Risk Assessment of Disinfection Byproducts in Poultry Chilled in Chlorinated Water

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Executive Summary

This report summarizes the assessment of the public health risk associated with consumption of poultry containing residual chlorine and disinfection byproducts (DBPs) after washing and chilling in chlorinated water, using information obtained from published scientific literature, and unpublished academic, government and industry sources. The objectives of the report are to: evaluate safety of chlorine and its DBPs at levels used in water chlorination; determine the risk of exposure to hazardous DBPs through consumption of poultry treated with chlorinated water; and assess the benefits and risks of chlorine use in poultry processing to public health.

Chlorine is a halogen element that has been successfully used to maintain the microbial safety of foods and water. The addition of chlorine to water and food leads to the formation of many DBPs such as trihalomethanes and haloacetic acids, which raises concerns about chemical safety of drinking water and food supply. Extensive research has been done to examine acute and subacute toxicity and reproductive, teratogenic and developmental disorders resulting from exposure to chlorine and chlorine derivatives in human and animal models. Some studies have suggested a possible link between DBPs and cancer. However, much of the evidence surrounding this association is inconclusive concerning levels of chlorine used for water treatment and food processing.

Microbiological safety of food and water continues to be a challenge worldwide. For over a century since it was first used, chlorination has established itself as a safe, cheap, convenient, and effective treatment for water and control of microbial contamination in foods. Chlorine is effective against a large variety of microorganisms including significant foodborne and waterborne pathogens such as *Salmonella, Escherichia coli, Shigella, Campylobacter, Vibrio* and noroviruses. Effectiveness of chlorine as a disinfectant depends highly on concentration and acidic pH. Chlorination of water used for chilling of poultry carcasses is effective if an initial chlorine load of 50 ppm is used and 5 ppm residual chlorine maintained.

Chilling poultry carcasses in chlorinated water ensures the reduction of important pathogenic microbes. However, residual DBPs have been detected in poultry products. As DBPs evaporate during cooking, human exposure to DBPs through the consumption of poultry products is minimal. Further analysis of daily diet exposure to DBPs (chloroform) revealed that water is the predominant source and contributes 99% of the daily exposure to DBPs. Poultry chilled with chlorinated water at 50 ppm accounts for

only 0.3-1 % of the exposure. Therefore, DBPs exposure from consuming poultry does not create a significant risk for cancer or other health conditions.

In conclusion, the current scientific evidence on the association of residual levels of DBPs with cancer does not warrant changing the guidelines of 50 ppm chlorine for chiller water treatment in poultry processing as the application is essential to reducing pathogen load and controlling cross contamination, thereby maintaining and improving the safety of poultry products. It is important to emphasize that public health gains from reduced waterborne and foodborne illnesses using chlorine in poultry processing far outweigh the risks from cancer.

Introduction

Chlorine is a halogen (group VII) element that is mainly produced by electrolysis of sodium chloride in water to produce chlorine gas, sodium hydroxide and hydrogen gas (Eifert & Sanglay 2002, IC controls 2005). Chlorine is added to drinking water to destroy harmful microorganisms and to protect water from recontamination. It was first added to a water main in Maidstone, UK, to control an outbreak of typhoid fever in 1897 (Eurochlor 2008). It is used for treatment of drinking water, decontamination of equipment and environment, and for control of microbial contamination in food production (ACC 2003, CDW 2008). As disinfectants, chlorine and chlorinated compounds have several advantages and disadvantages, as listed in Table 1.

Table 1. Advantages and disadvantages of chlorinated compounds*

Advantages	Disadvantages
Effective against bacteria , fungi and viruses	Exhausted with heat and organic materials
50 ppm for 30 s passes the chambers test	Mainly active at acidic pH
Low cost	Corrosive to stainless steel at acidic pH
No need to rinse equipment at < 200 ppm	Deteriorate when exposed to light or heat
Not affected by hard water salts	
Relatively non-toxic	

*based on Mariott, 1999.

