parks.

- Develop and implement ongoing physical activity promotion campaigns that involve high-visibility and multiple delivery channels and multiple sectors of influence.
- Coordinate efforts between numerous Federal and non-Federal initiatives, such as the President's Council on Fitness, Sports and Nutrition, *Let's Move!*, the National Physical Activity Plan, and Active Schools Acceleration Project.

CHAPTER SUMMARY

The findings outlined in this chapter provide strong evidence supporting the importance of regular physical activity for health promotion and disease prevention in the U.S. population. Physical activity is important for all people—children, adolescents, adults, older adults, women during pregnancy and the postpartum period, and individuals with disabilities. The findings further provide guidance on the dose of physical activity needed across the lifecycle to realize these significant health benefits.

Future Physical Activity Guidelines Advisory Committees will be asked to carefully review the most recent evidence so that the Federal government can fully update the PAG. Given the exceedingly low physical activity participation rates in this country, it will be critically important for the next PAGAC to identify proven strategies and approaches to increase population-level physical activity across the lifespan.

NEEDS FOR FUTURE RESEARCH

1. Evaluate best practices in programming at the community and national level and identify which local and national policies in the public and private sector have demonstrated the greatest effect on increasing physical activity participation across the lifespan, especially in populations with the greatest health disparities.

   Rationale: Physical activity participation rates are exceptionally low across all age groups, and are especially low in individuals with the greatest health disparities. Many different initiatives are currently underway in the private and public sector to help increase physical activity on a population level. Understanding which programs and policies are having the greatest impact will help focus valuable resources and national recommendations for maximum public health benefit.

2. Identify the dose of physical activity needed to achieve health benefits, as well as appropriate growth and development, for children younger than age 6 years.

   Rationale: Until recently, very little effort has been focused on understanding the health benefits of physical activity for young children. Given that this is a critical age of growth and development, considerable research should be focused on this age group.

3. Evaluate the effects of various modes and doses of physical activity on health outcomes in older adults.

   Rationale: Older adults are the fastest growing segment of the population. They also have the greatest burden of disease and functional (mental and physical) limitations. To reduce burden of disease and related economic impacts, research regarding mode and dose of physical activity should be focused on this age group.

4. Further evaluate the importance of light activity, short bouts of physical activity (i.e., 10-minutes or less) and modes of activity on health outcomes across the lifespan.

   Rationale: The review of the evidence in the 2008 PAGAC Report focused primarily on moderate- and vigorous-intensity activity. Emerging research highlights the positive effects of light activity as well as shorter bouts of vigorous activity on health outcomes. Understanding the health impact of the full range of mode, intensity, duration, frequency, and setting will help to further refine the PAG to support maximum public health benefit.

5. Further investigate the effects of sedentary behaviors on health outcomes, including duration, frequency, and mode of sedentary activities.

   Rationale: Increasing evidence demonstrates the negative health consequences of sedentary behaviors. Clarity on the types and duration of sedentary behaviors that have the most negative health impact would help to identify meaningful evidence-based public health recommendations.

REFERENCES

1. Physical Activity Guidelines Advisory Committee. Physical Activity Guidelines Advisory Committee Report, 2008. Washington, DC: U.S. Department of Health and Human Services; 2008. Available from: http://www.health.gov/paguidelines/Report/pdf/CommitteeReport.pdf
2. U.S. Department of Health and Human Services. 2008 Physical Activity Guidelines for Americans. Washington, DC: U.S. Department of Health and Human Services; 2008. Available from: http://www.health.gov/paguidelines/pdf/paguide.pdf
3. Blackwell DL, Lucas JW, Clarke TC. Summary health statistics for U.S. adults: national health interview survey, 2012. *Vital and health statistics. Series 10, Data from the National Health Survey.* Feb 2014(260):1-161. PMID: 24819891. http://www.ncbi.nlm.nih.gov/pubmed/24819891
4. Kann L, Kinchen S, Shanklin SL, Flint KH, Kawkins J, Harris WA et al. Youth Risk Behavior Surveillance — United States, 2013. *MMWR.* 2014;63(4). PMID: 24918634. http://www.ncbi.nlm.nih.gov/pubmed/24918634
5. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, McDowell M. Physical activity in the United States measured by accelerometer. *Medicine and science in sports and exercise.* Jan 2008;40(1):181-188. PMID: 18091006. http://www.ncbi.nlm.nih.gov/pubmed/18091006
6. Pleis JR, Lucas JW, Ward BW. Summary health statistics for U.S. adults: National Health Interview Survey, 2008. *Vital and health statistics. Series 10, Data from the National Health*

Survey. Dec 2009(242):1-157. PMID: 20821903. http://www.ncbi.nlm.nih.gov/pubmed/20821903

7. Pleis JR, Ward BW, Lucas JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2009. *Vital and health statistics. Series 10, Data from the National Health Survey*. Dec 2010(249):1-207. PMID: 21905346. http://www.ncbi.nlm.nih.gov/pubmed/21905346
8. Eaton DK, Kann L, Kinchen S, Shanklin S, Ross J, Hawkins J, et al. Youth Risk Behavior Surveillance — United States, 2009. *MMWR*. 2010;59(5). http://www.ncbi.nlm.nih.gov/pubmed/20520591
9. U.S. Department of Health and Human Services. Physical Activity Guidelines for Americans Midcourse Report: Strategies to Increase Physical Activity Among Youth. Washington, DC: U.S. Department of Health and Human Services; 2012. http://www.health.gov/paguidelines/midcourse/pag-mid-course-report-final.pdf

Appendix E-1: Needs for Future Research

CHAPTER 1: FOOD AND NUTRIENT INTAKES AND HEALTH: CURRENT STATUS AND TRENDS

1. Expand WWEIA participation to include more respondents from race/ethnic minorities and non-U.S. born residents.

   Rationale: Very little is known about the dietary habits of many of the cultural subgroups in the United States. This knowledge is essential to moving forward any nutrition programs for first and second generation immigrants. More data on the impact of acculturation also are needed on food and health behaviors. The number of participants in WWEIA using the derived acculturation variable was too small for any analysis. Finally, "Hispanic" is a very broad term and a better understanding is needed of the nutritional profiles (including shortfalls and excesses) across various Spanish-speaking people in the United States, who come from different cultural backgrounds with distinct eating patterns.

2. Include higher proportion of older Americans as respondents in WWEIA.

   Rationale: More data are needed on dietary intake of older adults; the sample sizes in WWEIA were too small for any meaningful analyses for those older than the age of 71 years. In addition to nutrient intake, additional information is needed on whether older adults are able to shop and cook, whether polypharmacy plays a role in nutritional adequacy, and whether co-morbidities, such as poor dentition, musculo-skeletal difficulties, arthralgias and other age-related symptoms, affect their ability to establish and maintain proper nutritional status.

3. Increase the number of pregnant women as respondents in WWEIA.

   Rationale: The number of pregnant women in WWEIA is currently too small to properly evaluate the status and trends in food and nutrient intake in pregnant women. Since good nutrition in pregnancy is critical to proper growth development of the infant it is critical to properly evaluate food and nutrient intake, which will inform recommendations and public policies for pregnant women.

4. Conduct research on nutrition transitions from childhood to shed light on how and why dietary intake changes so rapidly from early childhood through pre-adolescence and adolescence, and to identify the driving forces behind dietary intake change in these age groups and what programs are most effective at maintaining positive nutrition habits established in very young children.

   Rationale: Young children have better dietary intake than older children and adolescents. It is important to maintain the positive gains made in early childhood and identify factors responsible for the declines in intakes of fruit, dairy, and other food groups and increases in added sugars and refined grains as children become enter the elementary school age years, as poor eating patterns in elementary school seem to persist into adolescence and beyond.

5. Evaluate the effects of common variations in dietary patterns in small children on nutrient intakes.

   Rationale: Children from 2 to 4 years of age have a highly variable diet and often do not fit readily into the USDA Food Pattern food groups diet pattern analyses. Further information is needed to understand the broad range of diets and supplement use in small children and how this relates to nutrient intake and growth. Research is needed to better characterize their diets so that appropriate guidance can be offered.

6. Increase the quantity and quality of food composition databases available for research.

   Rationale: Accurate assessment of nutrient intake and trends over time in the U.S. population is dependent upon the quality of food composition data. Tens of thousands of foods are available for purchase and consumption in the United States, but accurate nutrient content data are available only for less than 10,000 foods and are almost non-existent for many ready-to-eat and restaurant-type foods. Analytic values from foods are needed on specific nutrients and components, such as vitamin D, fiber, added sugars, and sodium. Improved food composition data also is critical for needed research to better define, identify, and quantify total grain, whole grain consumption, and refined grain consumption in dietary studies.

7. Investigate the validity, reliability, and reproducibility of new biomarkers of nutrient intake and biomarkers of nutritional status.

   Rationale: Limited biomarkers are available and some that are available are difficult to interpret due to other contributing factors to the biomarker measure (e.g., vitamin D is obtained in the diet and is also endogenously synthesized).

8. Evaluate effects of fortification strategies and supplement use on consumer behavior related to the intake of foods and supplements containing key nutrients, including calcium, vitamin D, potassium, iron, and fiber

   Rationale: The intake of key nutrients of concern is considerably affected by the rapidly evolving marketplace of food fortification and supplementation. Understanding consumer behavior related to fortification and supplementation would be important in predicting the effects of interventions and marketplace changes in content of these nutrients. Special interest exists regarding fortification strategies of foods, including whole grains and yogurts, in allowing individuals to reach the RDA for vitamin D without using supplements. Data are needed on how supplements may help meet nutrients shortfalls and/or how use of supplements may place individuals at risk of overconsumption. Research on effective consumer guidance is needed.

9. Understand the rationale for and consequences of the use of supplements above the UL for vitamins and minerals. Identify biochemical markers that would indicate the effects of high-dose supplement use.

   Rationale: Consumer use of high-dose supplements has increased. Understanding the influences guiding this use would be helpful in considering how to educate consumers about safe upper intake limits.

10. Develop a standardized research definition for meals and snacks.

    Rationale: Multiple different criteria are used in studies to define a snack or meal occasion, such as time of day, the types or amounts of food consumed, or subjective assessment by the study respondent. Researchers should work toward a consensus on the use of standard definitions.

11. Understand better the concept of dietary patterns and design approaches to quantify the diet in large population-based studies.

    Rationale: More methodological work on dietary patterns is needed. For example, food frequency questionnaires, which are used in most diet assessment studies, do not capture data on meal timing, meal frequency, or the types of foods consumed together. Studies using diet recalls and records are better at capturing specific foods and their quantities consumed (portion sizes) and the types of foods eaten together, but often these detailed assessment methods are not feasible for large population-based studies. Quantification of food group intake is needed. In addition, dietary patterns research encompasses a broader scope of issues than can be addressed by diet scores and data drive approaches.

12. Consistently report the nutrients, foods, and food groups that are used to evaluate dietary patterns in published studies.

    Rationale: The current scientific literature evaluating dietary patterns and health is inconsistent in its provision of dietary patterns composition information. This makes it difficult to compare, across studies, the components of healthful patterns that are associated with health benefits.

13. Conduct population surveillance on the prevalence and trends of nutrition-related chronic diseases including type 2 diabetes, cardiovascular disease, some cancers osteoporosis and neurocognitive disorders.

    Rationale: Current data on diabetes in adults cannot be stratified by disease type (type I or type II), making it very difficult to monitor incidence and prevalence of type 2 diabetes.

Continued population surveillance is needed to effectively link nutritional factors with risk of these diseases.

CHAPTER 2: DIETARY PATTERNS, FOODS AND NUTRIENTS, AND HEALTH OUTCOMES

1. Conduct additional dietary patterns research for other health outcomes to strengthen the evidence beyond CVD and body weight in populations of various ethnic backgrounds and life course stages in order for future DGACs to draw stronger conclusions.

   Rationale: The NEL systematic reviews demonstrated that considerable CVD research related to dietary patterns is available. However, it also is important to note, that unlike CVD, some of the other health outcomes are more heterogeneous and thus may require greater specificity in the examination of diet and disease risk. There is a clear need for all studies examining the relationship between dietary patterns and health outcomes to include the full age spectrum and to take a life course perspective (including pregnancy); insufficient research is being devoted to children and how diseases may evolve over time. An increased emphasis should be placed on understanding how the diets of all those in the U.S. population from various ethnic backgrounds may be associated with health outcomes, thereby broadening knowledge beyond Hispanics and African Americans to include the diversity that exists in the United States today. This may require our national nutrition monitoring programs to over-sample individuals from other national origins to conduct subgroup analysis.

2. Improve the understanding of how to more precisely characterize dietary patterns by their food constituents and the implications of the food constituents on nutrient adequacy through the use of Food Pattern Modeling. More precise characterization, particularly of protein foods, is needed.

   Rationale: Researchers are characterizing dietary patterns very differently and yet sometimes use similar nomenclatures. This makes it difficult to compare results across studies and as demonstrated in the NEL systematic reviews, can impair the grading of the body of evidence as strong. The reason why researchers are not replicating others findings in different populations may be a function of publication bias. It is important for editors of scientific journals and peer reviewers to appreciate the replication of findings first and then value a research group's methodological nuance that may improve the examination of the association between dietary patterns and a health outcomes. Perhaps what should be stressed is a harmonization of research methods across various cohorts or randomized trials, similar to what is being done at the National Cancer Institute's Dietary Patterns Methods Project[9, 220] led by Drs. Krebs-Smith and Reedy. The use of Food Pattern Modeling as demonstrated in Chapter 1 allows questions about the adequacy of the dietary patterns given specific food constituents to be addressed and how modifications of the patterns by altering the foods for specific population groups or to meet specific nutrient targets can be achieved.

3. Examine the long-term cardio-metabolic effects of the various dietary patterns identified in the AHA/ACC/TOS Guidelines for the Management of Overweight and Obesity in Adults that are capable of resulting in short-term weight loss (see Question 2, above).

   Rationale: Although the research to date demonstrates that to lose weight, a variety of dietary pattern approaches can be used if a reduction in caloric intake is achieved, the long-term effects of these diets on cardio-metabolic health are not well known. Emerging research is exploring health effects of variations of the low-carbohydrate, higher protein/fat dietary pattern. In some approaches (such as Atkins), the dietary pattern which emphasizes animal products, may achieve a macronutrient composition that is higher in saturated fat. Others may emphasize plant-based proteins and fats and may achieve a lower saturated fat content and may be higher in polyunsaturated fats and dietary fiber. Research is needed to determine the impact of these alternative approaches, and perhaps others, on CVD risk profiles as well as other health outcomes. As mentioned in the review of the literature associated with saturated fat and cardiovascular disease in Chapter 6: Cross-Cutting Topics of Dietary Guidance and Public Health Importance, substituting one macronutrient for another may result in unintended consequences. Careful consideration to the types of foods that are used in these diets and in particular the type of fat and amount of added sugars should be taken into account.

CHAPTER 3: INDIVIDUAL DIET AND PHYSICAL ACTIVITY BEHAVIOR CHANGE

Eating Out

1. Develop a standard methodology to collect and characterize various types of eating venues.

   Rationale: This recommendation is fundamental to conducting rigorous research, evaluating findings from multiple studies, and developing policies to promote healthy eating among people who frequent eating out venues and/or consume take away meals.

2. Conduct rigorously designed research to examine the longitudinal impact of obtaining or consuming meals away from home from various types of commonly frequented venues on changes in food and beverage intakes (frequency, quantity, and composition), body weight, adiposity, and health profiles from childhood to adulthood in diverse (racial/ethnic, socioeconomic, cultural, and geographic) groups of males and females.

   Rationale: Most groups in the U.S. population regularly consume meals that are prepared away from home and the landscape of fast food and other types of food procurement and consumption venues is increasingly complex. The potential for eating out and/or take away meals to influence diet quality, energy balance, body mass and composition, and the risks of health-related morbidities across the life course among our diverse population underscores the importance of understanding this issue.

Family Shared Meals

3. Conduct studies in diverse populations that assess not only frequency of family shared meals, but also quality of family shared meals.

   Rationale: Our understanding of the importance of family shared meals in terms of how they contribute in a positive way to body weight and overall health and well-being requires a rigorous examination of the dietary quality of these meals compared to other meals consumed by family members.

4. Conduct RCTs to isolate the effect of interventions that increase the frequency of family meals from other health and parenting behaviors that may be associated with dietary intake and weight status.

   Rationale: Family shared meals are commonly implemented as one component of lifestyle interventions that include an array of other behavioral and parenting strategies for weight management. To improve our understanding of the causal pathway of how family shared meals contributes to maintaining or achieving a health weight, the specific contribution of family shared meals to weight outcomes independent of other behavioral strategies needs to be ascertained.

Sedentary Behavior

5. Develop improved and better standardized and validated tools to assess sedentary behaviors and activities that children, adolescents, and adults regularly engage in.

