United States Department of Agriculture
Agricultural Marketing Service | National Organic Program
Document Cover Sheet
https://www.ams.usda.gov/rules-regulations/organic/petitioned-substances

Document Type:

☒ **National List Petition or Petition Update**

A petition is a request to amend the USDA National Organic Program's National List of Allowed and Prohibited Substances (National List).

Any person may submit a petition to have a substance evaluated by the National Organic Standards Board (7 CFR 205.607(a)).

Guidelines for submitting a petition are available in the NOP Handbook as NOP 3011, National List Petition Guidelines.

Petitions are posted for the public on the NOP website for Petitioned Substances.

☐ **Technical Report**

A technical report is developed in response to a petition to amend the National List. Reports are also developed to assist in the review of substances that are already on the National List.

Technical reports are completed by third-party contractors and are available to the public on the NOP website for Petitioned Substances.

Contractor names and dates completed are available in the report.

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Attn: Jared Clark, Standards Division

Subject: "Chlorine materials" annotation change at 205.603(a)(10)

Introduction

A clarification on the use of chlorine materials in USDA organic livestock production is sought. Specifically, it is unclear whether chlorine materials, those allowed at 205.603(a)(10) on the National List, are allowed as direct livestock drinking water treatments.

In our work to resolve this issue, we spoke to many organic certifiers and worked with our Advisory Council. Both outreach activities yielded mixed results. Some believe, and have established policies, that the National List at 205.603(a)(10) allows for direct livestock drinking water treatments as long as the final drinking water meets Safe Drinking Water Act (SDWA) standards. Others read the 205.603(a)(10) annotation limits chlorine materials use to facilities and equipment disinfection and sanitization only.

Chlorine materials appeared on the original National List and have been renewed in the sunset review process since, although sunset votes were deferred in 2005 and 2010 so that the NOSB could receive additional technical assistance on these materials.

Petition

A.1. Section(s) of the National List

7 CFR 205.603(a)(10) entry for "Chlorine materials".

Chlorine materials—disinfecting and sanitizing facilities and equipment. Residual chlorine levels in the water shall not exceed the maximum residual disinfectant limit under the Safe Drinking Water Act.

A.2. OFPA Category - Crop and Livestock Materials

Production aid.

A.3. Inert Ingredients

Chlorine is not an inert ingredient.

B - Substance Information

We defer to the 2006 USDA "Chlorine/Bleach" technical report.

B.1. Substance Name

Four substances appear as allowed synthetic "chlorine materials" at 205.603(a)(10):

(i) Calcium hypochlorite.
(ii) Chlorine dioxide.
(iii) Hypochlorous acid—generated from electrolyzed water.
(iv) Sodium hypochlorite.

B.2. Petitioner and Manufacturer Information

The Organic Materials Review Institute (OMRI)
P.O.Box 11558
Eugene, Or 97440-3758

B.3. Intended or Current Use

According to the 205.603(a)(10) annotation, chlorine materials are allowed in organic livestock production for "*disinfecting and sanitizing facilities and equipment."* The annotation also specifies that "*Residual chlorine levels in the water shall not exceed the maximum residual disinfectant limit under the Safe Drinking Water Act."* Which uses this annotation is intending to allow is the matter under petition.

NOP Guidance 5026 further clarifies that "Residual chlorine levels in the water in direct food or animal contact (for example, drinking water) should not exceed the maximum residual disinfection level." It is unclear if the reference to drinking water is attempting to add, clarify or limit the use of chlorine as a livestock drinking water treatment.

B.4. Intended Activities and Application Rate

N/A

B.5. Manufacturing Process

See 2006 USDA "Chlorine/Bleach" technical report.

B.6. Ancillary Substances

See 2006 USDA "Chlorine/Bleach" technical report.

B.7. Previous Reviews

See the NOP Petitioned Substances Database entry "Chlorine Materials-Livestock" for a summary of previous reviews by the USDA National Organic Program (NOP) and the NOSB. The NOSB Processing Subcommittee document called *Measuring Effluent: Clarification of Chlorine Contact with Organic Food* (2003) provides a good historical summary of previous reviews.

B.8. Regulatory Authority

The regulatory authority over chlorine use is many and varied. In livestock production, the US Food and Drug Administration (FDA) exercises a substantial amount of regulatory authority over chlorine use. The US Environmental Protection Agency (EPA) also exercises regulatory authority over chlorine material use. See 2006 USDA "Chlorine/Bleach" technical report for more information.