Chlorine is very unstable and reacts with water to form hypochloric acid (at acidic pH) (CDC 2002) which has the main disinfection capacity. Hypochloric acid disinfects by oxidizing proteins, carbohydrates and other organic compounds, and becomes chloride ion. During water chlorination, approximately 2 % of the hypochlorite ion form carbon chlorine bonds and lead to main chlorination disinfection byproducts (DBPs) (Eurochlor 2005), including trihalomethanes (THMs) as the most common volatile DBPs and haloacetic acids (HAAs) as the major non-volatile DBPs (CDC 1997). Other DBPs, such as haloacetonitriles (HANs), chloropicrine and chlorinated furanones, are usually present at lower concentrations. The World Health Organization (WHO) as well as the US, EU, Canada and other countries has published guidelines and limits for chlorine and/or its byproducts in water (Table 2). Current guidelines of WHO, the Codex Alimentarius and the US allow a maximum concentration of 5 mg/L chlorine in drinking water and 50 mg/L chlorine in water in contact with poultry.

Table 2. Limits of chlorine and byproducts in water and intake in humans*

Authority	Parameter	Chemical levels**	Reference
WHO	Recommended limits	Chlorine 5 mg/L Chlorate 700 μg/L Chloride 700 μg/L Chloroform 300 μg/L Tricholoroacetate 200 μg/L Trihalomethanes 200 μg/L	WHO 2008
European Union	Chemical parameter limits	Trihalomethanes 100 μg/L	OJEC 1998
	Indicator parameters	Chloride 250 μg/L	
US EPA	Max. residual disinfection levels	Chlorine 4 mg/L Chloramine 4 mg/L Chlorine oxide 0.8 mg/L	EPA 1992
	Chronic oral reference dose (RfD)	Chloroform 0.01 mg/Kg/day	EPA 1999
	RfD for average adult (75 Kg)	Chloroform 0.75 mg/day	
US FSIS	Max. allowed in carcass wash	Chlorine up to 50 mg/L	FSIS 2004
Health Canada	Allowable level	Trihalomethanes 100 μg/L	Health Canada 2004

* derived from a variety of studies using mice and rats to estimate the hazards associated with the exposure to chlorine and its byproducts on human health.

** 1 mg/L = 1 ppm; 1 μg/L = 1ppb.

It is widely recognized that all chemical disinfectants form some potentially harmful byproducts. Chlorination DBPs are by far the most thoroughly studied. There have been concerns regarding DBPs when chlorine is used for disinfection of equipment and environment and for control of microbial contamination in food production, including fish, fresh produce, meat and poultry processing. The principal concerns are associated with the uptake of chlorine from washing and/or chilling water by food products and the uptake and/or formation of DBPs in food resulting from the use of chlorinated water. Some studies have shown possible associations between DBPs and cancers. However, much of the evidence is inconclusive concerning levels of chlorine used for water treatment and food processing. Public health gains from reduced waterborne and foodborne illnesses far outweighs the risks from cancer (WHO, 1998).

Safety of chlorine and DBPs

There is only limited information in the scientific literature on the health effects of DBPs resulting from the use of chlorine disinfection in food processing. Most studies in the literature focus on the formation of DBPs and the possible hazardous effects when drinking water is chlorinated. Two data sources are available: those derived from animal experiments in the laboratory (toxicology) and those from human population evaluations using epidemiology techniques. The risk evaluations derived from the two sets of data are often different due to a number of factors such as different endpoints, response time and exposure concentrations (Hamidin 2008).

Acute toxicity studies usually used mice or rats with extremely high exposure doses of DBPs to examine their health effects in a relatively short period of time, ranging from a few days up to weeks or months (Logomasini 2006). Such studies in animal models have shown adverse health effects of DBPs. For example, THM exposure resulted in tumor formation in liver and kidney, and affected reproductive capacity. However, the inferential value of high doses over a short period to estimate trace amount over a long period of time has not been proven (Whelan 2004). Other studies using relatively low exposure concentrations of DBPs showed no significant sign of adverse health effects in animals and humans. The results from some of the studies are presented in the following.

Toxicity

Investigators used C57BL/6 mice, CR1:CD1 mice and Sprague-Dawley rats to study the effect of hypochlorite on the function of the immune system. The rodents were fed hypochlorite concentrations up to 30 mg/L for up to 12 weeks. No statistical differences were observed in mesenteric lymph nodes, lymphocyte proliferation, spleen or thymus weight, antibody titers or number of antibody-forming cells between the control group and the experimental groups, including the 30 mg/L subgroup. The authors concluded that consuming increased amount of hypochlorite did not affect in vivo immune function of rats or mice (Herman et al. 1982, Exon et al. 1987, French et al. 1998).