   Rationale: Our understanding of the impact of sedentary behaviors on diet, energy balance, body mass, adiposity, and health is currently compromised by reliance on subjective assessments, including self-reports of daily activity patterns, and by inadequate techniques to document and quantify the array of sedentary activities people engage in (beyond TV viewing and (or) computer screen time). It also would be beneficial for researchers to document the potential benefits and implications of reducing one type of sedentary behavior (e.g. screen time) on other sedentary behaviors (e.g., reading for leisure, arts and crafts, listening to music) and indices of health (e.g. sleep quality and duration).

6. Conduct prospective research to examine the effects and mechanisms of the quantity, patterns, and changes of sedentary behaviors on diet quality, energy balance, body weight,

adiposity, and health across the life span in groups within the U.S. population with diverse personal, cultural, economic, and geographic characteristics.

Rationale: Emerging, but limited, evidence implicates sedentary behaviors with adverse health-related outcomes, especially in children and adolescents as they transition into adulthood. However, an improved understanding of why these relationships exist will help in developing appropriate and effective approaches and policies to reduce the amount of time people spend engaging in sedentary behaviors.

Self-Monitoring

7. Evaluate the impact of different types, modalities, and frequencies of self-monitoring on body weight outcomes during both the weight loss intervention and maintenance periods.

   Rationale: Self-monitoring is associated with improved weight management. However, the current practice of recommending daily self-monitoring may represent a barrier to its implementation and/or continued use. Hence, it is important to determine whether lower frequencies of self-monitoring can produce beneficial effects on weight outcomes.

8. Evaluate the comparative effectiveness of performance feedback from self-monitoring delivered through automated systems versus personal interactions with a counselor.

   Rationale: Automated feedback derived from self-monitoring data and delivered electronically can produce beneficial changes on weight outcomes. However, the comparative effectiveness and cost efficiency of feedback delivered through non-personal modalities versus personal interactions has yet to be determined.

9. Test the effectiveness of self-monitoring on weight outcomes in understudied groups, including ethnic/racial minorities, low education, low literacy, and low numeracy populations, males, and subjects younger than age 30 years and older than age 60 years.

   Rationale: Evidence regarding the effectiveness of self-monitoring has been derived largely from research conducted on well educated, middle-class, white women. Hence, it is important to determine whether the beneficial effects of self-monitoring on weight outcomes are generalizable to understudied groups.

10. Conduct RCTs based on sound behavioral change theories that incorporate self-monitoring, employ heterogeneous populations, and are powered for small effect sizes and high attrition rates, to test the short- (e.g., 3 months) and long-term (e.g., 12 months) effects of mobile health technologies on dietary and weight outcomes.

    Rationale: Mobile health technologies have the potential to reach larger portions of the populations than face-to-face interventions, but the effect sizes of mobile technologies may be small and the attrition rates may be large. Larger, more representative study populations and longer study periods will permit an assessment of the generalizability and sustainability of mobile health technologies.

Food and Menu Labeling

11. Develop novel labeling approaches to provide informative strategies to convey caloric intake values on food items consumed at home and in restaurant settings.

    Rationale: Menu labels can include different types of information in addition to calories. These include physical activity equivalents, and daily caloric needs. Very few studies have been designed to examine the optimal combination of menu label information to prevent excessive caloric intake. This will be very valuable evidence to inform the calorie label policy that has just been enacted by the FDA.

12. Compare labeling strategies across various settings, such as restaurants, stores, and the home to determine their efficacy in altering food selection and health outcomes, including weight.

    Rationale: The great majority of menu labeling RCT's have been conducted under laboratory conditions. Given the recent FDA regulations, future studies will be able to impact the effectiveness of these polices across settings as accessed by diverse free living populations.

13. Evaluate the process and impact of recent FDA menu labeling regulation.

    Rationale: The new FDA regulation provides a unique opportunity to understand the impact of menu labeling on consumers dietary behaviors in "real world" settings.

Household Food Insecurity

14. Conduct prospective cohort studies that cover a wide age range and include children, families, older adults, and ethnically/racially diverse populations and describe potential effect modifiers such as gender, ethnic and cultural factors, family structure, area of residence (i.e., urban vs. rural), employment, and use of social support systems while examining the relationship between household food insecurity, dietary intake, and body weight.

    Rationale: Understanding the temporal process of when and how long food insecurity occurs within a family/individual’s lifetime and their response to this economic stressor is critical to conducting rigorous research and comparing finding across studies in order to develop and implement intervention studies and policies to alleviate this public health problem.

15. Standardize research methodology, including developing a consistent approach to measuring food insecurity and use of measured height and weight to reduce the likelihood of responder bias.

    Rationale: The measurement error issues related to the use of self-reported weight have been well documented in the literature. In order to conduct rigorous studies in this area that can be compared and evaluated as to the causal nature of the role of food insecurity on body weight, standard methodology is warranted both in the measurement of the exposure as well as the outcome.

Acculturation

16. Conduct prospective longitudinal studies, including those that start in early childhood to track dietary intake, sedentary behaviors, body weight, and chronic disease outcomes across the life course. Include the diversity of ethnic/racial groups in the United States, including individuals and families of diverse national origins. Include comparison groups in countries of origin to rule out, among other things, the potential confounding by internal migration from rural to urban area within the country of origin.

    Rationale: Acculturation is a time-dependent life course process that requires longitudinal studies to be properly understood. Because the impact of acculturation on dietary, weight and health outcomes can be expected to be modified by the life course stage of life when individuals migrate to the United States, prospective acculturation studies need to start following individuals from very early childhood.

17. Develop a standard tool to measure acculturation or validation of multidimensional acculturation scales in different immigrant groups and in different languages.

    Rationale: Acculturation is a complex construct that is seldom measured with multidimensional scales that can capture the different paths that migrant scan take with regards to the acculturation process, including assimilation, integration, segregation, and marginalization. Although research in acculturation measurement has been conducted among Hispanic/Latinos, it has been predominantly based on Mexican American populations and little acculturation measurement research has been conducted among other groups, including individuals from Asia, Africa, Europe, and the Middle East.

Sleep Patterns

18. Conduct prospective studies that start in childhood (including transition to adulthood), to investigate the longitudinal effect of sleep patterns on diet and body weight outcomes while accounting for confounders, mediators, and moderators including: physical activity, socioeconomic variables (such as education, employment, household income), sex, alcohol intake, smoking status (including new smoker, new non-smoker), media use/screen time, and depression.

    Rationale: While research associates short sleep duration and disordered sleep patterns with adverse differences and changes in food and beverage consumption, body weight, and indices of metabolic and cardiovascular health, less is known about the impact of potential modifying lifestyle factors. This research will help delineate the role of sleep patterns, duration and quality, i.e., mediator or moderator, on diet and weigh-related outcomes. Research in children shows that sleep deprivation and weight are related but this relationship is not apparent in adult studies. This may be due to the fact that energy intake increases during transition to short sleep duration, but levels off when short sleep duration becomes consistent.

19. Conduct studies to assess the effects of diet on sleep quality to examine the mechanism by which dietary intake, energy intake, and energy expenditure may impact sleep.

    Rationale: Most research has focused on sleep quality and duration as modifying factors on diet, body weight, and health. A paucity of research exists on the potential impact of diet on sleep-related outcomes. This line of research would use diet as the means to improve indices of sleep, which in turn may subsequently improve health-related outcomes.

CHAPTER 4: FOOD ENVIRONMENT AND SETTINGS

1. Develop more valid and reliable methods for measuring all aspects of the food environment, including the total food environment of communities. These methods can then be used to assess the impact of the food environment on community health as well as on economic development and growth.

   Rationale: The food environment has become more complex, with more and more retail outlets selling food and beverages. Having valid and reliable methodologies for a variety of food environments and settings (tools and new analytical approaches) will allow more meaningful inquiry into the contributions of various settings in supporting or hindering nutritional health.

2. Identify, implement, evaluate, and scale up best practices (including private-public partnerships) for affordable and sustainable solutions to improving the food environment and increasing food access, especially in those environments of greatest need.

   Rationale: The environments in which people live, work, learn, and play greatly influence their food intake. To best guide efforts to improve the food environment, research is needed to identify and evaluate best practices to direct available resources to new programs and scale up.

3. Identify, implement, accelerate, evaluate, and scale up programs that improve access to healthy food and that can be integrated seamlessly with Federal nutrition assistance programs, such as SNAP, WIC and elder nutrition.

   Rationale: Federal nutrition assistance programs reach individuals and populations with the greatest health disparities. Identifying and evaluating initiatives that integrate improvements in the food environment with Federal programs will help ensure that Federal nutrition assistance programs have as great an impact as possible.

4. Conduct additional obesity prevention intervention research in child care settings (e.g., child-care centers, family child-care homes) to: 1) Identify the most potent components of the interventions and the optimal combinations for improving diet quality, physical activity, and weight outcomes; 2) Assess implementation and translation costs and benefits of the intervention, including impact, cost-effectiveness, generalizability and reach, sustainability and feasibility; 3) Develop and evaluate culturally appropriate and tailored interventions for preschool children in low-income and racial/ethnic communities, given the disproportionate impact of obesity in these groups; 4) Explore intervention strategies on how to use child care settings as access points to create linkages to parents, caretakers, and health care providers as partners in health promotion; 5) Evaluate the impact of Federal, state, and local policies, regulations, and support (e.g., provider training and technical assistance) for child care programs on the eating and physical activity practices and behaviors, and weight status of young children.

   Rationale: Early care and education settings are an important venue for interventions targeting young children. A strong evidence base is essential to identify and support evidence-based practices and policies that can be implemented at Federal, state, and local levels and to mobilize efforts to improve healthy eating and physical activity, leading to healthy weight development in these settings. Interventions found to effectively reduce risk of obesity in one setting need to be appropriately adapted for diverse groups and different settings.

5. Improve intervention research methods by the use of stronger study designs and the development of standardized assessments of body composition, weight status. Develop enhanced validated measures of diet quality, feeding and physical activity practices, and physical activity and eating behaviors and policies. Create standardized measures to assess the nutrition quality of meals and snacks in child care settings, as well as the food and physical activity environments. Create standardized methods for assessing the relationship of child care food, nutrition and physical activity-related measures to similar measures representing non-child care time are needed to provide greater consistency in determining the contributors to the development and progression of childhood overweight and obesity.

   Rationale: Although many of the studies included in these evidence reviews were methodologically strong and were controlled studies, some were limited by small sample size, lack of adequate control for confounding factors, and different outcome measures and different tools used to measure the outcome variables.

6. Examine the effect of the recommended Child and Adult Care Food Program (CACFP) through ongoing periodic evaluations and fill gaps in the knowledge regarding participation, demand, food procurement and practices, nutrient intake, and food security.

   Rationale: Improvements in school meals and the school food environment have been fostered by national data from periodic studies such as the USDA/FNS School Nutrition Dietary Assessment Studies (SNDA), the HHS/CDC School Health Policies and Practices Studies (SHPPS) and the HHS/NIH C.L.A.S.S. In contrast, considerably fewer periodic national studies are conducted of meals and dietary intake in child care settings and their relation to the child care food and physical activity environment.

7. Conduct new research to document the types and quantities of foods and beverages students consume both at school and daily outside of school, before, during, and after school-based healthy eating approaches and policies are implemented.

   Rationale: Effective school-based approaches and policies to improve the availability, accessibility, and consumption of healthy foods and beverages, and reduce competition from unhealthy offerings, are central to improving the weight status and health of children and adolescents. Accurate quantification of the types and quantities of foods and beverages the students consume before, during, and after approaches and policies are implemented is fundamental to assessing effectiveness. However, many of the studies included in the systematic reviews and meta-analyses used by the DGAC to address this issue did not comprehensively measure or report dietary information. Although the USDA/FNS-sponsored School Nutrition Dietary Assessment (SNDA) series collects student dietary intake data every 10 years, the DGAC recommends more frequent and consistent data collection, especially before and periodically after implementation of school-based nutrition and physical activity policy and program changes.

8. Improve the quality of research studies designed to assess the effects of school-based approaches and policies on dietary behaviors and body weight control to reduce the risk of bias, with an emphasis on randomized controlled trials.

   Rationale: Although the methodological quality of the systematic reviews and meta-analyses used by the DGAC to evaluate school-based approaches and policies on dietary intake and body weight outcomes was high, the authors of these reviews commented that the scientific quality of individual studies was generally poor and the risk of bias high. Many of the studies were done using quasi-experimental (with or without control), pre-post intervention, or cross-sectional designs. Future research should prioritize using prospective, repeated measures, randomized controlled trial experimental designs, with randomization at the individual, classroom, school, or school district level. Pilot feasibility studies also may be helpful to quickly identify promising novel approaches to improve dietary intake and weight control outcomes.

9. Conduct post-program follow-up assessments lasting longer than 1 year to determine the long-term retention of the changed nutrition behaviors as well as the usefulness of continuing to offer the programs while children advance in school grade. Also, conduct research is needed in adolescents (grades 9-12).

   Rationale: Literature supports that eating and physical activity behaviors and body weight status of children predict changes over time as they progress into adolescence and adulthood. Ideally, improvements in dietary intake and weight status achieved due to a given school-based approach or policy would be sustained over time and progressive improvements would occur long-term. The vast majority of published research focuses on children in grades K-8, or ages 4-12 years, and new and improved data are needed on adolescents and the transition from childhood to adolescence.

10. Encourage a wider variety of school-based approaches and policies to develop and evaluate innovative approaches focused on increasing vegetable intakes.

    Rationale: Consumption of non-potato vegetables is below 2010 Dietary Guidelines for Americans recommendations in both children and adolescents. Published research indicates that school-based approaches and policies designed to increase vegetable and fruit intakes are generally more effective at increasing fruit intake, except for –school gardens and economic incentives, which increase vegetable intake among school-aged children. Some past public policies (e.g. the Basic 4) treated fruit and vegetables and as a single food group, which props the need for new research that uses prospective, repeated measures, and randomized controlled trial experimental designs to specifically target increased consumption of healthy vegetables.

11. Conduct assessments of the effectiveness of worksite interventions that emphasize obesity prevention and weight control among workers across racially/ethnically diverse populations, blue and white collar employees, and at-risk populations. Scientifically rigorous studies (especially randomized controlled trials) addressing the long-term health impact of worksite-based approaches and policies that improve employee diet, physical activity, and body weight control would have public health relevance.

    Rationale: In light of the high rates of obesity and overweight, worksite interventions targeting obesity prevention and weight control through enhanced dietary behaviors and increased physical activity among workers is important. The majority of the studies to date have been conducted for relatively short periods of time, and the long-term impact of these approaches and policies may prove beneficial.

CHAPTER 5: FOOD SUSTAINABILITY AND SAFETY

Dietary Patterns and Sustainability

1. Conduct research to determine whether sustainable diets are affordable and accessible to all sectors of the population and how this can be improved, including how policy strategies could influence the supply chain (all steps from farm to plate) to affect this improvement.

   Rationale: Ensuring that sustainable diets are accessible and affordable to all sectors of the population is important to promote food security.

2. Develop, conduct, and evaluate in-depth analyses of U.S. domestic dietary patterns and determine the degree to which sustainability practices, domestically and internationally, are important to food choice and how to increase public awareness of the impact of food choices on environmental outcomes.

   Rationale: Understanding consumer choice across demographic groups and the degree to which either health and/or sustainability is a significant decisional criterion as well as the degree to which choice theory can be used to improve choices will be important to helping drive change.

3. Develop a robust understanding of how production practices, supply chain decisions, consumer behaviors, and waste disposal affect the environmental sustainability of various practices across the USDA food components of MyPlate.

   Rationale: Developing sustainable production and supply chain practices for all parts of MyPlate, especially meat and dairy products will be important to reduce their environmental impact.

4. Determine the potential economic benefits and challenges to supply chain stakeholders in relationship to findings in Research Recommendation 3.

   Rationale: Experience demonstrates that many practices over the past few decades that improve the environmental footprint of, for example, production practices, also have led to improved profit (e.g., Integrated Pest Management to reduce pesticide use in many fruit and vegetables). It is important to know how changes will affect profit to help enable future policy in both the private and public spheres.

Seafood Sustainability

5. Conduct research on methods to ensure the maintenance of nutrient profiles of high-trophic level farmed seafood and improve nutrient profiles of low-trophic farmed seafood concurrently with research to improve production efficacy.

   Rationale: The evidence supporting healthfulness of seafood consumption is based on consumption of predominantly wild caught species. Many popular low-trophic level farmed seafood have nutrient profiles that depend on feeds. Efficient production of seafood with nutrient profiles that are known to be healthful should be emphasized.

6. Conduct research to develop methods to ensure contaminant levels in all seafood remain at levels similar to or lower than at present. Maintain monitoring of contaminant levels for capture fisheries to ensure that levels caused by pollution do not rise appreciably. This research should include developing effective rapid response approaches if the quality of seafood supply is acutely affected.