In addition to the regulatory authority explored in the 2006 USDA technical report, the *Pasteurized Milk Ordinance* (PMO) is also an important authority, albeit not a "regulatory authority", on the use of chlorine materials in livestock production. Both FDA and U.S. Department of Health and Human Services (DHHS) endorse the PMO as the minimum standard to which many local and state regulators use when establishing standards for dairy producers. The requirements for chlorine use in dairies are included in the PMO, including those that give instructions for direct water sanitization measures such as "well shocking".

B.9. Chemical Abstracts Service (CAS) Number and Product Labels

See 2006 USDA "Chlorine/Bleach" technical report.

B.10. Physical and Chemical Properties

See 2006 USDA "Chlorine/Bleach" technical report.

B.11. Safety Information

See 2006 USDA "Chlorine/Bleach" technical report.

B.12. Research Information

The research information we are highlighting in this petition summarizes the historical approach to chlorine materials in USDA organic production. The original USDA Technical Advisory Panel (TAP) reports describe chlorine materials, including their use in livestock production. These TAP reports are accessible within the *Chlorine Materials-Livestock* entry in the NOP Petitioned Substances Database. The aforementioned 2006 USDA technical report on these materials is also available. Public comments received by USDA over almost 30 years are also available in addition to the NOSB meeting notes from all meetings in which these materials were discussed.

*NOP Guidance 5026: The Use of Chlorine Materials in Organic Production and Handling (*2011, updated 2024) and *NOP Notice 11-7 Issuance of Final Guidance and Response to Comments* (2011, updated 2024) are both published in the NOP's Program Handbook. *NOP 5023* provides the following definition for the term "facility", but it is unclear whether this definition should be used when considering the 205.603(a)(10) use restriction. NOP 5023 was published long after the term "facility" was included in annotation language for the 205.603(a)(10) "Chlorine materials" entry.

Facility. A structure or site where production, handling, processing, packaging or storage of organic products occurs. A facility could include packing lines, wash lines, storage units, coolers, freezing plants, feed mills, milk houses, production structures such as housing for livestock, greenhouses and mushroom buildings.

NOP Notice 11-7 introduces the term "direct use" which is a different term than the terms "equipment" and "facility" which are used in the 205.603(a)(10) "Chlorine materials" annotation. In *NOP Notice 11-7,* drinking water treatment is identified as a "direct use" and dairy pipelines would be a "facility use". The notice, however, does not go further to clarify whether drinking water treatment is an "equipment" use or a "facility" use or both.

The NOSB Processing Subcommittee document called *Measuring Effluent: Clarification of Chlorine Contact with Organic Food* (2003) also address the use of chlorine materials in USDA organic livestock production and provides a good historical summary of the regulatory status of this material.

B.13. Petition Justification Statement

We believe an annotation revision is needed to clarify whether chlorine materials are allowed for direct treatment of livestock drinking water. Therefore, this petition requests an amendment to the 205.603(a)(10) annotation for "Chlorine materials".

If the intention is to allow chlorine materials for use as a direct livestock drinking water treatment, the following annotation change is petitioned (<u>underline</u> shows addition, ~~strikethrough~~ shows deletion):

- Chlorine materials—disinfecting and sanitizing facilities<u>,</u> ~~and~~ equipment<u>, and livestock drinking water</u>. Residual chlorine levels in the water shall not exceed the maximum residual disinfectant limit under the Safe Drinking Water Act.

If the intention is to prohibit the use of chlorine materials as direct livestock drinking water treatments, the following annotation change is petitioned:

- Chlorine materials—disinfecting and sanitizing facilities and equipment. Residual chlorine levels in the water shall not exceed the maximum residual disinfectant limit under the Safe Drinking Water Act. Prohibited for use as a direct livestock drinking water treatment.

A plain language reading of the current annotation can leave stakeholders believing that chlorine materials may only be used to disinfect or sanitize facilities and equipment and are not allowed for direct treatment of livestock drinking water. Examples of facilities and equipment could include components of milking parlor infrastructure such as flooring, pipes, and tanks, as well as drinking water infrastructure such as water lines and stock tanks.

Another perspective is that 205.239(a)(1) requires clean water for livestock and that NOP 5026 [Section 4.2 (1)] could be read as clarifying that chlorine materials are allowed for livestock drinking water treatments if the resulting water meets SDWA.