In a similar study, F344 rats were fed hypochlorite at concentrations of up to 4000 mg/L in water for 92 days. The authors reported decreased water consumption in the experimental group along with slight organ shrinking (due to decreased water), but did not note any changes or pathological effects associated with ingesting extremely high concentrations of hypochlorites (Furukawa et al. 1980).

Additionally, Daniel et al. (1990, 1991) examined the effect of up to 200 mg/L chlorine on Crl:CDBR rats and B6C3F1 mice for 90 days. The authors noted a statistically significant decrease in water consumption in rodents receiving high doses of hypochlorite but did not see any signs of toxicity. Furthermore there were no histopathological changes or lesions detected, further establishing the safety of chlorinated water.

Several studies also directly examined adverse health effects of chlorine in humans. Subjects and military personnel drank hyperchlorinated water containing up to 90 ppm hypochlorite for several months. The subjects reported mouth irritation at 90 ppm (Muegge 1956). There were no noted adverse effects with any of the groups and no toxicity was observed (Muegge 1956, US EPA 1994, Australian NHMRC 2004).

In a different study, subjects drank 1.5 L water containing chlorine byproducts at 20 mg/L for 4 weeks (Wones et al. 1993). The authors examined the possible effect of highly chlorinated water on the thyroid gland and concluded that ingesting chlorinated water at that level did not pose a significant effect on lipid or thyroid metabolism.

Carcinogenicity examination/long term exposure

Hypochlorous acid was given to Sprague-Dawley rats at concentration up to 100 mg/L for one year. The authors assessed redox parameters, chloroform content of blood and studied hematological changes. No chloroform was detected in the blood throughout the study and no consistent hematological changes were associated with the consumption of chlorinated water or its derivatives (IRIS 1994).

Other studies administered water with up to 275 mg/L hypochlorite to F344/N rats and B6C3F1 mice for two years. These knockout animals had a high predisposition to develop different types of cancer. The treatment group showed a decrease in water consumption attributed to the aversion to chlorine taste at extremely high concentrations. In rats, the control and one of the experimental groups showed significant differences from the historically established baseline, thus no dose response relationship was established (US EPA 1994). In mice, survival rates of the treatment group were not significantly different from the controls and the authors did not detect any gross or microscopic lesions attributable to the treatment (WHO 2003).

Mutagenicity potential

Several studies examined the mutaginicity of chlorine and hypochlorite using microorganisms. Le Curieux (Le Curieux et al. 1993) reported a negative SOS chromtest, whereby no chromosomal damage was observed in the particular *E. coli* strain. The authors also reported a negative Ames test with *Salmonella* Typhimurium. The authors concluded that sodium hypochlorite was not responsible for any mutations in the tested bacterial strains (Le Curieux et al. 1993).

To further assess the mutagenicity potential of hypochlorite solutions, Syrian hamster embryo cells were exposed to a 12.6 % hypochlorite solution which amounts to 126,000 ppm. The authors examined the ability of sodium hypochlorite to induce unscheduled DNA synthesis. The study concluded that hypochlorite, even at these extremely high concentrations, did not induce unscheduled DNA synthesis in any of the test samples (Hamaguchi and Tsutsui 2000).

Effect of reproductive and developmental processes

Several studies administered chlorinated water to rodents on a regular basis to assess the potential adverse effects of chlorine and its derivatives on reproductive and developmental health by observing changes after mating, gestation and lactation.

Sodium hypochlorite at 100 mg/L was administered everyday to BDII rats over their entire lifetime, for seven consecutive generations. The authors did not note any changes in growth pattern, lifespan or fertility in any of the generations. Furthermore, liver, spleen and kidney analysis did not reveal any changes, and the rate of malignant tumors was identical to the control group, further proving the lack of any reproductive effect of hypochlorite at these concentrations (Druckey 1968).

Female Sprague-Dawley rats were given up to 100 mg/L hypochlorous acid for 75 days before insemination. On day 20 of gestation, the rodents were sacrificed and fetuses were examined. The authors recorded weight, malformation, skeletal abnormalities and soft tissue defects. No significant differences were observed in any of the previously mentioned parameters compared to the controls. The authors concluded the lack of any data suggesting a teratogenic or embryotoxic effect of hypochlorite in pregnant rats (Abdel-Rahman et al. 1982).