   Rationale: Current research findings support the contention that contaminant levels are generally well below those that significantly alter the healthfulness of seafood. As industry naturally improves efficiency, feeds and environmental conditions should be monitored to maintain or reduce priority contaminants and insure significant new contaminants do not enter the seafood supply.

Usual Caffeine/Coffee Intake

7. Evaluate the effects of coffee on health outcomes in vulnerable populations, such as women who are pregnant (premature birth, low birth weight, spontaneous abortion).

   Rationale: Given the limited evidence of the effects of coffee/caffeine consumption on pregnancy outcomes, future studies need to establish safe levels of coffee/caffeine consumption during pregnancy.

8. Examine the effects of coffee on sleep patterns, quality of life, and dependency and addiction.

   Rationale: Because coffee is a known stimulant, future research should examine the effect of coffee/caffeine on sleep quality, dependency, addiction, and overall quality of life measures.

9. Evaluate the prospective association between coffee/caffeine consumption and cancer at different sites.

   Rationale: Large well-conducted prospective cohort studies that adequately control for smoking (status and dosage) and other potential confounders are needed to understand the association of coffee (caffeinated and decaffeinated) with cancer at different sites.

10. Examine prospectively the effects of coffee/caffeine on cognitive decline, neurodegenerative diseases, and depression.

    Rationale: Neurodegenerative diseases affect millions of people worldwide and more than five million Americans are living with Alzheimer's disease. Given the limited evidence of coffee/caffeine on neurodegenerative diseases, well-designed prospective studies should examine the association of coffee/caffeine consumption on cognitive decline, depression, and Alzheimer's disease.

11. Understand the mechanisms underlying the protective effects of coffee on diabetes and CVD.

    Rationale: Evidence for a biological plausibility for coffee on risk of type 2 diabetes and CVD stems primarily from animal studies. Randomized controlled trials in humans should evaluate the effect of coffee/caffeine on measures of glycemia, insulin sensitivity, endothelial dysfunction, and inflammation.

12. Understand the association between coffee and health outcomes in individuals with existing CVD, diabetes, cancer, neurodegenerative diseases, or depressive symptoms.

    Rationale: Strong evidence supports a protective effect of moderate coffee consumption on chronic disease risk in healthy adults, but its association among those with existing diseases has been less studied. Given that a substantial number of people suffer from these chronic diseases, the role of coffee in preventing other health outcomes in such groups remains understudied.

High-dose Caffeine Intake

13. Define excessive caffeine intake and safe levels of consumption for children, adolescents, and young adults.

    Rationale: Current research on caffeine and health outcomes has focused primarily on adults. Given the increasing prevalence of energy drink consumption among children, adolescents, and young adults, research is needed to identify safe levels of consumption in these groups.

14. Determine the prevalence of excessive caffeine intake in children and adults beyond intake of energy drinks.

    Rationale: Data on the sources (other than energy drinks) and doses of caffeine intake in children and adults are limited. Identifying the sources and safe levels of consumption will help in formulating policy and framing recommendations.

15. Examine the effect of excessive consumption of caffeine and energy drinks on health outcomes in both children and adults.

    Rationale: Prospective studies of associations of excessive caffeine and energy drink intake with health outcomes in children and adults are necessary, as randomized controlled trials are not be feasible given ethical constraints.

16. Conduct observational studies to examine the health effects of alcohol mixed with energy drinks.

    Rationale: In recent years, consumption of alcohol energy drinks by adolescents has resulted in emergency room admissions and deaths. No data exist on the prospective association between consumption of alcohol energy drinks and health outcomes in both adolescents and adults.

Aspartame

17. Examine the risks of aspartame related to some cancers, especially hematopoietic ones, and pregnancy outcomes.

    Rationale: Limited and inconsistent evidence suggests a possible association between aspartame and risk of hematopoietic cancers (non-Hodgkin lymphoma and multiple myeloma) in men, indicating the need for long-term human studies. Additionally, limited and inconsistent evidence indicates a potential for risk of preterm delivery, which warrants further research.

CHAPTER 6: CROSS-CUTTING TOPICS OF PUBLIC HEALTH IMPORTANCE

1. Design and conduct studies with sufficient power to define the impact of improving dietary quality, including the lowering dietary sodium intake, on hypertension and relevant disease outcomes, including cardiovascular disease, stroke, peripheral vascular disease, kidney disease, and others. The interactions with patterns of therapeutic medication use (e.g., diuretics, antihypertensives, and lipid-lowering) should be considered.

   Rationale: The current literature is incomplete, limited in power and durations, and often compromised by methodological challenges that must be addressed in well-designed studies with relevant clinical outcomes.

2. Assess the accuracy of 24-hour urine collections for sodium assessment in populations with different health conditions (e.g., diabetes, chronic kidney disease, heart failure, cardiovascular disease) and interactions with different patterns of medication use (e.g., diuretics, antihypertensives).

Rationale: If there is systematic error in sodium assessment because individuals with various co-morbidities who are taking medications systematically do not provide accurate urine collections, paradoxical findings between sodium and health outcomes may be observed.

3. Examine the effect of behavioral interventions, with novel approaches (e.g., flavorful recipes, cooking techniques) on adherence to dietary sodium recommendations.

   Rationale: For decades, the population has exceeded dietary sodium intake recommendations. A public health approach that results in reformulation of commercially processed foods to lower sodium content should be the primary strategy for decreasing sodium intake in the U.S. population. However, individual support for public health policies will be needed to further document demand for changes in the sodium food environment. To this end, interventions that modify individual knowledge, attitudes, and behaviors around sodium intake should be evaluated.

4. Examine the effect of low sodium intake on taste preferences for sodium and healthy dietary patterns.

   Rationale: It has been argued that populations desire higher levels of sodium intake and will inevitably revert to higher levels of sodium intakes after acute reductions in sodium intake. It has also been argued that after six weeks of reduced sodium intake, taste preferences are modified such that higher sodium is no longer desirable. Studies are needed to elucidate the effects of lowering sodium intake on diet preferences.

5. Document the relationship between portion size and sodium intake.

   Rationale: These data are needed to inform whether dietary recommendations for sodium should be adjusted for caloric intake. It is known that the absolute amount of sodium intake is highly correlated with caloric intake. As a result, the absolute recommended amount of sodium is harder to achieve for a larger, high energy consuming person than for a smaller, low energy consuming person. The science to inform whether sodium density confers different risk than absolute intake of sodium is limited because of methodologic limitations in surveys where both calories and sodium intake can be calculated. Furthermore, the existing correlation between sodium and calories may be an artifact of the current food supply.

6. Determine the effects of replacement of saturated fat with different types of carbohydrates (e.g., refined vs. whole grains) on cardiovascular disease risk.

   Rationale: Most randomized controlled trials and prospective cohort studies compared saturated fat with total carbohydrates. It is important to distinguish different types of carbohydrates (e.g. refined vs. whole grains) in future studies.

7. Examine the effects replacement of saturated fat with polyunsaturated fat vs. monounsaturated fat on cardiovascular disease risk.

   Rationale: Most existing studies have examined the effects of substituting PUFA for saturated fat on cardiovascular disease risk. Future studies should also examine the potential benefits of substituting monounsaturated from plant sources such as olive oil and nuts/seeds for saturated fat on cardiovascular disease risk.

8. Examine lipid and metabolic effects of specific oils modified to have different fatty acid profiles (e.g. commodity soy oil (high linoleic acid) vs. high oleic soy oil).

   Rationale: As more modified vegetables oils become commercially available, it is important to assess their long-term health effects. In addition, future studies should examine lipid and metabolic effects of plant oils that contain a mix of *n*-9, *n*-6, and **n**-3 fatty acids, as a replacement for animal fat, on cardiovascular disease risk factors.

9. Examine the effects of saturated fat from different sources, including animal products (e.g. butter, lard), plant (e.g., palm vs. coconut oils), and production systems (e.g. refined deodorized bleached vs. virgin coconut oil) on blood lipids and cardiovascular disease risk.

   Rationale: Different sources of saturated fat contain different fatty acid profiles and thus, may result in different lipid and metabolic effects. In addition, virgin and refined coconut oils have different effects in animal models, but human data are lacking.

10. Conduct gene-nutrient interaction studies by measuring genetic variations in relevant genes that will enable evaluation of effects of specific diets for individualized nutrition recommendations.

    Rationale: Individuals with different genetic background may respond to the same dietary intervention differently in terms of blood lipids and other cardiovascular disease risk factors. Future studies should explore the potential role of genetic factors in modulating the effects of fat type modification on health outcomes.

11. Identify sources and names of added sugars and low-calorie sweeteners used in the food supply and quantify their consumption levels and trends in the U.S. diet.

    Rationale: It is unclear whether all food and nutrient databases capture all added sugars because: 1) added sugars have varied and inconsistent nomenclature and may not be recognized as added sugars in nutrient analyses; and 2) many foods with added sugars have formulations considered proprietary by the manufacturers and for this reason actual added sugars content is difficult to obtain. Accurate assessment of added sugars in the U.S. diet is needed to quantify the population level exposure and subsequent health risks from added sugars. The lack of information on the various added sugars in the food supply hinders efforts to make policy about consumption.

12. Conduct prospective research with strong experimental designs and multiple measurements of the consumption of added sugars and low-calorie sweeteners on health outcomes, such as body weight, adiposity, and clinical markers of type 2 diabetes and cardiovascular disease.

    Rationale: High heterogeneity exists among published research with regard to the types and forms of added sugars and low-calorie sweeteners-containing foods/beverages used for interventions, which precludes assessing the effects of specific added sugars and low-calorie sweeteners on body weight, adiposity, and cardio-metabolic health in adults and children. Many studies use single baseline measurements of diet to reflect usual patterns and quantities of intake over time. New research should emphasize assessments within the context of usual dietary intakes and patterns of food and beverage consumption in free-living populations, along with specific added sugars and low-calorie sweeteners, especially those that are currently understudied. Large prospective studies with repeated measurements of low-calorie sweeteners are needed to monitor their long-term effects on cancer and other health outcomes.

13. Design studies that emphasize assessments of relationships between the intakes of added sugars and low-calorie sweeteners and body weight, adiposity, and cardio-metabolic health in diverse sub-populations who are at high risk of obesity and related morbidities.

    Rationale: Insufficient evidence exists to assess the impact of added sugars and low-calorie sweeteners contained in foods and beverages on individuals from diverse populations who have high risk for adverse health outcomes. These include (but are not limited to) different race/ethnicity groups; low income groups, especially those with food insecurity; groups who live in specific geographic locations with high prevalence of obesity (e.g. inner city, rural, and Southern regions of the United States); and age and sex groups (women, children, and elderly adults).

14. Assess and improve approaches and policies to reduce the amount of added sugars in the food and beverage supply as well as in school and community settings.

    Rationale: Results from this research would assist policy makers and the private sector in establishing sustainable approaches and policies to limit the availability and consumption of added sugars. These approaches and policies would also be important for multi-component strategies to improve weight control and health among people living in the United States.

15. Conduct consumer research to identify and test elements of a standardized, easily understood front-of-package label.

    Rationale: Research is needed to provide an evidence base to support the need and identify critical elements of a front of package label. This is particularly important to support the Food and Drug Administration in implementing a front-of-package labeling system.

CHAPTER 7: PHYSICAL ACTIVITY

1. Evaluate best practices in programming at the community and national level and identify which local and national policies in the public and private sector have demonstrated the greatest effect on increasing physical activity participation across the lifespan, especially in populations with the greatest health disparities.

   Rationale: Physical activity participation rates are exceptionally low across all age groups, and are especially low in individuals with the greatest health disparities. Many different initiatives are currently underway in the private and public sector to help increase physical activity on a population level. Understanding which programs and policies are having the greatest impact will help focus valuable resources and national recommendations for maximum public health benefit.

2. Identify the dose of physical activity needed to achieve health benefits, as well as appropriate growth and development, for children younger than age 6 years.

   Rationale: Until recently, very little effort has been focused on understanding the health benefits of physical activity for young children. Given that this is a critical age of growth and development, considerable research should be focused on this age group.

3. Evaluate the effects of various modes and doses of physical activity on health outcomes in older adults.

   Rationale: Older adults are the fastest growing segment of the population. They also have the greatest burden of disease and functional (mental and physical) limitations. To reduce burden of disease and related economic impacts, research regarding mode and dose of physical activity should be focused on this age group.

4. Further evaluate the importance of light activity, short bouts of physical activity (i.e., 10-minutes or less) and modes of activity on health outcomes across the lifespan.

   Rationale: The review of the evidence in the 2008 PAGAC Report focused primarily on moderate- and vigorous-intensity activity. Emerging research highlights the positive effects of light activity as well as shorter bouts of vigorous activity on health outcomes. Understanding the health impact of the full range of mode, intensity, duration, frequency, and setting will help to further refine the PAG to support maximum public health benefit.

5. Further investigate the effects of sedentary behaviors on health outcomes, including duration, frequency, and mode of sedentary activities.

   Rationale: Increasing evidence demonstrates the negative health consequences of sedentary behaviors. Clarity on the types and duration of sedentary behaviors that have the most negative health impact would help to identify meaningful evidence-based public health recommendations.

Appendix E-2: Supplementary Documentation to the 2015 DGAC Report

The 2015 DGAC used a variety of scientifically rigorous approaches to address its science-based questions. These approaches are described in ***Part C. Methodology***. Slightly more than one-third of the questions were answered using a state-of-the-art systematic review process, and these reviews are publically available in the Nutrition Evidence Library (NEL) at www.NEL.gov.

The remaining questions were answered using existing sources of evidence (including systematic reviews, meta-analyses, or reports), data analyses, and food pattern modeling analyses. These three approaches allowed the Committee to ask and answer its questions in a systematic, transparent, and evidence-based way.

Appendix E-2 provides a list of supplementary documentation related to the existing sources of evidence and data analyses used by the Committee in evidence reviews (see ***Appendix E-3*** for ***USDA Food Patterns for Special Analyses***). These sources are publically available online through active links within this document at www.DietaryGuidelines.gov.

CHAPTER 1: FOOD AND NUTRIENT INTAKES AND HEALTH: CURRENT STATUS AND TRENDS

NUTRIENTS OF CONCERN

Appendix E-2.1	Usual intake distributions, 2007-2010, by age/gender groups
Appendix E-2.2	Usual intake distributions as a percent of energy for fatty acids and macronutrients, 2007-2010, by age/gender groups
Appendix E-2.3	Usual intake distributions for individuals age 71 and older, 2007-2010
Appendix E-2.4	Usual intake distributions, 2007-2010, for pregnant and non-pregnant women in the U.S. ages 19-50 years
Appendix E-2.5	Usual intake distributions for supplement users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/gender groups
Appendix E-2.6	Usual intake distributions for non-supplement users for folate, folic acid, vitamin D, calcium, and iron, 2007-2010, by age/gender groups

FOOD CATEGORIES

Appendix E-2.7	Major categories and subcategories used in DGAC analyses of WWEIA Food Categories
Appendix E-2.8	Percent of total food group intake, 2009-2010, for U.S. population ages 2 years and

EATING BEHAVIORS

HEALTH CONDITIONS

Appendix E-2.24 Prevalence of high and borderline high blood pressure (BP), children and adolescents ages 8-17 years, NHANES 2009-2012

DIETARY PATTERNS

Appendix E-2.25 Average Healthy Eating Index-2010 scores for Americans ages 2 years and older (National Health and Nutrition Examination Survey 2009-2010)