Water sources should be evaluated for reliability and water quality. When supplied via piping, waterers, and troughs, well water can be safe from the impacts of drought, muddy floodwaters, and toxic algae blooms, although regular cleaning of this equipment is necessary (https://extension.uga.edu/publications/detail.html?number=C1264&title=maintaining-a-clean-water-trough-for-cattle). Municipal water supplies are not available to all livestock operations. Wells are common in many rural areas where livestock are raised. Wells can become contaminated by a variety of factors, including flood and proximity to biological contaminants (https://extension.arizona.edu/sites/extension.arizona.edu/files/pubs/az1605.pdf).
Shock chlorination is a common practice to address well water contaminants. Additionally, application of chlorine-based water sanitizer in cases where sub-optimal microbial water quality is a concern on a farm may be an element considered as part of a comprehensive water sanitation program https://www.sciencedirect.com/science/article/pii/S1056617124000242).

Finally, there are other materials that appear on the National List that may be used as livestock drinking water treatments, including iodine and hydrogen peroxide. (https://www.ams.usda.gov/sites/default/files/media/Iodine%20TR%202015.pdf, https://www.ams.usda.gov/sites/default/files/media/Hydrogen%20Peroxide%203%20TR%202015.pdf).

Taking into consideration everything presented, how should we interpret the 205.603(a)(10) annotation?

The potential of the substance for detrimental chemical interactions with other materials used in organic farming systems: see Evaluation Question #6 in the 2006 USDA "Chlorine/Bleach" technical report for further details. Relevant excerpts from this report are included here:

"There is little chance, however, for the bleach materials to migrate from the equipment/facilities to crops or fields unless wastewater from the equipment/facilities were recycled in irrigation or the bleach materials were misused or accidentally spilled. The potential for bleach materials to detrimentally affect other substances used in organic crop or livestock production depends on the concentrations of the chemicals and their breakdown products in irrigation water discharged from treated systems. No information is currently available on the post-treatment concentrations of these chemicals."

The toxicity and mode of action of the substance and of its breakdown products or any contaminants, and their persistence and areas of concentration in the environment: see Evaluation Question #5 in the 2006 USDA "Chlorine/Bleach" technical report for further details. Relevant excerpts from this report are included here:

Sodium and Calcium Hypochlorite

"Although sodium and calcium hypochlorite are low in toxicity to avian wildlife, they are highly toxic to freshwater fish and invertebrates. Discharges of hypochlorite-containing wastes from facilities (i.e., point sources) are regulated through

issuance of site-specific wastewater discharge permits intended to ensure that the amount of hypochlorites discharged will not pose a significant adverse effect to wildlife (EPA,1991). Additionally, current NOSB approval is conditioned on residual chlorine levels in the water not exceeding the limit set by the Safe Drinking Water Act (4 mg/L)."

Chlorine Dioxide

"Chlorine dioxide is a very reactive compound and breaks down quickly in the environment (ATSDR, 2004a). In air, sunlight rapidly causes chlorine dioxide to break down into chlorine gas and oxygen. When used as a disinfecting agent, however, the product of chlorine dioxide is primarily chlorite. Although chlorite in water may move into groundwater, reactions with soil and sediments may reduce the amount of chlorite reaching groundwater. The toxic action of chlorite is primarily in the form of oxidative damage to red blood cells at doses as low as 10 mg/kg of body weight. Toxic reaction products are not known to occur when chlorite is mixed with organic materials. EPA has set a maximum contaminant level (MCL) of 0.8 mg/L for chlorine dioxide in drinking water and 1 mg/L for chlorite (EPA, 2002)."

The probability of environmental contamination during manufacture, use, misuse or disposal of the substance: see Evaluation Question #4 in the 2006 USDA "Chlorine/Bleach" technical report for further details. Relevant excerpts from this report are included here:

Chlorine Dioxide

"…during the "activation" of chlorine dioxide (i.e., activating dilute aqueous solutions of sodium chlorite with an acid to produce chlorine dioxide), the release of gas to the air or "off gassing" can be a safety hazard to users.

According to ATSDR (2004b), chlorine dioxide has not been found at any of the 1,647 current or former National Priorities List (NPL) sites that are targeted by EPA for long-term federal clean-up activities.