Hypochlorite at 0, 1, 2 and 5 mg/L was given to Long-Evans rats through breeding, gestation, lactation and until weaning. The authors examined females for fertility,

length of gestation, maternal behavior and changes in the reproductive tract and found no significant differences compared to the control group. Furthermore, the hypochlorite treatment group did not show any changes in pup viability, litter size, day of eye opening or weight compared to the untreated controls. The authors found no signs of hematological, developmental or teratogenic detrimental effects in the treatment group compared to the control (Carlton et al. 1986).

In summary, numerous acute, subacute, developmental, teratogenic and mutagenic assays presented the lack of any significant risk from the consumption of chlorinated byproducts at levels matching those in drinking water. The only adverse effects observed were in animals given doses that are 1000s to 10,000 folds higher than the ones regularly consumed through drinking water.

Importance of chlorine to microbiological safety control

Foodborne diseases are a widespread and growing public health problem, both in developed and developing countries. The global incidence of foodborne disease is difficult to estimate, but it has been reported that in 2005 alone 1.8 million people died from diarrheal diseases (WHO, 2007). A great proportion of these cases can be attributed to contamination of food and drinking water. In the United States, 76 million cases of foodborne diseases, resulting in 325,000 hospitalizations and 5,000 deaths, are estimated to occur each year (Mead, 1999). Major foodbonre pathogens include *Salmonella*, *Campylobacter*, *Listeria*, pathogenic *Escherichia coli* and *Vibrio cholera*. Poultry products are frequently contaminated with *Campylobacter* and *Salmonella*.

The use of chlorine for sanitation and disinfection purposes was certainly a milestone in combating infectious diseases in human history (Eurochlor 2008). In Europe, hypochlorite successfully controlled and halted a *Salmonella* (typhoid) outbreak in UK (Eurochlor 2008). Since then, chlorination established itself as a safe, cheap, convenient, and effective treatment for foods and water (WHO 2003). Hypochloric acid has the main antimicrobial activity. Being uncharged at acidic pH, it can penetrate the membrane of different microorganisms and dissociate inside, thus acidifying the cytoplasm in addition to the oxidizing effect (Eifert and Sanglay 2002). Hypochloric acid kills cells by inhibiting glucose oxidation through reaction with the sulfhydryl group of certain enzymes (Eifert and Sanglay 2002). Other modes of action have been suggested for chlorine, including: protein synthesis disruption, reaction with nucleic acids especially purines and pyrimidines, causing lesion in DNA and oxidative decarboxylation of amino acids leading to aldehydes and nitrites (Mariott 1999).

Table 3. Significant food and waterborne microbial pathogens and their chlorine susceptibility*

Pathogen	Health significance	Resistance to chlorination	Animal source
Bacteria			
Campylobacter jejuni, C. coli	High	Low	Yes
Escherichia coli – Pathogenic	High	Low	Yes
E. coli – Enterohaemorrhagic	High	Low	Yes
Legionella spp.	High	Low	No
Salmonella typhi	High	Low	No
Other salmonellae	High	Low	Yes
Shigella spp.	High	Low	No
Vibrio cholerae	High	Low	No
Yersinia enterocolitica	High	Low	Yes
Viruses			
Enteroviruses	High	Moderate	No
Hepatitis A virus	High	Moderate	No
Hepatitis E virus	High	Moderate	Potentially
Noroviruses	High	Moderate	Potentially
Rotavirus	High	Moderate	No

*Adopted from a WHO report (WHO 2008)

Chlorine and its byproducts are effective against a large variety of microorganisms (Table 3) including bacteria and viruses (ACC 2003, CDW 2008, WHO 2008). Chlorine antibacterial activity includes Gram-positive, Gram negative as well as some spore-forming bacteria (Mariott 1999). *Salmonella, E. coli, Shigella, Campylobacter,* and *Vibrio* are some of the most prominent ones (CDW2008). Chlorination can also limit the growth of biofilms on food surfaces thereby preventing the spoilage of the food and generation of unpleasant odors (LeChevalier 1998).