CHAPTER 2: DIETARY PATTERNS, FOODS AND NUTRIENTS, AND HEALTH OUTCOMES

DIETARY PATTERNS AND RISK OF CARDIOVASCULAR DISEASE

Appendix E-2.26 Evidence Portfolio

DIETARY PATTERNS AND MEASURES OF BODY WEIGHT

Appendix E-2.27 Evidence Portfolio

DIETARY PATTERNS AND RISK OF TYPE 2 DIABETES

Appendix E-2.28 Evidence Portfolio

CHAPTER 4: FOOD ENVIRONMENT AND SETTINGS

SCHOOL-BASED APPROACHES AND DIETARY INTAKE

Appendix E-2.29a Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

SCHOOL-BASED POLICIES AND DIETARY INTAKE

Appendix E-2.30 Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

SCHOOL-BASED APPROACHES AND WEIGHT STATUS

Appendix E-2.31 Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

SCHOOL-BASED POLICIES AND WEIGHT STATUS

Appendix E-2.32 Evidence Portfolio

Appendix E-2.29b Search and Sort Plan

WORKSITE-BASED APPROACHES AND DIETARY INTAKE

Appendix E-2.33a Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

WORKSITE-BASED POLICIES AND DIETARY INTAKE

Appendix E-2.34 Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

WORKSITE-BASED APPROACHES AND WEIGHT STATUS

Appendix E-2.35 Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

WORKSITE-BASED POLICIES AND WEIGHT STATUS

Appendix E-2.36 Evidence Portfolio

Appendix E-2.33b Search and Sort Plan

CHAPTER 5: FOOD SUSTAINABILITY AND SAFETY

DIETARY PATTERNS AND FOOD SUSTAINABILITY

Appendix E-2.37 Evidence Portfolio

SEAFOOD AND SUSTAINABILITY

Appendix E-2.38 Evidence Portfolio

USUAL CAFFEINE CONSUMPTION AND HEALTH

Appendix E-2.39a Evidence Portfolio

Appendix E-2.39b Systematic Review/Meta-Analysis Data Table

HIGH-DOSE CAFFEINE CONSUMPTION AND HEALTH

Appendix E-2.40 Evidence Portfolio

ASPARTAME CONSUMPTION AND HEALTH

Appendix E-2.41 Evidence Portfolio

CHAPTER 6: CROSS-CUTTING TOPICS OF PUBLIC HEALTH IMPORTANCE

SODIUM AND BLOOD PRESSURE IN ADULTS

Appendix E-2.42 Evidence Portfolio

SATURATED FAT AND RISK OF CARDIOVASCULAR DISEASE

Appendix E-2.43 Evidence Portfolio

ADDED SUGARS AND LOW-CALORIE SWEETENERS

Appendix E-2.44 Evidence Portfolio – Added Sugars and Measures of Body Weight

Appendix E-2.45 Evidence Portfolio – Added Sugars and Risk of Type 2 Diabetes

Appendix E-2.46 Evidence Portfolio – Added Sugars and Dental Caries

Appendix E-2.47 Evidence Portfolio – Low-Calorie Sweeteners and Measures of Body Weight

Appendix E-2.48 Evidence Portfolio – Low-Calorie Sweeteners and Risk of Type 2 Diabetes

CHAPTER 7: PHYSICAL ACTIVITY

PHYSICAL ACTIVITY

Appendix E-2.49 Existing Report Data Table

Appendix E-3: USDA Food Patterns for Special Analyses

The 2015 DGAC identified specific questions that they felt could best be addressed through a food pattern modeling approach, using the USDA Food Patterns and the modeling process developed to address similar requests by the 2005 and 2010 DGACs. The approach used for the 2015 DGAC food pattern modeling questions is described in ***Part C*: *Methodology*.**

Seven modeling analyses requested by the Committee were completed by staff working closely with Subcommittee 1 members, and provided as reports for the full Committee to consider. The food pattern modeling analyses conducted for the 2015 DGAC are listed below. Full reports for each analysis are available online through active links within this document at www.DietaryGuidelines.gov.

E-3.1 Adequacy of USDA Food Patterns
How well do updated USDA food intake patterns meet IOM Dietary Reference Intakes and 2010 Dietary Guidelines nutrient recommendations? How do the recommended amounts of food groups compare to current distributions of usual intakes for the American population?

E-3.2 Food Group Contributions to Nutrients in USDA Food Patterns and Current Nutrient Intakes
What is the contribution of whole grain foods and fruits and vegetables to (1) total fiber intake and (2) total nutrient intake in the USDA Food Patterns? What is the contribution of fruits and vegetables to current nutrient intake (focus on nutrients of concern, including fiber)?

E-3.3 Meeting Vitamin D Recommended Intakes in USDA Food Patterns
Can vitamin D EARs and/or RDAs be met with careful food choices following recommended amounts from each food group in the USDA Food Patterns? How restricted would food choices be, and how much of the vitamin D would need to come from fortified food products?

E-3.4 USDA Food Patterns—Adequacy for Young Children
How well do the USDA Food Patterns meet the nutritional needs of children 2 to 5 years of age and how do the recommended amounts compare to their current intakes? Given the relatively small empty calorie limit for this age group, how much flexibility is possible in food choices?

E-3.5 Reducing Saturated Fats in the USDA Food Patterns

What would be the effect on food choices and overall nutrient adequacy of limiting saturated fatty acids to 6 percent of total calories by substituting mono- and polyunsaturated fatty acids?

E-3.6 Dairy Group and Alternatives

What would be the impact on the adequacy of the patterns if (1) no Dairy foods were consumed, (2) if calcium was obtained from nondairy sources (including fortified foods), and (3) if the proportions of milk and yogurt to cheese were modified?

What is the relationship between changes in types of beverages consumed (milk compared with sugar-sweetened beverages) and diet quality?

E-3.7 Developing Vegetarian and Mediterranean-style Food Patterns

Using the Food Pattern Modeling process, can healthy eating patterns for vegetarians and for those who want to follow a Mediterranean-style diet be developed? How do these patterns differ from the USDA Food Patterns previously updated for the 2015 DGAs?

Appendix E-4: NHANES Data Used in DGAC Data Analyses

Most of the DGAC data analyses used the National Health and Nutrition Examination (NHANES) data and its dietary component, What We Eat in America (WWEIA), NHANES (Zipf et al., 2013). These data were used to answer questions about food and nutrient intakes because they provide national and group level estimates of dietary intakes of the U.S. population on a given day as well as usual intake distributions. These data contributed substantially to questions answered using data analyses. This appendix describes the NHANES data in greater detail.

NHANES

NHANES consists of ongoing, comprehensive, cross-sectional, population-based surveys designed to collect data on health, nutritional status, and health behaviors of the non-institutionalized civilian population living in households in the United States. It is conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC). NHANES has had a long history starting in the early 1960s (Zipf et al., 2013); it has been monitoring food and nutrient intake and nutritional status of the U.S. population since 1971, starting with NHANES I. Since then, several cycles of NHANES have been conducted as a series of cross-sectional surveys focusing on different population groups in terms of age and race/ethnicity, or health topics. In 1999, NHANES became a continuous survey, sampling U.S. residents of all ages, with a changing focus on a variety of health and nutrition measurements to meet emerging needs. The goals of the continuous NHANES are to provide prevalence data on selected diseases and risk factors for the U.S. population; to monitor trends in selected diseases, behaviors, and environmental exposures; to explore emerging public health needs; and to maintain a national probability sample of baseline information on health and nutritional status of the U.S. household population (Zipf et al., 2013).

NHANES has a complex, multi-stage, probability sampling design and examines a nationally representative sample of about 5,000 persons each year. In NHANES, certain subgroups have been periodically oversampled. These include low income, older Americans, infants and children, pregnant women and certain race/ethnic groups (e.g., Hispanics, including Mexican Americans, African Americans, and more recently, Asian Americans). The NHANES survey is unique because it combines personal interviews with standardized physical examinations and laboratory tests administered by a specially trained staff that travels with the Mobile Examination Center (MEC) to survey sites selected to represent the U.S. population (Zipf et al., 2013).

In the continuous NHANES, dietary intake is assessed through two 24-hr recalls, administered by trained dietary interviewers using the USDA's Automated Multiple Pass Method (AMPM) (Blanton et al., 2006) through What We Eat in America (WWEIA). The first 24-hr recall (day 1) is collected in-person at the MEC and a subsequent 24-hr recall (day 2) is obtained 7 to 10 days later over the telephone. Information on dietary supplements consumed during the 24-hour recall period is also collected. The strengths of the WWEIA, NHANES dietary data include that because two 24-hour recalls are available in WWEIA, NHANES (from 2003 onwards), usual intake distributions can be estimated based on statistical techniques that reduce the effect of intra-individual variation in food and nutrient intakes in 24-hour recalls (Nusser et al. 1996; Tooze et al. 2006; Dodd et al. 2006).

The WWEIA, NHANES dietary data are one of the few sources that can provide national estimates of total nutrient intake from diet and dietary supplements for the U.S. population. Moreover, dietary intakes can be described by specific socio-demographic groups including race/ethnic groups, income status, and participation in Federal nutrition assistance programs (e.g., Supplemental Nutrition Assistance Program). Dietary data from WWEIA, NHANES can be linked to thorough anthropometric, laboratory, and clinical evaluation data as well health outcomes to examine cross-sectional associations at the national and large subgroup levels. It must be recognized that WWEIA, NHANES dietary data are not designed for individual-level assessment. These data can be useful to inform nutrition policy, but not sufficient by themselves to form policy recommendations.

No single perfect method for assessing dietary intake information is available in surveys (Willett 1998; Gibson 2005; Berdainer et al., 2008) and different methods may be indicated for specific purposes (Willett 1998; Beaton et al., 1983; Berdainer et al., 2008). NCHS has been actively involved in researching and reviewing its data collection methods, including dietary data, over the years internally and in consultation with expert groups (Wright et al., 1994; Briefel & Sempos, 1992). The methods used in NHANES are adapted in light of its large sample size and complex design, cost and feasibility, and respondent burden to ensure a high response rate to derive nationally representative estimates. Some examples of adaptations in methods include the transition to USDA's standardized automated multi-pass method for collection of dietary recalls by trained interviewers that has been evaluated and associated with reduced measurement error (Moshfegh et al., 2008). Other examples include collection of an additional 24-hour dietary recall in NHANES since 2003 (for a total of two 24-hour recalls), coupled with targeted food frequency questionnaires over various NHANES cycles.

The strengths and shortcomings of these dietary assessment methods have been discussed over time in various meetings (e.g., International Conference on Diet and Activity Methods and American Society for Nutrition/Experimental Biology), workshops, and expert groups. This has also been discussed for several years in the scientific literature (Beaton 1994; Berdainer et al.,

2008) and in recent articles (Archer et al., 2013; Hébert et al., 2014; Webb, 2013). No assessment method is perfect and the choice of dietary method is based on the purpose for which it is intended. For NHANES, repeated 24-hour recalls remain the backbone of dietary assessment and monitoring. These data are useful in providing national- and group-level estimates of dietary intakes of the U.S. population, on a given day as well as in describing usual intake distributions using appropriate statistical approaches, to inform nutrition policy.

REFERENCES

Archer E, Hand GA, Blair SN. Validity of U.S. Nutritional Surveillance: National Health and Nutrition Examination Survey Caloric Energy Intake Data, 1971-2010. PloS one 2013;8(10):e76632.

Beaton GH, Milner J, McGuire V, Feather TE, Little JA. Source of variance in 24-hour dietary recall data: implications for nutrition study design and interpretation. Carbohydrate sources, vitamins, and minerals. Am J Clin Nutr. 1983;37(6):986-95.

Beaton GH. Approaches to analysis of dietary data: relationship between planned analyses and choice of methodology. Am J Clin Nutr 1994; 59(1Suppl):253S-61S.

Berdainer CD, Dwyer J, Feldman EB. Handbook of Nutrition and Food. New York, NY: CRC Press, 2008

Blanton CA, Moshfegh AJ, Baer DJ, Kretsch MJ. The USDA Automated Multiple-Pass Method accurately estimates group total energy and nutrient intake. J Nutr 2006;136(10):2594-9.

Briefel RR, Sempos CT, eds. Dietary methodology workshop for the third National Health and Nutrition Examination Survey. National Center for Health Statistics. Vital and Health Stat 4(27) March 1992.

Dodd KW, Guenther PM, Freedman LS, Subar AF, Kipnis V, Midthune D, Tooze JA, Krebs-Smith SM. Statistical methods for estimating usual intake of nutrients and foods: a review of the theory. J Am Diet Assoc. 2006;106:1640-50.

Hébert JR, Hurley TG, Steck SE, Miller DR, Tabung FK, Peterson KE, Kushi LH, Frongillo EA. Considering the value of dietary assessment data in informing nutrition-related health policy. Adv Nutr. 2014;5(4):447-55.

Gibson R. Principles of Nutritional Assessment. 2nd ed. Oxford: Oxford University Press, 2005.

Moshfegh AJ, Rhodes DG, Baer DJ, et al. The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. Am J Clin Nutr 2008;88(2):324-32.

Nusser, S. M., Carriquiry, A. L., Dodd, K. W., and Fuller, W. A. (1996). A semiparametric transformation approach to estimating usual daily intake distributions. J Am Stat Assoc 1996;91:1440-49.

Tooze JA, Midthune D, Dodd KW, Freedman LS, Krebs-Smith SM, Subar AF, Guenther PM, Carroll RJ, Kipnis V. A new statistical method for estimating the usual intake of episodically consumed foods with application to their distribution. J Am Diet Assoc. 2006;106:1575-87.

Webb D, Leahy MM, Milner JA, Allison DB, Dodd KW, Gaine PC, Matthews RA, Schneeman BO, Tucker KL, Young SS. Strategies to optimize the impact of nutritional surveys and epidemiological studies. Adv Nutr. 2013 Sep 1;4(5):545-7.

Willett W. Nutritional Epidemiology. 2nd Edition ed. New York, NY: Oxford University Press, 1998.

Wright JD, Ervin B, Briefel RR, eds. Consensus Workshop on Dietary Assessment: Nutrition Monitoring and Tracking the Year 2000 Objectives. National Center for Health Statistics. December 1994.

Zipf G, Chiaapa M, Porter K, Ostchega Y, Lewis B, Dostal J. The National Health and Nutrition Examination Survey: Plan and Operations, 1999-2010. Vital Health Stat, 1;56: National Center for Health Statistics, 2013

Appendix E-5: Glossary of Terms

Aquaculture—The farming of aquatic organisms, including fish, mollusks, crustaceans, and aquatic plants. Farming includes activities to enhance production, such as regular stocking, feeding, and protection from predators.

Acculturation—The process by which immigrants adopt the attitudes, values, customs, beliefs, and behaviors of a new culture. Acculturation is the gradual exchange between immigrants' original attitudes and behavior and those of the host culture.

Added sugars—Sugars that are either added during the processing of foods, or are packaged as such. They include sugars (free, mono- and disaccharides), syrups, naturally occurring sugars that are isolated from a whole food and concentrated so that sugar is the primary component (e.g., fruit juice concentrates), and other caloric sweeteners. Names for added sugars include: Brown sugar, corn sweetener, corn syrup, dextrose, fructose, fruit juice concentrates, glucose, high-fructose corn syrup, honey, invert sugar, lactose, maltose, malt sugar, molasses, raw sugar, turbinado sugar, trehalose, and sucrose.

Behavioral weight-management program—A structured, multi-component program that encompasses a number of behavior changes, including diet and physical activity with the intent to improve weight (lose weight or maintain weight loss).

Biodiversity—The variety and variability among living organisms and the ecosystems in which they occur. Biodiversity includes the numbers of different items and their relative frequencies; these items are organized at many levels, ranging from complete ecosystems to the biochemical structures that are the molecular basis of heredity. Thus, biodiversity expresses the relative abundance of different ecosystems, species, and genes.

Body mass index (BMI)—A measure defining weight in kilograms (kg) divided by height in meters (m) squared. BMI is an indicator of deficient or excess body tissue, both fat and muscle. BMI status categories include underweight, normal weight, overweight, and obese. (Normal weight is often referred to as "healthy" weight.) Overweight and obese describe ranges of weight that are greater than what is considered healthy for a given height, while underweight describes a weight that is lower than what is considered healthy. Because children and adolescents are growing, their BMI is plotted on growth charts for sex and age. The percentile indicates the relative position of the child's BMI among children of the same sex and age. This is generally referred to as a **BMI z-score**.

	Children and Adolescents (ages 2 to 19 years)	Adults
Underweight	Less than the 5th percentile	Less than 18.5 kg/m^2
Normal weight	5th percentile to less than the 85th percentile	18.5 to 24.9 kg/m^2
Overweight	85th to less than the 95th percentile	25.0 to 29.9 kg/m^2
Obese	Equal to or greater than the 95th percentile	
Obese class I		30.0 to 34.9 kg/m^2
Obese class II		35.0 to 39.9 kg/m^2
Obese class III		40.0 kg/m^2 and greater

Built environment—The physical form of communities, including urban design (i.e., how a city is designed; its physical appearance and arrangement), land use patterns (i.e., how land is used for commercial, residential, and other activities), and the transportation system (i.e., the facilities and services that link one location to another).

Calorie—A unit commonly used to measure energy content or energy use. It is used as a convenient measure to relate the energy content of food to the energy needs of the body. A calorie is equal to the amount of energy required to raise the temperature of one liter of water 1 degree centigrade. Energy, as measured in calories, is required to sustain the body's various functions, including metabolic processes and physical activity. Carbohydrate, fat, protein, and alcohol provide all of the energy supplied by foods and beverages.

Carbohydrates—One of the three classes of macronutrients. Carbohydrates include sugars, starches, and fibers:

- **Sugars—**A simple carbohydrate composed of one unit (a monosaccharide, such as glucose and fructose) or two joined units (a disaccharide, such as lactose and sucrose). Sugars include white and brown sugar, fruit sugar, corn syrup, molasses, and honey. (See **Added sugars**)

- **Starches—**Many glucose units linked together. Examples of foods containing starch include vegetables, dry beans and peas, and grains (e.g., brown rice, oats, wheat, barley, corn).