No information was found in the literature on concentrations of chlorine dioxide in air, sediments, or soil. In sediments and soil, concentrations of chlorine dioxide are expected to be small or not detectable due to its high reactivity (ATSDR, 2004b).

Chlorine dioxide contamination in water is difficult to identify because it is intentionally added to drinking water as a disinfectant in some municipal water-treatment systems. EPA has set a maximum contaminant level (MCL) of 0.8 mg/L for chlorine dioxide in drinking water and 1 mg/L for chlorite (EPA, 2002). Levels of chlorite ion were sampled from drinking water distribution systems of publicly owned treatment works (POTW) facilities that utilized chlorine dioxide in the United States as part of the Information Collection Rule (ICR) in 1998; approximately 16 percent had levels of chlorite ion over the MCL of 1 mg/L (ATSDR, 2004b)."

The effect of the substance on human health: see Evaluation Question #11 in the 2006 USDA "Chlorine/Bleach" technical report for further details. Relevant excerpts from this report are included here:

Calcium Hypochlorite or Sodium Hypochlorite

"Potential human health effects due to calcium hypochlorite or sodium hypochlorite use as a disinfecting and/or sanitizing agent for livestock facilities and/or equipment occur dermally or via inhalation…Long-term exposure to low levels of hypochlorite can cause dermal irritation. Inhalation of chlorine gas released from concentrated hypochlorite solutions may cause nasal irritation, sore throat, and coughing…"

Chlorine Dioxide

"Inhalation and dermal exposure are the main routes of concern for human exposure when chlorine dioxide is used as a disinfecting and/or sanitizing agent for livestock facilities and/or equipment. Chlorine dioxide is a severe respiratory and

eye irritant. According to the Occupational Safety and Health Administration (OSHA), inhalation can produce coughing, wheezing, respiratory distress, and congestion in the lungs. Irritating effects in humans were intense at concentration levels of 5 ppm. OSHA has set a limit of 0.1 parts of chlorine dioxide or chlorite per million parts of air (0.1 ppm) in the workplace during an 8-hour shift, 40-hour workweek…"

Additional literature on human health effects are well documented in Guidelines for Canadian Drinking Water Quality: Guideline Technical Document – Chlorine, particularly Part II. Science and Technical Considerations (https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-chlorine-guideline-technical-document/page-3-guidelines-canadian-drinking-water-quality-chlorine-guideline-technical-document.html).

The effects of the substance on biological and chemical interactions in the agroecosystem, including the physiological effects of the substance on soil organisms (including the salt index and solubility of the soil), crops, and livestock: see Evaluation Question #8 in the 2006 USDA "Chlorine/Bleach" technical report for further details. Relevant excerpts from this report are included here:

"No information sources reviewed for this report described or evaluated potential detrimental physiological effects on soil organism, crops, or livestock when bleach materials (i.e., calcium hypochlorite, sodium hypochlorite, or chlorine dioxide) are used as a disinfecting and/or sanitizing agent for livestock facilities and/or equipment. It is unlikely that bleach materials would cause such effects unless misused or accidentally spilled."

However, this is an area of continued research interest. It is known that high levels of water chlorination may be applicable and even necessary in organic livestock situations. Research acknowledges though that excess chlorine may have different impacts depending on class of animals. For instance, high levels of chlorine in water may affect the efficiency of the rumen microbial population and subsequently metabolic impairment of rumen function may occur in ruminant livestock. Alternatively, monogastric livestock will likely be less affected by direct effects of chlorine, and most affected by pathogens in drinking water, so risk-benefit analysis would suggest that more aggressive water disinfection may be beneficial in this class of farm animals in situations where risk of bacterial contamination is high. However, more research is needed to determine appropriate levels of chlorine for different types of livestock (https://www.ag.ndsu.edu/waterquality/livestock/Livestock_Water_QualityFINALweb.pdf).

The alternatives to using the substance in terms of practices or other available materials; and
Its compatibility with a system of sustainable agriculture: see Evaluation Question #13 and #14 in the 2006 USDA "Chlorine/Bleach" technical report for further details. Relevant excerpts from this report are included here:

Evaluation Question #13: *Hydrogen Peroxide*

"…Hydrogen peroxide is registered for use in dairy/cheese processing plants, on food processing equipment, and in pasteurizers in breweries, wineries, and beverage plants (EPA, 2003b). Unlike other chemical substance, hydrogen peroxide does not produce residues or gasses; however, high concentrations of hydrogen peroxide are required for disinfection. Additionally, hydrogen peroxide reacts with numerous substances and slowly decomposes into water and oxygen."