Chlorine is widely used in the food industry for reducing microbial contamination as a microbial safety control practice rather than a decontamination treatment. The effectiveness of chlorine against microbial pathogens depends on concentrations (more importantly free residual chlorine) and contact time among other factors. For example, bacterial reduction is greater in chill systems than spray systems because of the greatly increased contact time (45-60 minutes versus a couple of minutes). The concentration of chlorine, however, is not nearly as important as the amount of organic material in the water. In drinking water systems where very little organic material is present in the water, 0.5 ppm chlorine is effective for eliminating bacteria, whereas up to 50 ppm is required in the chiller of poultry processing where high organic loads are encountered.

For fruits and vegetables, 25-250 ppm free chlorine is used in washing water due to relatively short contact time (a few minutes) (Eifert and Sanglay 2002).

Chlorine was first used to extend product shelf-life in poultry processing. However, it has little direct effect on carcass bacteria as attached or entrapped pathogens are not readily accessible to chlorine (Lillard, 1993). The main benefit from chlorination of process water lies in its ability to control microbial contamination of the processing environment and equipment. Mead and Thomas (1973) reported that chlorine at a total residual of 45-50 ppm could be used to keep the chill water virtually free from viable bacteria, thereby reducing the opportunity for cross-contamination of carcasses.

Several studies investigated the effect of chlorinated water at different levels on the survival of foodborne pathogens. A 2-log reduction in bacterial vegetative cells can be achieved by using 13 ppm free available chlorine for up to 100 s (Mariott 1999). To achieve similar reduction in spores, the free available chlorine should be around 1000 ppm for up to 20 min (Odlaug 1981). Using chlorine at less than 50 ppm did not destroy *Listeria monocytogenes* cells (Mariott 1999). Park et al. (2002) reported 2.6 log reduction in *C. jejuni* counts in chicken immersed in 50 ppm chlorinated water at 4 °C for 10 min. A poultry wash step in chlorinated water at 25-35 ppm produced a 0.5 log CFU decrease in *C. jejuni* counts (Bashor et al. 2004).

Canadian agencies found that the optimal residual chlorine to control bacterial growth in drinking water is up to 5 mg/L or 5 ppm (CFIA 2004, CDW 2008). The data assume that all the organic materials have been neutralized and the main purpose is to prevent growth of microorganisms in case of a contamination. This residual level cannot be achieved if the initial chlorine in poultry carcass chilling is set at 0.5 ppm.

A study used a 45 min chlorinated water immersion wash step at 4 °C, using 25 ppm initial chlorine, and showed no significant reduction in *S.* Typhimurium or *E. coli* on poultry. The microbial counts of treated samples were within 0.5 log range of the untreated control (Fabrizio et al. 2002).

In responding to issues regarding effectiveness of chlorine treatment in poultry pre-chiller and chiller water for microbiological safety, the Food Safety Service and Inspection (FSIS) of US Department of Agriculture issued a notice in 2003 to clarify and reiterate its policy. FSIS stated that potable water used to initially fill the pre-chiller, chiller, or red water system, or that is added as makeup water, may contain up to 50 ppm free available chlorine measured at intake. Water from the red water system that is re-used in the pre-chiller or chiller may contain no more than 5 ppm free available chlorine measured at influent to pre-chiller or chiller (FSIS, 2003).

In summary, chlorination of water used for chilling of poultry carcasses is only effective if an initial chlorine load of 50 ppm is used and 5 ppm residual chlorine maintained. This step reduces microbial contamination and extends the shelf-life of the meat. The efficacy of chlorine as a bactericidal agent is affected by the pH value of the solution and the amount of organic material present. In poultry chillers, levels of free available chlorine are kept at 20 to 50 ppm. Because the chiller environment has a relatively high organic load, the effectiveness of chlorine is often reduced.

Residual DBPs in poultry, and impact of cooking

Chilling poultry carcasses in chlorinated water ensures the reduction of microbes. The practice is especially effective in minimizing levels of *Salmonella* and *Campylobacter*, two most common causes of human gastroenteritis. However, formation of DBPs in poultry chiller water has been detected. Several studies also showed residual DBPs in poultry products. Fortunately, the trace amount of DBPs should not cause a concern regarding adverse health effects. For example, immersing chicken carcass for 20 minutes in 50 ppm chlorinated water at 2.5°C resulted in 46 ppb of chloroform in chicken fat (where chloroform accumulates), which is low compared to 300 ppb in drinking water (Robinson et al. 1981). Chicken breast had the lowest chloroform content, at 17 ppb, which is minimal compared to the content of regular drinking water, especially when the amount consumed is calculated (Robinson et al. 1981). A risk assessment study focusing on the accumulation of chloroform in skin and fat of poultry after going through a chlorinated water chilling step revealed that the skin and fat contained a median of 3.6 and 4.5 ppb, respectively (FSIS 1994).