- **Fiber—**Nondigestible carbohydrates and lignin that are intrinsic and intact in plants. Fiber consists of dietary fiber, the fiber naturally occurring in foods, and functional fiber, which are isolated, nondigestible carbohydrates that have beneficial physiological effects in humans.

Child-care settings—Locations that include child-care centers and child-care provided in homes. Early childhood education settings, such as preschool and Head Start programs, also are included.

Competitive foods—Foods and beverages offered at schools that are sold or offered outside of the Federally reimbursed school lunch and breakfast programs. Competitive foods include food and beverage items sold through à la carte lines, snack bars, student stores, vending machines, and school fundraisers.

Comprehensive lifestyle intervention—Interventions that are designed to address chronic disease risk factors and improve health. They generally include three principal components—a diet component, a physical activity component, and a program of behavior change to facilitate adherence to diet and physical activity recommendations.

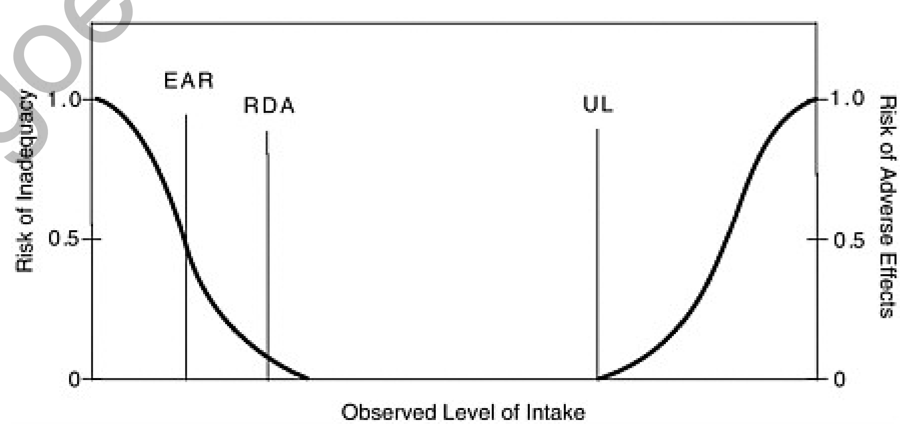
Comprehensive lifestyle intervention team—A multidisciplinary team of highly trained professionals, including registered dietitians and nutritionists, exercise specialists, and behaviorists who work with individuals on weight loss or other lifestyle behavior change to improve health and reduce chronic disease risk. (See **Interventionist**)

Cross-contamination—The spread of bacteria, viruses, or other harmful agents from one surface to another.

Cup equivalent (cup eq)—The amount of a food product that is considered equal to 1 cup from the vegetable, fruit, or milk food group. A cup eq for some foods may differ from a measured cup in volume because (1) the foods have been concentrated (such as raisins or tomato paste), (2) the foods are airy in their raw form and do not compress well into a cup (such as salad greens), or (3) the foods are measured in a different form (such as cheese).

Dietary pattern—The quantities, proportions, variety or combinations of different food and beverages in diets, and the frequency with which they are habitually consumed.

Dietary Reference Intakes (DRIs)—A set of nutrient-based reference values that expand upon and replace the former Recommended Dietary Allowances (RDAs) in the United States and the Recommended Nutrient Intakes (RNIs) in Canada. They include the values shown in the graphic (http://www.dsld.nlm.nih.gov/dsld/dri.jsp) and described here:



- **Acceptable Macronutrient Distribution Ranges (AMDR)**—Range of intake for a particular energy source that is associated with reduced risk of chronic disease while providing intakes of essential nutrients. If an individual's intake is outside of the AMDR, there is a potential of increasing the risk of chronic diseases and/or insufficient intakes of essential nutrients.
- **Adequate Intakes (AI)—**A recommended average daily nutrient intake level based on observed or experimentally determined approximations or estimates of mean nutrient intake by a group (or groups) of apparently healthy people. This is used when the Recommended Dietary Allowance cannot be determined.
- **Estimated Average Requirements (EAR)—**The average daily nutrient intake level estimated to meet the requirement of half the healthy individuals in a particular life stage and sex group.
- **Recommended Dietary Allowance (RDA)—**The average dietary intake level that is sufficient to meet the nutrient requirement of nearly all (97 to 98 percent) healthy individuals in a particular life stage and sex group.
- **Tolerable Upper Intake Level (UL)—**The highest average daily nutrient intake level likely to pose no risk of adverse health effects for nearly all individuals in a particular life stage and gender group. As intake increases above the UL, the potential risk of adverse health effects increases.

Eating out—A behavior that includes meals eaten outside of the home at a variety of venues and takeout or ready-to-eat meals purchased and consumed either away from or in the home.

Empty calories—The calories from components of a food or beverage that contribute few or no nutrients. Major sources of empty calories are solid fats and added sugars. Other sources of empty calories include refined starches (e.g., corn starch, potato starch) and alcohol. In some foods, such as soda and many candies, all the calories are empty calories. However, empty calories also can be found in foods that contain important nutrients. For example, whole milk contains solid fats (butterfat) and sweetened applesauce contains added sugars, which means that some of their calories are empty calories.

Energy drink—A beverage that contains caffeine as a major active ingredient, along with other ingredients, such as taurine, herbal supplements, vitamins, and sugar. It is usually marketed as a product that can improve perceived energy, stamina, athletic performance, or concentration.

Enrichment—The addition of specific nutrients (iron, thiamin, riboflavin, and niacin) to refined grain products in order to replace losses of the nutrients that occur during processing.

Environmental sustainability—Long-term maintenance of ecosystem components and functions for future generations.

Existing reports—Previously published reports or articles that were used as sources of evidence to answer some questions posed by the 2015 DGAC. These sources included reports (e.g., *the 2013 American College of Cardiology/ American Heart Association (ACC/AHA) Guidelines on Lifestyle Management to Reduce Cardiovascular Risk*), systematic reviews, and meta-analyses. (See **Meta-analysis**)

Fast food—Foods designed for ready availability, use or consumption and sold at eating establishments for quick availability or take-out. Fast food restaurants are also known as quick-service restaurants.

Fats—One of the three classes of macronutrients. (See **Solid Fats** and **Oils**)

- **Monounsaturated Fatty Acids—**Monounsaturated fatty acids (MUFAs) have one double bond. Plant sources that are rich in MUFAs include nuts and vegetable oils that are liquid at room temperature (e.g., canola oil, olive oil, high oleic safflower and sunflower oils).

- **Polyunsaturated fatty acids—**Polyunsaturated fatty acids (PUFAs) have two or more double bonds and may be of two types, based on the position of the first double bond.

    - ***n*-6 PUFAs—**Linoleic acid, one of the n-6 fatty acids, is required because it cannot be synthesized by humans and, therefore, is considered essential in the diet. Primary sources are nuts and liquid vegetable oils, including soybean oil, corn oil, and safflower oil. Also called omega-6 fatty acids.

    - ***n*-3 PUFAs—**Alpha-linolenic acid is an *n*-3 fatty acid that is required because it cannot be synthesized by humans and, therefore, is considered essential in the diet. Primary sources include soybean oil, canola oil, walnuts, and flaxseed. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are very long chain *n*-3 fatty acids that are contained in fish and shellfish. Also called omega-3 fatty acids.

- **Saturated fatty acids—**Saturated fatty acids have no double bonds. Major sources include animal products such as meat and dairy products, and tropical oils such as coconut or palm oils. In general, fats high in saturated fatty acids are solid at room temperature.

- ***trans* fatty acids—***Trans* fatty acids are unsaturated fatty acids that contain one or more isolated (i.e., nonconjugated) double bonds in a *trans* configuration. Sources of *trans* fatty acids include partially-hydrogenated vegetable oils that have been used to make traditional shortening and some commercially prepared baked goods, snack foods, fried foods, and traditional stick margarine. *Trans* fatty acids also are present in foods that come from ruminant animals (e.g., cattle and sheep) and are called "natural" or rTFA. Such foods include dairy products, beef, and lamb.

Fight Bac!®—A national public education campaign to promote food safety to consumers and educate them on how to handle and prepare food safely. In this campaign, pathogens are represented by a cartoonlike bacteria character named “BAC.” For more information, visit: http://www.fightbac.org.

Fishery—An activity leading to harvesting of fish. It may involve capture of wild fish or the raising of fish through aquaculture.

Food access—Accessibility to sources of healthy food, as measured by distance to a store or the number of stores in an area; individual-level resources such as family-income or vehicle availability; and neighborhood-level indicators of resources, such as average income of the neighborhood and the availability of public transportation.

Food categories—A method of grouping similar foods in their as-consumed forms, for descriptive purposes. The USDA/ARS has created 150 mutually exclusive food categories to account for each food or beverage item reported in What We Eat in America (WWEIA), the food intake survey component of the National Health and Nutrition Examination Survey (for more information, visit: http://seprl.ars.usda.gov/Services/docs.htm?docid=23429). Examples of WWEIA Food Categories include soups, nachos, and yeast breads. In contrast to food groups, items are not disaggregated into their component parts for assignment to food categories. For example, all pizzas are put into the pizza category.

Food environments—Factors and conditions that influence food choices and food availability. These environments include settings such as home, child care (early care and education), school, after-school programs, worksites, food retail stores and restaurants, and other outlets where children and their families make eating and drinking decisions. The food environment also includes macro-level factors and includes food marketing, food production and distribution systems, agricultural policies, Federal nutrition assistance programs, and economic price structures.

Food groups—A method of grouping similar foods for descriptive and guidance purposes. Food groups in the USDA Food Pattern are defined as fruits, vegetables, grains, dairy, and protein foods. Some of these groups are divided into subgroups, such as dark-green vegetables or whole grains, which may have intake goals or limits (for more information, see ***Appendix E3.1 Table A1.*** USDA Healthy U.S.-Style Food Patterns—Intake Amounts). For assignment to food groups, mixed dishes are disaggregated into their major component parts. For example, pizza may be disaggregated into the grain (crust), dairy (cheese), vegetable (sauce and toppings), and protein foods (toppings) food groups.

Food pattern modeling—The process of developing and adjusting daily intake amounts from food categories or groups to meet specific criteria, such as meeting nutrient intake goals, limiting nutrients or other food components, or varying proportions or amounts of specific food categories or groups.

Food policies—Regulations, laws, policy-making actions or formal or informal rules established by formal organizations or government units. Food and nutrition policies are those that influence the food environment and eating behavior to improve eating and body weight.

Food security—A condition in which all people, now and in the future, have access to sufficient, safe, and nutritious food to maintain a healthy and active life. (See **Household food insecurity**)

Fortification—The addition of one or more essential nutrients to a food whether or not it is normally contained in the food for the purpose of preventing or correcting a demonstrated deficiency of one or more nutrients in the population or specific population groups.

Greenhouse gases (GHG)—Any gas that absorbs infrared radiation in the atmosphere. Greenhouse gases include carbon dioxide, methane, nitrous oxide, ozone, chlorofluorocarbons, hydrochlorofluorocarbons, hydrofluorocarbons, perfluorocarbons, and sulfur hexafluoride.

Health—A state of complete physical, mental and social well-being and not merely the absence of disease or infirmity.

Household food insecurity—Circumstances in which the availability of nutritionally adequate and safe food, or the ability to acquire acceptable foods in socially acceptable ways, is limited or uncertain.

- **Persistent household food insecurity**—Occurs when people are unable to meet their minimum food requirements over a sustained period of time.
- **Progressing household food insecurity—**A change in situation from food secure to food insecure or from acute or temporary food insecurity to persistent food insecurity.
- **Household food insufficiency**—A similar measure to food insecurity considered more severe than the concept of food security, although not as severe as hunger.

Interventionist—Trained health professionals (e.g., registered dietitians, psychologists, exercise physiologists, health counselors, or professionals in training) who adhere to formal protocols in providing healthy lifestyles counseling and treatment, such as for weight management. In a few cases, lay persons are used as trained interventionists; they received instruction in protocols (designed by health professionals) for programs that have been validated in high-quality trials and published in peer-reviewed journals.

Isocaloric—Having the same caloric values. For example, two dietary patterns that vary in macronutrient proportions but have the same calorie content are isocaloric.

Lean meat—Any meat with less than 10% fat by weight, or less than 10 grams of fat per 100 grams, based on USDA and FDA definitions for food label use. Examples include 95% lean ground beef, cooked; broiled beef steak, lean only eaten; baked pork chop, lean only eaten; roasted chicken breast or leg, no skin eaten; and smoked/cured ham, lean only eaten.

Life Cycle Assessment (LCA)—A technique for assessing the biophysical environmental aspects and potential impacts associated with a product, by:

- Compiling an inventory of relevant inputs and outputs of a product system;
- Evaluating the potential environmental impacts associated with those inputs and outputs;
- Interpreting the results of the inventory analysis and impace assessment phases in relation to the objectives of the study.

LCA studies the environmental aspects and potential impacts throughout a product's life (i.e., cradle to grave), from raw material acquisition through production, use, and disposal. The general categories of environmental impacts needing consideration include resource use, human health, and ecological consequences.

Macronutrient—A dietary component that provides energy. Macronutrients include protein, fats, and carbohydrates. Alcohol also provides energy but, for purposes of the DGAC report, it is not considered when discussing macronutrients.

Meta-analysis—The statistical analysis of multiple individual studies for the purpose of integrating the findings and deriving conclusions from the body of literature.

Mobile Health (mHealth)—The use of mobile and wireless technologies to support the achievement of health objectives.

Moderate alcohol consumption—Average daily consumption of up to one drink per day for women and up to two drinks per day for men, with no more than three drinks in any single day for women and no more than four drinks in any single day for men. One drink is defined as 12 fl. oz. of regular beer, 5 fl. oz. of wine, or 1.5 fl. oz. of distilled spirits.

Nutrient-dense foods—Foods that are naturally rich in vitamins, minerals, and other substances that may have positive health effects, and are lean or low in solid fats and without added solid fats, sugars, starches, or sodium and that retain naturally-occurring components such as fiber. All vegetables, fruits, whole grains, fish, eggs, and nuts prepared without added solid fats or sugars are considered nutrient-dense, as are lean or low-fat forms of fluid milk, meat, and poultry prepared without added solid fats or sugars. Nutrient-dense foods provide substantial amounts of vitamins and minerals (micronutrients) and relatively few calories compared to forms of the food that have solid fat and/or added sugars.

Nutrition Evidence Library (NEL) systematic review—A process that uses state-of-the-art methodology to search, evaluate, and synthesize food and nutrition-related research. This rigorous, protocol-driven methodology is designed to minimize bias, maximize transparency, and ensure relevant, timely, and high-quality systematic reviews to inform Federal nutrition-related policies, programs, and recommendations. The NEL is a division of the USDA Center for Nutrition Policy and Promotion. For more detailed information, visit: www.nel.gov.

Oils—Fats that are liquid at room temperature. Oils come from many different plants and some fish. Some common oils include canola, corn, olive, peanut, safflower, soybean, and sunflower oils. A number of foods are naturally high in oils, such as: nuts, olives, some fish, and avocados. Foods that are mainly made up of oil include mayonnaise, certain salad dressings, and soft (tub or squeeze) margarine with no *trans* fats. Oils are high in monounsaturated or polyunsaturated fats, and lower in saturated fats than solid fats. A few plant oils, termed tropical oils, including coconut oil, palm oil and palm kernel oil, are high in saturated fats and for nutritional purposes should be considered as solid fats. Partially-hydrogenated oils that contain *trans* fats should also be considered as solid fats for nutritional purposes. (See **Fats**)

Ounce equivalent (oz eq)—The amount of a food product that is considered equal to one ounce from the grain or protein foods food group. An oz eq for some foods may be less than a measured ounce in weight if the food is concentrated or low in water content (nuts, peanut butter, dried meats, flour) or more than a measured ounce in weight if the food contains a large amount of water (tofu, cooked beans, cooked rice or pasta).

Persistent organic pollutants (POPs)—Toxic chemicals that can adversely affect human health and the biophysical environment. Because they can be transported by wind and water, most POPs generated in one country may affect people and wildlife distant to where they are used and released. They can persist for long periods of and can accumulate and pass from one species to the next through the food chain.

Plant-based foods—Foods such as vegetables, fruits, whole grains, nuts and seeds.

Point-of-purchase—A place where sales are made. Various intervention strategies have been proposed to affect individuals' purchasing decisions at the point of purchase, such as board or menu labeling with various amounts of nutrition information or shelf tags in grocery stores.

Portion size—The amount of a food served or consumed in one eating occasion. A portion is not a standardized amount, and the amount considered to be a portion is subjective and varies.

Processed meat—Meat, poultry, or seafood products preserved by smoking, curing or salting, or addition of chemical preservatives. Processed meat includes bacon, sausage, hot dogs, sandwich meat, packaged ham, pepperoni, and salami.