Evaluation Question #14: *UV*

"UV radiation (generated from a special lamp) effectively destroys bacteria and viruses. A secondary disinfectant must be used to prevent regrowth of microorganisms. UV radiation can be attractive as a primary disinfectant for small systems because it is readily available, it produces no known toxic residuals, it requires short contact times, and the equipment is easy to operate and maintain."

Iodine

Additionally, iodine appears on the National List with no annotation. Iodine, like chlorine, kills most disease-causing organisms and requires short to moderate contact times (https://www.gov.mb.ca/health/publichealth/factsheets/devices.pdf). However, iodine is not very effective against biofilms https://poultry-science.uark.edu/_resources/pdf/AvianAdvice_Sept2017.pdf). Iodine should also not be used for long-term continuous disinfection because it is physiologically active and ingestion in excess may be harmful (https://www.gov.mb.ca/health/publichealth/factsheets/devices.pdf).

Hydrogen Peroxide

Hydrogen peroxide also appears on the National List with no annotation and is an oxidizer like chlorine. Furthermore, it is another common disinfectant used for drinking water treatment (https://poultry-science.uark.edu/_resources/pdf/avianadvice_spr09.pdf), in addition to its uses as a sanitizer for tools and equipment.

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Maintaining a Clean Water Trough for Cattle

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Water is one of the most important parts of cattle diets. It is essential for digestion, thermoregulation, growth, reproduction, and circulatory and nervous system functions. Adult cattle need 8 to 20 gallons of water per day, depending on size, diet, status, and weather. Research shows that unrestricted access to clean water improves feed intake and average daily weight gains, increases milk production, and decreases illness and disease. On the other hand, restricted access to water and poor water quality negatively impact cattle production and can potentially cause illness and death. Bad odor and taste from water sources contaminated with high amounts of minerals, salt, nitrogen, bacteria, algae, or manure likely will keep cattle from drinking enough water and can cause significant health risks or death. Water sources can become contaminated or polluted by livestock animals, wildlife, local hydrology, or soil and bedrock features. To keep drinking water supplies clean and consistently available for cattle, consider the available water sources, how to exclude wildlife, how cattle will access water and its location, and trough cleaning methods.

Water Sources

Water sources should be evaluated for reliability and water quality. Before initial use, the water source should be tested for nitrates, dissolved solids, salts, pH, and fecal coliform bacteria. It is important to ensure the water source is sufficient to meet the demands of livestock, especially in times of drought and warmer weather. Surface waters, including streams, ponds, lakes, and springs, have long been a popular water source for cattle producers. Wells that access groundwater sources also are used to keep cattle hydrated. When supplied to troughs, well water can be safe from the impacts of drought, muddy floodwaters, and toxic algae blooms, although regular trough cleaning is necessary (Figure 1). Wells use pumps that require a continuous supply of electric power. During a power outage, backup water sources such as streams and ponds provide insurance against water supplies running low. Keeping large tanks filled also buys time while addressing electrical failures. Municipal water supplies may provide more consistent water quality but could incur higher operational costs.

Figure 1. Troughs in corrals need more frequent cleaning.

Excluding Wildlife

Some water sources can be attractive to wildlife seeking clean water, particularly in times of drought and heat stress. Wildlife conflict is most common when using water troughs, and steps should be taken to minimize wildlife's impact on the safety of cattle drinking–water supplies. Smaller animals such as birds and rodents may get trapped in troughs and drown. These animals can pollute water from feces or urine in addition to the animal carcass and may introduce harmful pathogens such as Leptospira. These contaminants cause a decrease in cattle water intake, feed consumption, and weight gains. While it's difficult to completely exclude wildlife from accessing water sources, producers can install escape ramps (Figures 2 and 3) that provide a means for trapped animals to exit water troughs. Keeping open troughs completely empty when not in use can prevent wildlife from drowning, and empty tanks will deter wildlife activity. Closed–ball watering systems (Figure 4) are one way to prevent wildlife from disturbing cattle drinking water.