Studies also tested the levels of chloroform as a representative of THMs during cooking of the meat chilled in chlorinated water. Cooking resulted in a significant reduction in chloroform compared to the raw product (255 ppb to 46 ppb). Since chloroform is in gas state at high cooking temperatures (Boiling point 62 °C; CDC 2005), FSIS recognizes that chloroform may evaporate during cooking (FSIS 1994). It's been also suggested that trihalomethanes and many of the halocarbon derivatives will evaporate during cooking (Eurochlor 2005, CAC 2000). Entz et al. (1982) examined halocarbon residue in meat, poultry and fish using gas chromatography and found that most of the halocarbons evaporated after the meat was cooked. These findings agree with previous studies reporting a significant decrease in halocarbons after cooking.

Table 4. Chloroform standards and its present in water and food

	Chloroform levels	Reference
Standards		
Maximum limits	80 μg/L	American Chemistry 2003
Chronic Oral Reference Dose (RfD)	10 μg /Kg/day	US EPA 1992
RfD for average adult (75 Kg)	750 μg /day	
Limits based on 3 L/day	300 μg/L	WHO 2008
Chemical parameters (THM)	THM 100 μg/L	OJEC 1998
Water and Food		
Drinking water	311 μg/L	Robinson et al. 1981
Butter	670 μg/kg	Heikes 1987
Sausages	90 μg/kg	WHO 1994
Cheddar cheese	80 μg/kg	Heikes 1987
Coffee	80 μg/kg	WHO 1994
Peanut butter	29 μg/kg	Heikes 1987
Breaded shrimp	24 μg/kg	Heikes 1987
Poultry fat	146 μg/kg	Robinson et al. 1981
Poultry skin	30 μg/kg	Robinson et al. 1981
Poultry muscle	17 μg/kg	Robinson et al. 1981

Potential daily exposure to DBPs (chloroform)

Trihalomethanes (THMs) and HAAs are two major classes of DBPs commonly found in waters treated with chlorine. Chloroform, also known as trichloromethane, TCH belonging to THMs, is most widely regulated, and potentially has adverse health effects on humans. Due to its significance, extensive research has been conducted to determine its presence in food and environment (WHO, 2004). It is interesting to note that chloroform is not limited to drinking water or poultry washed in chlorinated chilling water. A study examining a broad variety of ready-to-eat foods found chloroform in cheddar cheese (80 μg/kg), fried shrimp (24 μg/kg), butter (670 μg/kg), peanut butter (29 μg/kg) and coffee (80 μg/kg) at levels comparable or greater than those found in poultry (Heikes 1987) (Table 4).

The US EPA has established 750 μg /day as a chronic oral reference dose for adults (US EPA, 1992), whereas WHO sets the limit to 900 μg/day based on daily consumption of 3 L drinking water. These chloroform standards and its content levels in various foods and water can be used to estimate daily intake values of chloroform through drinking water and food (Figure 1). Based on the recommendations on serving sizes and daily requirements in the Dietary Guidelines published by USDA (USDA 2005), we converted the chloroform content of different foods and water to daily chloroform exposure using "mypyramid.gov" database (http://www.mypyramid.gov/index.html).