Protein—One of the three macronutrients classes. Protein is the major functional and structural component of every animal cell. Proteins are composed of amino acids, nine of which are indispensable, meaning they cannot be synthesized by humans and therefore must be obtained from the diet. The quality of dietary protein is determined by its amino acid profile relative to human requirements as determined by the body's requirements for growth, maintenance, and repair. Protein quality is determined by two factors: digestibility and amino acid composition.

- **Animal protein—**Protein from meat, poultry, seafood, eggs, and milk and milk products.
- **Vegetable protein—**Protein from plants such as dry beans, whole grains, fruit, nuts, and seeds.

Refined grains—Grains and grain products missing the bran, germ, and/or endosperm; any grain product that is not a whole grain. Many refined grains are low in fiber but enriched with thiamin, riboflavin, niacin, and iron, and fortified with folic acid.

Screen time—Time in front of a computer, television, video or computer game system, or smart phone or tablet or related device.

Seafood—Marine animals that live in the sea and in freshwater lakes and rivers. Seafood includes fish, such as salmon, tuna, trout, and tilapia, and shellfish, such as shrimp, crab, and oysters.

Sedentary behavior—Any waking activity predominantly done while in a sitting or reclining posture. A behavior that expends energy at or minimally above a person's resting level (between 1.0 and 1.5 metabolic equivalents), is considered sedentary behavior.

Self-monitoring—Self-monitoring refers to the process by which an individual observes and records specific information about his or her behaviors. For example, in weight management self-monitoring, observations and records would reflect dietary intake, physical activity, and/or body weight.

Solid fats—Fats that are usually not liquid at room temperature. Solid fats are found in animal foods except for seafood, and can be made from vegetable oils through hydrogenation. Some tropical oil plants, such as coconut and palm, are considered as solid fats due to their fatty acid composition. Solid fats contain more saturated fats and/or *trans* fats than liquid oils (e.g., soybean, canola, and corn oils), with lower amounts of monounsaturated or polyunsaturated fatty acids. Common fats considered to be solid fats include: butterfat, beef fat (tallow, suet), chicken fat, pork fat (lard), stick margarine, shortening, coconut oil, palm oil and palm kernel oil. Foods high in solid fats include: butter, full-fat cheeses, creams, whole milk, full fat ice creams, marbled cuts of meats, regular ground beef, bacon, sausages, poultry skin, and many baked goods made using these products (such as cookies, crackers, doughnuts, pastries, and croissants).The fat component of milk and cream (butter) is solid at room temperature. (See **Fats**)

Sugar-sweetened beverages—Liquids that are sweetened with various forms of added sugars (see **Added Sugars** and **Carbohydrates: Sugars**). These beverages include, but are not limited to, soda, fruitades, and sports drinks. Also called calorically-sweetened beverages.

Sustainable diets—A pattern of eating that promotes health and well-being and provides food security for the present population while sustaining human and natural resources for future generations.

Trophic level—A functional classification of species that is based on feeding relationships. Generally, aquatic and terrestrial green plants comprise the first, or lowest, trophic level, herbivores comprise the second, and primary carnivores comprise the third, or highest level. Examples of high trophic fish species are salmon and trout. Low trophic fish species include crayfish and catfish.

Whole grains—Grains and grain products made from the entire grain seed, usually called the kernel, which consists of the bran, germ, and endosperm. If the kernel has been cracked, crushed, or flaked, it must retain the same relative proportions of bran, germ, and endosperm as the original grain in order to be called whole grain. Many, but not all, whole grains are also sources of dietary fiber.

Appendix E-6: History of Dietary Guidance Development in the United States and the Dietary Guidelines for Americans

In early 1977, after years of discussion, scientific review, and debate, the U.S. Senate Select Committee on Nutrition and Human Needs, led by Senator George McGovern, recommended Dietary Goals for the American people (U.S. Senate Select Committee, 1977). The Goals consisted of complementary nutrient-based and food-based recommendations. The first Goal focused on energy balance and recommended that, to avoid overweight, Americans should consume only as much energy as they expended. Overweight Americans should consume less energy and expend more energy. For the nutrient-based Goals, the Senate Committee recommended that Americans:

- Increase consumption of complex carbohydrates and “naturally occurring sugars;” and
- Reduce consumption of refined and processed sugars, total fat, saturated fat, cholesterol, and sodium.
- For the food-based Goals, the Senate Committee recommended that Americans:
- Increase consumption of fruits, vegetables, and whole grains;
- Decrease consumption of:
    - refined and processed sugars and foods high in such sugars;
    - foods high in total fat and animal fat, and partially replace saturated fats with polyunsaturated fats;
    - eggs, butterfat, and other high-cholesterol foods;
    - salt and foods high in salt; and
- Choose low-fat and non-fat dairy products instead of high-fat dairy products (except for young children).

The Dietary Goals was met with considerable debate and controversy, as industry groups and the scientific community expressed doubt that the science available at the time supported the specificity of the numbers provided in the Dietary Goals. To support the credibility of the science used by the Senate Committee, the U.S. Department of Agriculture and U.S. Department of Health and Human Services (then called the Department of Health, Education, and Welfare) selected scientists from the two Departments and obtained additional expertise from the scientific community throughout the country to address the public’s need for authoritative and consistent guidance on diet and health.

In February 1980, the two Departments collaboratively issued *Nutrition and Your Health: Dietary Guidelines for Americans*, a brochure that, in describing seven principles for a healthful diet, provided assistance for healthy people in making daily food choices (USDA/HHS, 1980). These Guidelines were based, in part, on the 1979 *Surgeon General's Report on Health Promotion and Disease Prevention* (DHEW/PHS, 1979) and reflected findings from a study on the relationship between dietary practices and health outcomes (ASCN, 1979). Ideas for incorporating a variety of foods to provide essential nutrients while maintaining recommended body weight were a focus. The brochure also provided guidance on limiting dietary components such as fat, saturated fat, cholesterol, and sodium, which were beginning to be considered risk factors in certain chronic diseases. Both the Dietary Goals and the first Dietary Guidelines for Americans were different from previous dietary guidance in that they reflected emerging scientific evidence and changed the historical focus on nutrient adequacy to also identify the impacts of diet on chronic disease. These documents discussed the concepts of moderation as well as nutrient adequacy.

Even though the recommendations of the 1980 *Dietary Guidelines for Americans* were presented as innocuous and straightforward extrapolations from the science base, they, too, were met with controversy from a variety of industry and scientific groups.

The debate about the 1980 *Dietary Guidelines for Americans* led to Congressional report language that directed the two Departments to convene an advisory committee that would ensure that outside advice, both formal and informal, was captured in developing future editions of the Dietary Guidelines. A Dietary Guidelines Advisory Committee composed of scientific experts outside the Federal sector was established shortly after that directive and was very helpful in the development of the 1985 *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1985). The Departments made relatively few changes from the first edition, but this second edition was issued with much less debate from either industry or the scientific community. The 1985 Dietary Guidelines were widely accepted and were used as the framework for consumer nutrition education messages. They also were used as a guide for healthy diets by scientific, consumer, and industry groups.

In 1989, USDA and HHS established a second scientific advisory committee to review the 1985 Dietary Guidelines and make recommendations for revision. The basic tenets of earlier Dietary Guidelines were reaffirmed, and the 1990 *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1990) promoted enjoyable and healthful eating through variety and moderation, rather than dietary restriction. For the first time, the Guidelines also suggested quantitative goals for total fat and saturated fat, though they stressed that the goals were to be met through dietary choices made over several days, not through choices about one meal or one food.

The 1980, 1985, and 1990 editions of the Dietary Guidelines were issued voluntarily by the two Departments. With the passage of the 1990 National Nutrition Monitoring and Related Research

Act (Section 301 of Public Law 101-445, 7 USC 5341, Title III) (US Congress, 1990), the 1995 edition of *Nutrition and Your Health: Dietary Guidelines for Americans* became the first Dietary Guidelines policy document mandated by statute. This Act directed the Secretaries of USDA and HHS to jointly issue at least every 5 years a report entitled *Dietary Guidelines for Americans*.

A Dietary Guidelines Advisory Committee was established to prepare technical reports that advised the Federal government on the status of the evidence on nutrition and health. These technical reports were used in developing the 1995, 2000, 2005, and 2010 versions of the *Dietary Guidelines for Americans* (HHS/USDA, 1995a; HHS/USDA, 1995b; USDA/HHS, 2000a; USDA/HHS, 2000b; HHS/USDA, 2004; HHS/USDA, 2005a; USDA/HHS, 2010; USDA/HHS, 2011). This report of the 2015 Dietary Guidelines Advisory Committee will serve a similar purpose for HHS and USDA as the Departments develop the 2015 edition of *Dietary Guidelines for Americans*.

Since 1980, the Dietary Guidelines have been notably consistent in their recommendations on the components of a healthful diet, but they also have changed in some significant ways to reflect emerging science as well as public health concerns, such as the increasing prevalence of major chronic diseases among the majority of the general population. In keeping with growing emphasis on data quality in developing recommendations, the 2005 Committee used a modified systematic approach for reviewing the scientific literature. This systematic review of the evidence was further realized for the 2010 Dietary Guidelines Advisory Committee with the establishment of the USDA's Nutrition Evidence Library, a process that uses state-of-the-art methodology to search, evaluate, and synthesize food and nutrition-related research. This rigorous, protocol-driven methodology is designed to minimize bias, maximize transparency, and ensure relevant, timely, and high-quality systematic reviews to inform Federal nutrition-related policies, programs, and recommendations. (See ***PART C: Methodology*** for a brief description of the systematic evidence review process used by the 2015 Dietary Guidelines Advisory Committee and www.NEL.gov for additional information about the Nutrition Evidence Library.)

Over the past two decades, *Nutrition and Your Health: Dietary Guidelines for Americans* has evolved to become a broadly accepted, science-based document that serves as the Federal nutrition policy on which nutrition standards, nutrition programs, and nutrition education are based. The Dietary Guidelines have proven to be a mechanism for addressing public health concerns by providing focused guidance that can help to promote health and reduce chronic disease risk. As such, while earlier editions of the Dietary Guidelines focused specifically on healthy Americans ages 2 years and older, more recent editions also have included those who are at increased risk of chronic disease. The Dietary Guidelines, however, are not directly intended for disease treatment, but they can be used as a basis for developing clinical guidelines.

Future editions of the Dietary Guidelines will continue to evolve to address public health concerns and the nutrition needs of specific populations. For example, a Federal initiative has been established to develop comprehensive guidance for infants and toddlers from birth to 24 months and women who are pregnant so that by 2020, the Dietary Guidelines will also include these important populations comprehensively. For now, nutrition and health professionals actively promote the Dietary Guidelines as a means of encouraging Americans to focus on eating a healthful diet and being physically active throughout the entire lifespan.

HISTORY OF DIETARY GUIDANCE DEVELOPMENT IN THE UNITED STATES AND THE DIETARY GUIDELINES FOR AMERICANS – A CHRONOLOGY

1977 *Dietary Goals for the United States* (the "McGovern Report") was issued by the U.S. Senate Select Committee on Nutrition and Human Needs (U.S. Senate Select Committee, 1977). The Dietary Goals reflected a shift in focus from obtaining adequate nutrients to avoiding excessive intake of food components linked to chronic disease. These goals were controversial among some nutritionists and others concerned with food, nutrition, and health.

1979 The American Society for Clinical Nutrition formed a panel to study the relationship between dietary practices and health outcomes (ASCN, 1979). The findings, presented in 1979, were reflected in *Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention* (DHEW/PHS, 1979).

1980 Seven principles for a healthful diet were jointly issued by the then U.S. Department of Health, Education, and Welfare (now HHS) and the U.S. Department of Agriculture (USDA) in response to the public's desire for authoritative, consistent guidelines on diet and health. These principles became the first edition of *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1980). The 1980 Guidelines were based on the most up-to-date information available at the time and were directed to healthy Americans ages 2 and older. The Guidelines generated some concern among consumer, commodity, and food industry groups, as well as some nutrition scientists, who questioned the causal relationship between certain guidelines and health.

1980 A U.S. Senate Committee on Appropriations report directed that an external advisory committee be established to review scientific evidence and recommend revisions to the 1980 *Nutrition and Your Health: Dietary Guidelines for Americans* (U.S. Senate, 1980).

1983 An external Federal advisory committee of nine nutrition scientists was convened to review and make recommendations in a report to the Secretaries of USDA and HHS about the first (1980) edition of the Dietary Guidelines (USDA/HHS, 1985a).

1985 USDA and HHS jointly issued the second edition of *Nutrition and Your Health: Dietary Guidelines for Americans* (USDA/HHS, 1985b). This edition was nearly identical to the first, retaining the seven guidelines from the 1980 edition. Some changes were made for clarity, while others reflected advances in scientific knowledge of the associations between diet and chronic diseases. The second edition received wide acceptance and was used as the basis for dietary guidance for the general public as well as a framework for developing consumer education messages.

1987 Language in the *Conference Report of the House Committee on Appropriations* indicated that USDA, in conjunction with HHS, "shall reestablish a Dietary Guidelines Advisory Group on a periodic basis. This Advisory Group will review the scientific data relevant to nutritional guidance and make recommendations on appropriate changes to the Secretaries of the Departments of Agriculture and Health and Human Services" (U.S. House of Representatives, 1987).

1989 USDA and HHS established a second Federal advisory committee of nine members, which considered whether revisions to the 1985 Dietary Guidelines were needed and made recommendations for revision in a report to the Secretaries (USDA/HHS, 1990a). The 1988 Surgeon General's Report on Nutrition and Health (HHS/PHS, 1988) and the 1989 National Research Council's report *Diet and Health: Implications for Reducing Chronic Disease Risk* were key resources used by the Committee (NAS/NRC, 1989).

1990 USDA and HHS jointly released the third edition of *Nutrition and Your Health*: *Dietary Guidelines for Americans* (USDA/HHS, 1990b). The basic tenets of the 1985 Dietary Guidelines were reaffirmed, with additional refinements made to reflect increased understanding of the science of nutrition and how best to communicate the science to consumers. The language of the new Dietary Guidelines was positive, was oriented toward the total diet, and provided specific information regarding food selection. For the first time, quantitative recommendations were made for intakes of dietary total fat and saturated fat.

1990 The 1990 National Nutrition Monitoring and Related Research Act (Section 301 of Public Law 101-445, 7 USC 5341, Title III) directed the Secretaries of the USDA and HHS to jointly issue at least every 5 years a report entitled *Dietary Guidelines for Americans* (U.S. Congress, 1990). This legislation also required USDA and HHS to review all Federal publications containing dietary advice for the general public.

1993 HHS initiated a charter establishing the 1995 Dietary Guidelines Advisory Committee.

1994 An 11-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of HHS and USDA to review the third edition of the Dietary Guidelines and determine whether changes were needed. If so, the Committee was to recommend suggestions and the rationale for any revisions.

1995 The report of the Dietary Guidelines Advisory Committee to the Secretaries of HHS and USDA was published (HHS/USDA, 1995a).

1995 Using the 1995 report of the Dietary Guidelines Advisory Committee as the foundation, HHS and USDA jointly developed and released the fourth edition of *Nutrition and Your Health*: *Dietary Guidelines for Americans* (HHS/USDA, 1995b). This edition continued to support the concepts from earlier editions. New information included the Food Guide Pyramid, Nutrition Facts label, boxes highlighting good food sources of key nutrients, and a chart illustrating three weight ranges in relation to height.

1997 USDA initiated the charter establishing the 2000 Dietary Guidelines Advisory Committee.

1998 An 11-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of USDA and HHS to review the fourth edition of the Dietary Guidelines to determine whether changes were needed and, if so, to recommend suggestions for revision.

2000 The Committee submitted its report to the Secretaries of USDA and HHS (USDA/HHS, 2000a). This report contained the proposed text for the fifth edition of *Nutrition and Your Health: Dietary Guidelines for Americans*.

2000 The President of the United States spoke of the Dietary Guidelines in his radio address after USDA and HHS jointly issued the fifth edition of *Nutrition and Your Health: Dietary Guidelines for Americans* earlier in the day (USDA/HHS, 2000b). Earlier versions of the Guidelines included seven statements. This version included 10—created by breaking out physical activity from the weight guideline, splitting the grains and fruits/vegetables recommendations for greater emphasis, and adding a new guideline on safe food handling.

2003 HHS initiated the charter establishing the 2005 Dietary Guidelines Advisory Committee.

2003 A 13-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of HHS and USDA to review the fifth edition of the Dietary Guidelines to determine whether changes were needed and, if so, to recommend suggestions for revision.