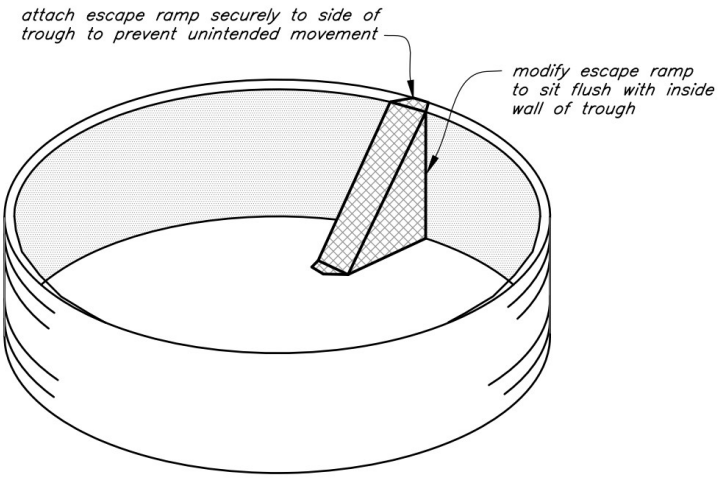

Figure 2. Diagram of a wildlife escape ramp. Source: USDA Natural Resource Conservation Service.

Figure 3. A wildlife escape ramp installed in a water tank. Photo: Kelly Melton.

Water Access and Location

Planning water access and location is essential when constructing new livestock facilities or renovating older operations. Cattle always need access to adequate clean drinking water, and a variety of water sources and locations may be required to accomplish this goal. Water troughs should be located at least 150 ft away from feeding bunks or hay feeding areas to avoid contamination from feed debris. Spreading apart the feed and water areas also increases distribution of manure throughout the pasture. Since water troughs are high–use areas, placing the trough on an elevated concrete pad can minimize fecal contamination from manure. Installing a heavy–use gravel pad around the trough also can reduce hoof damage to cattle from standing on poorly drained surfaces.

Figure 4. Closed–ball watering system.

Cattle prefer drinking water that is between 40–77 °F, and their intake declines when water temperatures rise over 80 °F. Shallow water sources and those placed in direct sunlight are more likely to heat up in hot weather and lead to decreased consumption or increased algae growth. Static trough water also will heat up more quickly than groundwater that is pumped into larger tanks that automatically refill. Placing troughs within tree–shaded areas or using closed–ball waterers also can reduce sun exposure and keep water temperature within an ideal range. Be sure to consider any overhanging vegetation that may drop leaves or other materials into open–water troughs (Figure 5).

Cleaning Methods

Water troughs are a common means of providing adequate hydration to a herd and can have positive impacts on herd performance compared to surface water sources. However, whether the water is supplied from a well or pumped from a stream, spring, or pond, water troughs easily become contaminated with sediment and bacteria. Producers should clean out physical debris regularly, and chemically disinfect troughs at least two times per year. More frequent cleaning might be required during periods of heavy use or when significant amounts of debris have accumulated. Targeted cleaning during the late spring and summer months will help keep bacteria levels down.

Figure 5. Example of an open water trough.

When checking the condition of a trough:

- Water should never be colored or murky.
- Algae mats should not fully cover the surface or container walls.
- There should be no noticeable odors, particularly those of sewage, rotten eggs, mold, or animal waste.

Such conditions can indicate dirty water and require a trough cleaning. However, not all water contamination is visible. Dissolved salts, high or low pH, minerals, and metals may not produce visible effects. Water should be tested initially to check the chemical properties of new water sources and annually for fecal coliform bacteria contamination.

To thoroughly clean a water trough:

1. Empty it completely and remove all debris.
2. Rinse the tank twice with a 10% bleach solution (1 part bleach to 9 parts water, or about 1.5 cups bleach in 1 gallon of water).
3. Let the bleach solution contact the tank surfaces for 15 min.
4. Rinse the tank twice more with clean water.
5. Refill the tank.

In addition, 8 oz of household bleach per 1,000 gallons of water can be added when refilling the tank. This results in a 3 ppm concentration of chlorine in the water, which is safe for cattle to drink and helps control algal and bacterial growth in the water. Bleach can be added again after each total volume turnover, based on the cattle's drinking rate. For example, 20 cows that drink 15 gallons per day solely from an autofilled 1,000–gallon tank would turn over the volume in 3 days. Table 1 shows typical daily water intake by beef cattle under different ambient air temperatures.

Adding bleach at a greater concentration could risk creating high chlorine contamination levels and deter cattle from drinking. Unscented regular household bleach (5–6% concentration; no highly concentrated solutions, pastes or gels) should be used for these ratios.