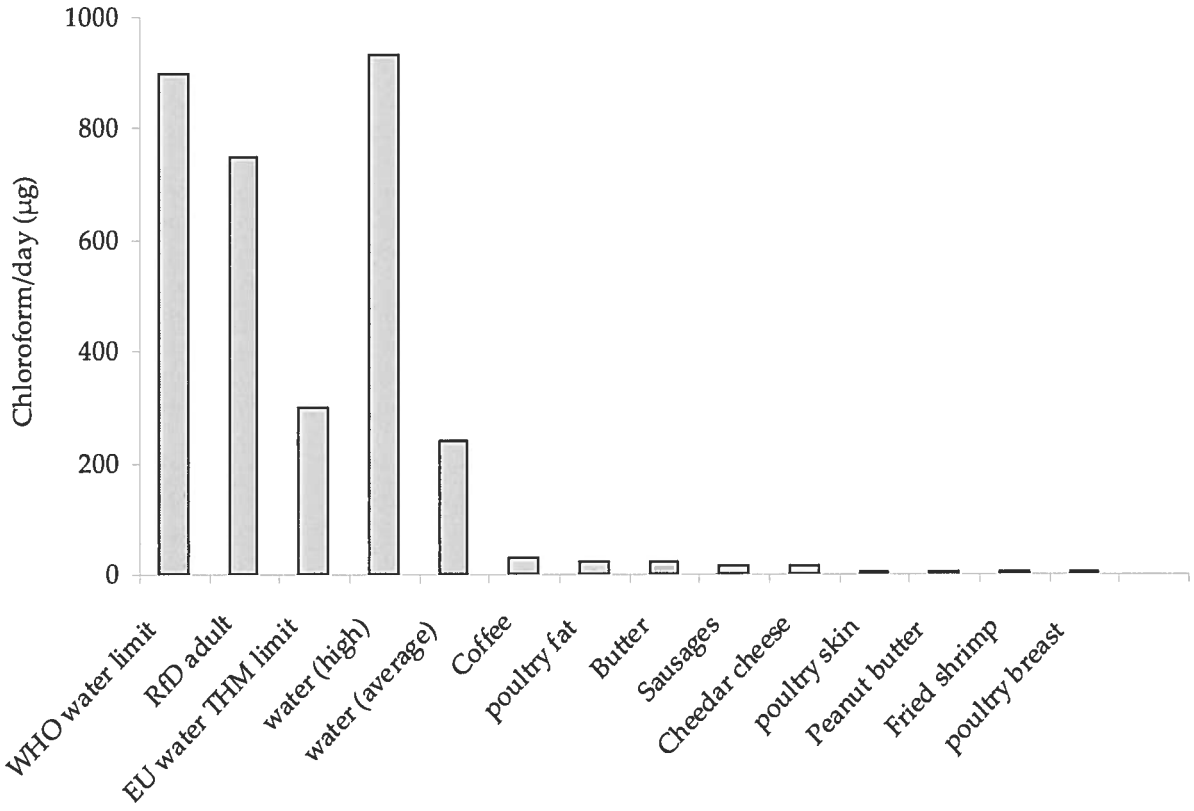

Figure 1: Daily intake of chlorinated products, represented by chloroform, from a variety of foods and water sources compared to the allowed limits. Calculation was based on "my pyramid" daily requirements.

The daily chloroform exposures (μg/day) from drinking water and foods were plotted on a bar graph (Figure 1) to allow for a visual comparison of the contribution of poultry chilled with chlorinated water at 50 ppm chlorine to chloroform intake by ingesting chlorinated water and consuming a variety of common foods. It is clear that water is the predominant source of DBPs (chloroform) in the daily exposure. In fact, the daily intake of chloroform from chlorinated water can be up to 30 times greater than that

from food with the highest chloroform content. Moreover, consuming full daily requirements of chicken exposes you to 100-300 fold less chloroform than what you get from water. Thus, poultry (156 g daily requirement, USDA 2005) chilled in chlorinated water contributes only 0.3-1 % of the daily chloroform exposure, whereas water contributes most (99%) of the exposure.

Conclusion

Chlorine is a common disinfectant used in water treatment and food processing worldwide. It is very effective to destroy foodborne pathogens such as *Salmonella* and *Campylobacter* that are frequently present in poultry products. Efficacy of chlorine in killing microorganisms and preventing contamination/recontamination depends highly on concentrations and acidic pH. Chlorination of water in chiller at 50 ppm in poultry process has proven to be an effective method in reducing microbial contamination, thereby improving microbial safety of poultry products.

Chlorine DBPs have been identified to have potential adverse health effects at high level exposure. Poultry chilled with chlorinated water at 50 ppm is an insignificant source of chlorinated DBPs, especially after cooking. The contribution of DBPs exposure from consuming the poultry is minimal and does not cause significant risk to cancer or other health conditions.

The current scientific evidence on the association of residual levels of DBPs with cancer does not warrant changing the guidelines of 50 ppm chlorine for chiller water treatment in poultry processing as the application is essential to reducing pathogen load and controlling cross contamination. It is important to emphasize that public health gains from reduced waterborne and foodborne illnesses using chlorine in poultry processing far outweigh the risks from cancer.

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