2003-2004 In keeping with renewed emphasis on data quality, the Committee used a modified "systematic approach" to review the scientific literature and develop its recommendations. Committee members initially posed approximately 40 specific research questions that were answered using an extensive search and review of the scientific literature. Issues relating diet and physical activity to health promotion and chronic disease prevention were included in the Committee's evidence review. Other major sources of evidence used were the Dietary Reference Intake (DRI) reports prepared by expert committees convened by the Institute of Medicine (IOM) as well as various Agency for Healthcare Research and Quality (AHRQ) and World Health Organization (WHO) reports. In addition, USDA completed numerous food intake pattern modeling analyses and the Committee analyzed various national data sets and sought advice from invited experts.

2004 The Committee submitted its technical report to the Secretaries of HHS and USDA (HHS/USDA, 2004). This 364-page report contained a detailed analysis of the science and was accompanied by many pages of evidence-based tables that were made available electronically. After dropping some questions because of incomplete or inconclusive data, the Committee wrote conclusions and comprehensive rationales for 34 of the 40 original questions.

2005 Using the Committee's technical report as a basis, HHS and USDA jointly prepared and issued the sixth edition of *Dietary Guidelines for Americans* in January 2005 (HHS/USDA, 2005a). This 80-page policy document was the first time the Departments prepared a policy document that was intended primarily for use by policy makers, healthcare professionals, nutritionists, and nutrition educators. The content of this document included nine major Dietary Guidelines messages that resulted in 41 Key Recommendations, of which 23 were for the U.S. population overall and 18 for specific population groups. The policy document highlighted the USDA Food Guide and the DASH Eating Plan as two examples of eating patterns that exemplify the Dietary Guidelines recommendations. A companion, 10-page brochure called Finding Your Way to a Healthier You (HHS/USDA, 2005b) was released concurrently with the Dietary Guidelines to provide advice to consumers about food choices that promote health and decrease the risk of chronic disease. Shortly thereafter, USDA released the MyPyramid Food Guidance System, an update of the Food Guide Pyramid, which included more detailed advice for consumers to help them follow the Dietary Guidelines.

2008 USDA initiated the charter establishing the 2010 Dietary Guidelines Advisory Committee.

2008 A 13-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of USDA and HHS to review the sixth edition of *Dietary Guidelines for Americans* to determine whether changes were needed and, if so, to recommend suggestions for revision.

2008-2009 USDA's Center for Nutrition Policy and Promotion established the Nutrition Evidence Library (NEL) to conduct systematic reviews to help inform Federal nutrition policy and programs. The NEL supported the Dietary Guidelines Advisory Committee in answering approximately 130 of the total 180 diet and health-related questions posed. This was the most rigorous and comprehensive approach used to date for reviewing the science in order to develop nutrition-related recommendations for the public. Other sources of evidence for answering scientific questions included modeling analyses of USDA's Food Patterns, review of reports from various data analyses, as well as other available authoritative reports (e.g., 2005 DGAC Report and IOM reports). An elaborate web-based public comments database was developed and provided a successful mechanism for the public to provide comments and thereby participate in the Committee's evidence review process. The database also allowed the public to read other comments that were submitted. This database eventually included more than 800 public comments related to the DGAC process.

2010 The Committee submitted its technical report to the Secretaries of USDA and HHS (USDA/HHS 2010). This 445-page report contained a detailed analysis of the science and was accompanied by additional 230 pages of food pattern modeling appendices made available electronically at www.DietaryGuidelines.gov.

2011 Using the Committee's technical report as the basis, HHS and USDA jointly prepared and published the seventh edition of *Dietary Guidelines for Americans* released publically in January 2011 (USDA/HHS, 2011). The 95-page policy document encompassed the overarching concepts of maintaining calorie balance over time to achieve and sustain a healthy weight, and consuming nutrient-dense foods and beverages. The policy document included 23 key recommendations for the general population and six additional key recommendations for specific populations. To assist individuals to build a healthy diet based on the Dietary Guidelines, the USDA Food Patterns were updated and new vegetarian adaptations were included. The DASH Eating Plan also was included as an example of a healthy dietary pattern. This publication will serve as the basis for Federal nutrition policy until the next policy document is released in 2015. In June, USDA released MyPlate, a new visual icon, and the ChooseMyPlate.gov website that provides tools to help consumers of all ages, educators, and health professionals learn about and follow the Dietary Guidelines.

2013 HHS initiated the charter establishing the 2015 Dietary Guidelines Advisory Committee.

2013 A 15-member Dietary Guidelines Advisory Committee was appointed by the Secretaries of USDA and HHS to review the seventh edition of *Dietary Guidelines for Americans* and recommend suggestions for revision. One member resigned due to professional obligations within the first three months after appointment; 14 members served the remainder of the two-year charter. The Committee also added three consultant subcommittee members during its work to address specific issues; these members participated in discussions and decision at the subcommittee level but were not members of the full Committee.

2015 The Committee submitted this technical report to the Secretaries of USDA and HHS in January 2015. This 580-page report contained a detailed analysis of the science and was accompanied by substantial documentation of the process made available electronically at www.DietaryGuidelines.gov and www.NEL.gov.

REFERENCES

American Society for Clinical Nutrition (ASCN). Symposium. Report of the Task Force on the evidence relating six dietary factors to the nation's health. *Am J Clin Nutr*. 1979;32(Supplement):2621-748.

Murphy S. Development of the MyPyramid Food Guidance System. *J Nutr Educ Behav*. 2006;38(6S):S77-S162.

National Academy of Science, National Research Council (NAS, NRC). *Diet and Health: Implications for Reducing Chronic Disease Risk.* Washington (DC): National Academy Press, 1989.

U.S. Congress. National Nutrition Monitoring and Related Research Act of 1990, Public Law 445, 101st Cong., 2nd Session, Section 301, 7 USC 5341, October 22, 1990.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans.* 1st edition. Washington (DC): USDA/HHS, 1980. Home and Garden Bulletin No. 232.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 1985*. Washington (DC): USDA, Human Nutrition Information Service, 1985a.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans*. 2nd edition. Washington (DC): USDA/HHS, 1985b. Home and Garden Bulletin No. 232.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 1990*. Washington (DC): USDA, Human Nutrition Information Service, 1990a.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans*. 3rd edition. Washington (DC): USDA/HHS, 1990b. Home and Garden Bulletin No. 232.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2000*. Washington (DC): USDA, Agricultural Research Service, 2000a.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Nutrition and Your Health: Dietary Guidelines for Americans*. 5th edition. Washington (DC): USDA/HHS, 2000b. Home and Garden Bulletin No. 232.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010*. Washington (DC): USDA, Agricultural Research Service, 2010.

U.S. Department of Agriculture and U.S. Department of Health and Human Services (USDA/HHS). *Dietary Guidelines for Americans*. 7th edition. Washington (DC): U.S. Government Printing Office, 2011.

U.S. Department of Health and Human Services and U.S. Department of Agriculture (HHS/USDA). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 1995.* Washington (DC): USDA, Agricultural Research Service, 1995a.

U.S. Department of Health and Human Services and U.S. Department of Agriculture (HHS/USDA). *Nutrition and Your Health: Dietary Guidelines for Americans*. 4th edition. Home and Garden Bulletin No. 232. Washington (DC): HHS/USDA, 1995b.

U.S. Department of Health and Human Services and U.S. Department of Agriculture (HHS/USDA). *Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2005*. Washington (DC): USDA, Agricultural Research Service, 2004.

U.S. Department of Health and Human Services and U.S. Department of Agriculture (HHS/USDA). *Dietary Guidelines for Americans*. 6th edition. Washington (DC): U.S. Government Printing Office, 2005a.

U.S. Department of Health and Human Services and U.S. Department of Agriculture (HHS/USDA). *Finding Your Way to a Healthier You*. Washington (DC): U.S. Government Printing Office, 2005b. HHS-ODPHP-205-01-DGA-A; USDA Home and Garden Bulletin No. 232.

U.S. Department of Health and Human Services, Public Health Service (HHS, PHS). *The Surgeon General's Report on Nutrition and Health.* Washington (DC): DHHS, PHS, 1988. Publication No. 88-50215.

U.S. Department of Health, Education, and Welfare, Public Health Service (DHEW, PHS). Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention. Washington (DC): DHEW, PHS, 1979. DHEW Publication No. 79-55071.

U.S. House of Representatives Conference Committee (U.S. House of Representatives). 100th Cong., 1st sess., H. Rep. 498, 1987.

U.S. Senate Agricultural Appropriations Committee (U.S. Senate). 96th Cong., 1st sess., S. Rep. 1030, 1980.

U.S. Senate Select Committee on Nutrition and Human Needs. Dietary Goals for the United States. 2nd edition. Washington (DC): U.S. Government Printing Office, 1977.

Appendix E-7: Public Comments

As a government advisory committee, the Dietary Guidelines Advisory Committee (DGAC) is required by the Federal Advisory Committee Act to function in an open process in which the public may participate. This is accomplished through public submission of written comments and oral testimony given to the DGAC.

Federal Register notices alerted the public to DGAC meetings held in-person and/or by webcast. In these notices the public was invited and reminded to submit their comments to an online database at www.DietaryGuidelines.gov. The public comments process opened on May 29, 2013. Comments continued to be submitted throughout the time the DGAC operated. Following the submission of the 2015 DGAC Report to the Secretaries of HHS and USDA, the Federal government will alert the public of its availability through a *Federal Register* notice. This notice also will announce a public comment period and the date of an in-person meeting where the public can provide comments to the Federal government about the DGAC Report.

A public comments online database was developed for the 2015 DGAC process based on the structure and content used for the 2010 process, but with many enhancements that were intended to streamline submission of comments by the public and processing by staff.

When submitting comments, the public selected one or more topic areas into which they felt their comments belonged. Initially, these topic areas were: Food Groups, Eating Patterns-Diets, Energy Balance, Carbohydrates, Protein, Fats, Micronutrients, Water and Nonalcoholic Beverages, Alcoholic Beverages, Food Safety, Behavior and Food Environment, Lifespan Needs, and Other. During their deliberations and at the DGAC's request, the topic area "Behavior and Food Environment" was split into two distinct topic areas, "Behavior" and "Food Environment," and a new topic area, "Sustainability," also was added. Individual submissions were allowed to include up to five attachments, such as journal articles, reports, and other scientific material for the DGAC to consider. The submission page noted that submitters should take care to not violate copyright laws when submitting attachments.

For the first time, the 2015 DGAC requested public comments related to specific topic areas. Subcommittee (SC) 2 requested comments on "steps the food industry is taking or has taken to reduce the nutrients listed below in the food supply, including what nutrients have been increased as a consequence of reductions where applicable: sodium, added sugars, fats (i.e., total fats, saturated fats, *trans* fats, and other individual fatty acids)." SC 5 requested public comments on "a targeted topic on food system sustainability, including comments from both the private and public sectors and addressing local, regional, national, or international scales. Specifically, it seeks approaches and current examples of sustainability in the food system. Comments are encouraged that address: (a) Elements of a whole food system; (b) Information on specific food groups or commodities; and (c) Sustainability metrics that have been implemented or are in development."

In addition, for the first time, the Committee also provided specific guidance to the public on "length and timing of public comments." This guidance was shared through the *Federal Register* and on www.DietaryGuidelines.gov. This guidance stated to "provide a brief summary (approx. 250 words) of the points or issues in the comment text box." It asked that "if providing literature or other resources, one of the following forms is preferred: complete citation, as in a bibliographic entry; abstract; electronic link to full article or report." The public was encouraged to "provide comments as early as possible in the Committee's process to increase the opportunity for meaningful impact." Lastly, as of April 2014, it stated that "a deadline for comment submission prior to each public meeting will no longer be used."

For all public comments, submitters were required to provide the following information: topic area(s), the comment itself (5,000 character limit), any accompanying attachments, full name (with option to make it public), affiliation, and organization. They also were required to provide their email address, phone number, and zip code, but this information was not included when the comment was posted on the www.DietaryGuidelines.gov public comments page. Submitters were given the option, but not required, to also provide their business or academic credentials and postal address, including country. This information was not posted on the public website. After the comment was submitted, confirmation was provided to the submitter by e-mail.

Staff reviewed each submitted comment. Only a few comments were not posted; reasons were: (1) duplicate submission of another comment posted by the same submitter, (2) test submission, (3) partial comment due to the 5,000 character limit, which was corrected by a shorter comment being submitted, and (4) comments that did not pertain to the DGAC.

At the request of the DGAC, staff generated reports and drafted summaries on each topic area for comments submitted since the previous meeting or since the previous comment summary. On occasion, various Committee members also chose to access the public comments database themselves in order to read comments.

A total of 972 comments were submitted from May 29, 2013 through the closing of the public comments database on December 30, 2014. Of these, 918 were relevant to the DGAC's work.

The majority of comments submitted fell into these topic areas: Food Groups; Eating Patterns-Diets; Sustainability; and Energy Balance. However, comments were received in all 18 topic areas and covered a wide range of issues. Comments came from the United States, Australia, India, Spain, Canada, Brazil, France, Belgium, Norway, Iraq, United Kingdom, Pakistan, Indonesia, and Denmark.

In addition to written comments, oral comments from 53 individuals were presented at the January 2014 public meeting. The list of presenters along with their affiliations is located on www.DietaryGuidelines.gov under Meeting 2 (January 13-14, 2014). These 53 individuals each provided 3 minutes or less of testimony before the Committee, and they submitted a brief outline of their comments when registering to participate in the comment session.

The oral and written comments provided by the public were valuable in that they helped the Committee gather background information and understand public and professional perceptions. Comments from the public brought new issues to light as well as new approaches to current issues and emerging evidence. They also highlighted and ensured consideration of topics deemed to be important by the submitters, who represented a variety of backgrounds and focus areas. The public comments will remain archived at www.DietaryGuidelines.gov.

Appendix E-8: Biographical Sketches of the 2015 DGAC

Chair: Barbara Millen, DrPH, RD: *Professor, Department of Family Medicine, Boston University School of Medicine, Boston, MA (through 2009).* Dr. Millen is currently the Founder and President of Millennium Prevention, Inc., a U.S.-based start-up company with a public health mission, which develops web-based platforms and mobile applications to encourage healthy preventive lifestyle behaviors for clinical settings and corporate, academic, and community wellness initiatives. Dr. Millen is a nutrition epidemiologist whose academic research career focused on dietary patterns and lifestyle determinants of health and chronic disease risk as well as evidence-based clinical and public health strategies to promote optimal nutrition and well-being in younger and older adults as well as low-income and minority populations. During her 30-year tenure at Boston University, she was the Founding Chairman of the Graduate Programs in Medical Nutrition Sciences, the Associate Dean for Research and Faculty Development of the School of Public Health, the Chairman of the Faculty Council, and Director of Nutrition Research for the internationally-renown Framingham Heart Study. She has advised research groups nationally and globally, including the World Health Organization, and served from 2008 to 2013 on the expert panels for the American Heart Association (AHA)/American College of Cardiology (ACC)/The Obesity Society (TOS) Guideline for the Management of Overweight and Obesity in Adults and the AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk.

Vice Chair: Alice H. Lichtenstein, DSc: *Stanley N. Gershoff Professor of Nutrition Science and Policy, Friedman School of Nutrition Science and Policy, Tufts University, Boston, MA.* Dr. Lichtenstein is also Director and Senior Scientist, Cardiovascular Nutrition Laboratory, Jean Mayer USDA Human Nutrition Research Center on Aging and Professor of Medicine at Tufts University School of Medicine. Dr. Lichtenstein has broad expertise in nutrition and cardiovascular disease risk reduction. She previously served as a member of the 2000 Dietary Guidelines Advisory Committee and as a member of the Institute of Medicine (IOM) Dietary Reference Intake Panel on Macronutrients. Dr. Lichtenstein recently served as the vice-chair of the IOM Committee on Examination of Front-of-Package Nutrient Rating System and Symbols, a member of the IOM Committee on the Consequences of Sodium Reduction in Populations, the vice-chair of the ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults expert panel, a member of the AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk expert work group, and is chair of the American Heart Association's Nutrition Committee. She is currently a member of the IOM Food and Nutrition Board.

Steven Abrams, MD: *Professor of Pediatrics, Baylor College of Medicine, Houston, TX.* Dr. Abrams also is an Adjunct Professor at the University of Texas School of Public Health and the Medical Director for the Neonatal Nutrition Program at Baylor College of Medicine. He is an expert on mineral requirements in children, including calcium, zinc, iron, magnesium, and copper. He has served on the IOM Panels on Calcium and Vitamin D and the Use of Dietary Reference Intakes in Nutrition Labeling, and on the IOM Subcommittee on Upper Safe Reference Levels of Nutrients. Dr. Abrams currently is a member of the American Academy of Pediatrics Committee on Nutrition and the American Society for Bone and Mineral Research.

Lucile Adams-Campbell, PhD: *Professor of Oncology, Georgetown University Medical Center, Lombardi Comprehensive Cancer Center, Washington, DC*. Dr. Adams-Campbell also serves as the Associate Director of Minority Health and Health Disparities Research and Associate Dean of Community Health and Outreach at Georgetown University Medical Center Lombardi Comprehensive Cancer Center. Dr. Adams-Campbell is an epidemiologist who specializes in community health research, interventions, and outreach and is a current member of the Institute of Medicine of the National Academies. She has played a leading role in the Washington, DC cancer and public health communities. Her research focuses on energy balance, diet and exercise. Dr. Adams-Campbell has participated in and led several large cohort studies of African-American women, and she played a leading role in bringing the Boston University Black Women's Health Study to the District of Columbia—the largest study of African-American women.

Cheryl Anderson, PhD, MPH: *Associate Professor of Preventive Medicine, Department of Family and Preventive Medicine, School of Medicine, University of California, San Diego, La Jolla, Calif*. Dr. Anderson also is an Adjunct Associate Professor, Department of Epidemiology at the Bloomberg School of Public Health, Johns Hopkins University. Her research expertise includes evaluating the role of nutritional factors in chronic disease prevention in minority and underserved populations, with emphasis on the role of dietary sodium and potassium intake in cardiovascular disease prevention. Dr. Anderson currently serves as a member of the IOM Food and Nutrition Board and has served on several other IOM committees including the 2013 IOM Committee on the Consequences of Sodium Reduction in Populations.

J. Thomas Brenna, PhD: *Professor of Human Nutrition, of Chemistry and Chemical Biology, and of Food Science, Cornell University, Ithaca, NY*. Dr. Brenna also is an Adjunct Professor, Department of Public Health Sciences at the University of Rochester College of Medicine and Dentistry. He is an expert in the field of fatty acid and lipid metabolism and in food fatty acid composition. His research focuses on the role of polyunsaturated fatty acids throughout the life cycle, in particular the effect of intake during pregnancy and lactation on fetal and infant development. Dr. Brenna has served as a panelist and author for the Expert Consultancy on Fats and Fatty Acids in Human Nutrition for the Food and Agriculture Organization and the World Health Organization.

Wayne Campbell, PhD: *Professor, Department of Nutrition Science, Purdue University, West Lafayette, IN*. Dr. Campbell also is an Adjunct Faculty in the Department of Health and Kinesiology, Purdue University. He is the Director of the Indiana Clinical Research Center at Purdue, which is a component of the NIH-supported Indiana Clinical and Translational Science Institute at the Indiana University School of Medicine. Dr. Campbell's expertise includes determining the dietary protein requirements of old and very old adults and evaluating the effects of protein, carbohydrate, and energy intakes and exercise training on macronutrient metabolism, body composition, and muscle strength and function. In addition, his research endeavors include studying the effects of food form, portion size, and dietary patterning on appetite and weight control with a special emphasis on the aging population.

Steven Clinton, MD, PhD: *John B. and Jane T. McCoy Chair of Cancer Research, The Ohio State University Comprehensive Cancer Center, and Professor, Division of Medical Oncology, Department of Internal Medicine, The Ohio State University School of Medicine, Columbus, OH.* Dr. Clinton also holds appointments in the Department of Human Nutrition in the College of Education and Human Ecology and in the Division of Environmental Health Sciences in the College of Public Health. He is a physician-scientist who has devoted his career to research in cancer etiology and prevention. Dr. Clinton's research focuses on epidemiology, clinical trials, community research, and experimental models, as well as cell and molecular systems. He has published extensively on the role of dietary energy balance and obesity in cancer risk, on a variety of foods associated with cancer prevention properties, as well as on several nutrients including vitamin D, calcium, omega-3 fatty acids, and vitamin E. He served on the IOM Committee on Dietary Reference Intakes for Vitamin D and Calcium.

Frank Hu, MD, PhD, MPH: *Director, Harvard Transdisciplinary Research in Energetics and Cancer Center, Department of Nutrition, Harvard School of Public Health, Boston, MA.* Dr. Hu also serves as Director, Boston Nutrition and Obesity Research Center Epidemiology and Genetics Core, a Professor of Nutrition and Epidemiology at the Harvard School of Public Health, and a Professor of Medicine at Harvard Medical School and Channing Division of Network Medicine, Brigham and Women's Hospital. Dr. Hu is an epidemiologist and an expert in the areas of dietary and lifestyle determinants of obesity, type 2 diabetes, and cardiovascular disease. He is the principal investigator for the diabetes component of the Nurses' Health Study. Dr. Hu has served as an academic leader in a variety of roles, including on the National Heart, Lung, and Blood Institute Obesity Guidelines Expert Panel and the IOM Committee on Preventing the Global Epidemic of Cardiovascular Disease.

Miriam Nelson, PhD: *Associate Dean, Jonathan M. Tisch College of Citizenship and Public Service, Tufts University, Boston, MA.* Dr. Nelson also is a Professor in the Friedman School of Nutrition Science and Policy. Dr. Nelson is an expert on nutrition and physical activity, with extensive research experience integrating the science of energy balance into national-scale approaches. Her work combines civic engagement, public policy, communications, and systems thinking to create change. Dr. Nelson is Founder of the Strong Women Initiative and Co-Founder of ChildObesity180 at Tufts University. Dr. Nelson served as Vice Chair of the Physical Activity Guidelines Advisory Committee in 2008 and was a member of the 2010 Dietary Guidelines Advisory Committee.

Marian Neuhouser, PhD, RD: *Full Member, Cancer Prevention Program, Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA.* Dr. Neuhouser also is an Affiliate Professor in the Department of Epidemiology and Core Faculty in the Graduate Program in Nutritional Sciences, School of Public Health, University of Washington. Dr. Neuhouser is a nutritional epidemiologist with broad experience in large clinical trials, including the Women's Health Initiative and the Prostate Cancer Prevention Trial, small-scale controlled dietary interventions, and large observational cohorts. She has expertise in the role of numerous dietary components in cancer risk, including carbohydrates, dietary fiber, and vitamin D. Her research focuses on methods to improve diet and physical activity assessment, diet and physical activity in relation to energy balance, diet-related health disparities, and dietary factors related to breast and prostate cancer prevention and survivorship.

Rafael Pérez-Escamilla, PhD: *Professor of Epidemiology and Public Health, Yale School of Public Health, New Haven, CT.* Dr. Pérez-Escamilla also serves as Director, Office of Public Health Practice and the Global Health Concentration at the Yale School of Public Health. He is an internationally recognized scholar in the area of community nutrition. Dr. Pérez-Escamilla has specialized experience with Hispanic and low-income Americans, as well as populations in low and middle income countries. His research program seeks to understand how best to protect, promote, and support breastfeeding, causes and consequences of food insecurity, and how to improve diabetes self-management through community health workers. Dr. Pérez-Escamilla has published numerous articles that have led to improvements in breastfeeding outcomes, iron deficiency anemia among infants, household food security measurement, and community nutrition education programs worldwide. He is past-chair of the Global Nutrition Council of the American Society for Nutrition and is a member of the IOM Food and Nutrition Board. Previously, Dr. Pérez-Escamilla served as a member of the IOM Committee to Re-examine IOM Pregnancy Weight Guidelines and was a member of the 2010 Dietary Guidelines Advisory Committee.

Anna Maria Siega-Riz, PhD, RD: *Associate Dean for Academic Affairs and Professor, Departments of Epidemiology and Nutrition, Gillings School of Global Public Health, University of North Carolina at Chapel Hill, Chapel Hill, NC.* Dr. Siega-Riz serves as the Program Leader for the Reproductive, Perinatal, and Pediatric Program in the Department of Epidemiology. Dr. Siega-Riz has focused her research on maternal nutritional status, including maternal obesity and gestational weight gain and their effect on birth outcomes as well as the determinants of early childhood obesity. She studies dietary patterns among Hispanic adults and children, in general, and served on the Scientific Advisory Panel for the Feeding Infants and Toddlers Study. Dr. Siega-Riz has served on multiple committees for the IOM, examining topics from the WIC food packages to standards for systematic reviews in health care and currently serves on the advisory council of the National Heart, Lung, and Blood Institute.

Mary Story, PhD, RD: *Professor, Community and Family Medicine and Global Health, Duke University, Durham, NC*. Before coming to Duke in January 2014 she was Senior Associate Dean for Academic and Student Affairs and Professor in the Division of Epidemiology and Community Health in the School of Public Health, University of Minnesota. Dr. Story concurrently serves as Director of the National Program Office for the Robert Wood Johnson Foundation Healthy Eating Research Program that supports research on environmental and policy strategies to promote healthy eating among children to prevent childhood obesity. She has conducted numerous school and community-based environmental intervention and obesity prevention studies for children, adolescents, and families. Dr. Story was elected to the IOM in 2010 and is currently a member of the IOM Food and Nutrition Board and vice co-chair of the IOM Roundtable on Obesity Solutions.

Consultant Subcommittee Members to the 2015 DGAC

Timothy S. Griffin, PhD: *Director, Agriculture and Environment Program, Friedman School of Nutrition Science and Policy, Tufts University, Boston, MA.* Dr. Griffin also is an Associate Professor at Tufts University where he serves on the Water: Systems, Science and Society faculty steering committee and is a Faculty Co-Director for the Tufts Institute for the Environment. His research expertise and interests include the intersection of agriculture and the environment, and the development and implementation of sustainable production systems. Previously he worked as a Research Agronomist and Lead Scientist with USDA-ARS New England Plant Soil and Water Lab, and as Extension Sustainable Agriculture Specialist at the University of Maine.

Michael W. Hamm, PhD: *Director, Center for Regional Food Systems, Michigan State University, East Lansing, MI.* Dr. Hamm is also the C.S. Mott Professor of Sustainable Agriculture in the Department of Community Sustainability in the College of Agriculture and Natural Resources and has appointments in the Department of Food Science Human Nutrition and the Department of Plant, Soil and Microbial Sciences at Michigan State University. His research expertise and interests include regional and sustainable food systems and food security.

Michael G. Perri, PhD, ABPP: *Dean, College of Public Health and Health Professions University of Florida; Gainesville, FL.* Dr. Perri is also the Robert G. Frank Endowed Professor of Clinical and Health Psychology. His research focuses on health promotion and disease prevention through changes in diet and physical activity. His NIH-funded studies involve the translation, dissemination, and implementation of effective programs for the management of obesity in underserved rural communities. Dr. Perri has served as a member on NIH data and safety monitoring boards, including serving as chair of the recent NIH/NHLBI Data and Safety Monitoring Board for the "EARLY Weight Loss Trials."

Appendix E-9: Work Structure and Member Organization

Work Group Structure

Work structure from inception through fall 2013.

Work Group 1: Environmental Determinants of Food, Diet, and Health

Miriam Nelson (Lead)
Steven Abrams
Lucile Adams-Campbell
Mary Story

Work Group 2: Dietary Patterns and Quality and Optimization through Lifestyle Behavior Change

Rafael Pérez-Escamilla (Lead)
Cheryl Anderson
Gary Foster[*]
Frank Hu
Anna Maria Siega-Riz

Work Group 3: Foods, Beverages, and Nutrients and Their Impact on Health Outcomes

Alice H. Lichtenstein (Lead)
J. Thomas Brenna
Wayne Campbell
Steven Clinton
Marian Neuhouser

[*] Dr. Gary Foster assumed a new position shortly after being appointed as a member of the 2015 DGAC. Due to the significant demands of the new position, it became necessary for Dr. Foster to resign his appointment to the 2015 DGAC (August 2013).

Subcommittee Structure

Work structure from fall 2013 through completion of the report.

Science Review Subcommittee *(In place from inception through completion of the report.)*
Barbara Millen (Chair)
Alice H. Lichtenstein (Vice Chair)
Miriam Nelson (2010 and 2015 DGAC member)
Rafael Pérez-Escamilla (2010 and 2015 DGAC member)

SC 1: Food and Nutrient Intakes, and Health: Current Status and Trends
Marian Neuhouser (Chair)
Alice H. Lichtenstein (Chair/Vice Chair Representative)
Steven Abrams
Cheryl Anderson
Mary Story

SC 2: Dietary Patterns, Foods and Nutrients, and Health Outcomes
Anna Maria Siega-Riz (Chair)
Alice H. Lichtenstein (Chair/Vice Chair Representative)
Cheryl Anderson
Tom Brenna
Steven Clinton
Frank Hu
Rafael Pérez-Escamilla
Marian Neuhouser

SC 3: Diet and Physical Activity Behavior Change
Rafael Pérez-Escamilla (Chair)
Barbara Millen (Chair/Vice Chair Representative)
Wayne Campbell
Steven Clinton
Anna Maria Siega-Riz
Lucile Adams-Campbell
Michael Perri (Consultant)

SC 4: Food and Physical Activity Environments
Mary Story (Chair)
Barbara Millen (Chair/Vice Chair Representative)
Lucile Adams-Campbell
Wayne Campbell
Mim Nelson

SC 5: Food Sustainability and Safety
Mim Nelson (Chair)
Barbara Millen (Chair/Vice Chair Representative)
Steven Abrams
Tom Brenna
Frank Hu
Michael Hamm (Consultant)
Tim Griffin (Consultant)

Working Group Structure

Work structure developed as need identified from spring 2014 through completion of report.

Added Sugars Working Group
Miriam Nelson (Co-Lead)
Mary Story (Co-Lead)
Cheryl Anderson
Wayne Campbell
Frank Hu
Alice H. Lichtenstein
Barbara Millen
Marian Neuhouser

Sodium Working Group
Cheryl Anderson (Lead)
Wayne Campbell
Steven Clinton
Alice H. Lichtenstein

Saturated Fat Working Group
Frank Hu (Lead)
Tom Brenna
Alice H. Lichtenstein
Barbara Millen

Physical Activity Writing Group
Miriam Nelson (Lead)
Wayne Campbell
Alice H. Lichtenstein

Appendix E-10: Dietary Guidelines Advisory Committee Report Acknowledgments

Invited Expert Speakers

Steven Abrams, MD
Dawn Alley, PhD
Sonia Angell, MD, MPH
Amelia Arria, PhD
Robert Brackett, PhD
Laurel Bryant,
Kathryn B. H. Clancy, PhD
William H. Dietz, MD, PhD
Linda Duffy, PhD, MPH
Robert H. Eckel, MD
Lorraine Gunzerath, PhD, MBA
Van Hubbard, MD, PhD
Susan M. Krebs-Smith, PhD, MPH, RD
Antonia Mattia, PhD
J. Michael McGinnis, MD, MA, MPP
Kathleen Merrigan, PhD
Alanna Moshfegh, MS, RD
Suzanne Murphy, PhD, RD
Catherine Oakar, MPH
Rafael Pérez-Escamilla, PhD
Barry M. Popkin, Ph.D
John Ruff, MA
Michael B. Rust, PhD
Jill Reedy PhD, MPH, RD
Donna H. Ryan, MD
Marie-Pierre St-Onge, PhD, FAHA
Pam Starke-Reed, PhD
Patrick Stover, PhD
Deborah F. Tate, PhD
Katherine Tucker, PhD
Connie M. Weaver, PhD
CAPT Andrew Zajac

Staff, Contract, and/or Technical Support

Nadine S. Braunstein, PhD, RD
Eileen Dykes, MS, RD
Janet East
Janie Fleming
Yolande Gary
Vibhuti Giltrap
Joseph Goldman, MA
Hazel Hiza, PhD, RDN
Joy Jackson Farrar
Ashlee Johnson
Olga Nelson
Andrea Popp
Cikena Reid
Ronnie Rogers
Ken Ryland
India Taylor
Jennifer Wilkinson
Miyuki Shimizu, PhD Candidate
Jeff Steele
Teresa T. Fung, ScD, RD

National Service Volunteer Evidence Abstractors

Brian R. Barrows, PhD
Samantha Berger, MS, MPH
Cynthia Blanton, PhD, RD
Stacy Blondin, MSPH
Eric Calloway, PhD
Sheau Ching Chai, PhD, RD
Sarah Forrestal, PhD
Carol C. Giesecke, PhD, RD, LDN
Maryam S. Hamidi, MSc, PhD
Heidi L. Himler, MPH, RD, LD
Tiffany Huang, MS
Sarah A. Johnson, PhD, RD, CSO
J. Philip Karl MS, RD
Lydia Kaume Ph.D
Alexandra Kazaks, PhD, RDN
Kathryn Lawson, MS, RDN, CD
Annie W. Lin, MS, RD, CDN
Dalia Majumdar, PhD
Melissa A. Masters, PhD, RDN
Kevin Pietro, MS, RD, LDN
Maja Redzic, MS
Amy Steffey, DVM, MPH
Maria L. Stewart, PhD
Libo Tan, PhD
Alison K. Ventura, PhD
Ding Ding Wang, MPH
Marquitta C. Webb, Ph.D., LN
Sahar Zaghloul, MBBS, PhD