Table 1. Beef cattle water-intake estimates.

Daily water intake estimates (gallons) at air temperature						
	40 °F	50 °F	60 °F	70 °F	80 °F	90 °F
Weight, lb	**Growing beef calves**					
400	4	4.3	5	5.8	6.7	9.5
600	5.3	5.8	6.5	7.8	8.9	12.7
800	6.3	6.8	7.9	9.2	10.6	15
	Finishing cattle					
600	6	6.5	7.4	8.7	10	14.3
800	7.3	7.9	9.1	10.7	12.3	17.4
1,000	8.7	9.4	10.8	12.6	14.5	20.6
	Pregnant cows					
900	6.7	7.2	8.3	9.7	no data	no data
	Mature bulls					
1,400	8	8.6	9.9	11.7	13.4	19
1,600+	8.7	9.4	10.8	12.6	14.5	20.6

Note. Adapted from *Nutrient Requirements of Beef Cattle* (8th ed.) by National Academies of Sciences, Engineering, and Medicine, 2016 (https://doi.org/10.17226/19014). Copyright 2016 by the National Academies Press.

Summary

Keeping a clean trough and tank is essential for maintaining water palatability and intake, reducing disease and pathogen risk, and contributing to overall performance of cattle. Cleaning and disinfecting troughs will help maintain safe water sources for cattle regardless of any particular system design. Water testing and monitoring for potential contaminants at the water source is highly recommended. Your local University of Georgia Cooperative Extension agent can provide guidance on water testing, treating well water with chlorine bleach (i.e., shocking), and addressing other common water quality issues, such as mineral contamination and algae growth.

References

Bicudo, J. R., Agouridis, C. T., Workman, S. R., Gates, R. S., & Vanzant, E. S. (2003, July 27–30). *Effects of air and water temperature, and stream access on grazing cattle water intake rates* [Paper presentation, Paper No. 034034]. American Society of Agricultural and Biological Engineers Annual International Meeting, Las Vegas, NV, United States. https://doi.org/10.13031/2013.15027

Brantley, E., Mullinex, K., Marks, L., & Stanford, K. (2019). *Keeping it clean: Livestock water tank maintenance* (Publication No. ANR-2583). Alabama Cooperative Extension System. https://www.aces.edu/blog/topics/beef/keeping-it-clean-livestock-water-tank-maintenance/

Dyer, T. G. (2017). *Water requirements and quality issues for cattle* (Publication No. SB 56). University of Georgia Cooperative Extension. https://extension.uga.edu/publications/detail.html?number=SB56

Higgins, S. F., Agouridis, C. T., & Gumbert, A. A. (2008). *Drinking water quality guidelines for cattle* (Publication No. ID-170). University of Kentucky Cooperative Extension Service. http://www2.ca.uky.edu/agcomm/pubs/id/id170/id170.pdf

Higgins, S. F., Moser, L., & Laurent, K. (2016). *Providing water for beef cattle in rotational grazing systems* (Publication ID-236). University of Kentucky Cooperative Extension Service. http://www2.ca.uky.edu/agcomm/pubs/ID/ID236/ID236.pdf

Lardner, H. A., Kirychuk, B. D., Braul, L., Willms, W. D., & Yarotski, J. (2005). *The effect of water quality on cattle performance on pasture.* Australian Journal of Agricultural Research, 56(1), 97–104. https://doi.org/10.1071/AR04086

National Academies of Sciences, Engineering, and Medicine. (2016). *Nutrient Requirements of Beef Cattle* (8th ed.). The National Academies Press. https://doi.org/10.17226/19014

Shirley, R. L. (1974). *Nutrients and toxic substances in water for livestock and poultry – A report of the Subcommittee on Nutrient and Toxic Elements in Water, Committee on Animal Nutrition, Board on Agriculture and Renewable Resources, Commission on Natural Resources, National Research Council.* National Academy of Sciences.

Oregon Natural Resources Conservation Service. (2012). *Wildlife escape ramps for livestock watering troughs.* United States Department of Agriculture. https://www.nrcs.usda.gov/Internet/FSE_DOCUMENTS/nrcs142p2_041023.pdf

Parish, J. A., Rinehart, J. D., & Karish, B. (2019). *Beef cattle water requirements and source management* (Publication No. P2490). Mississippi State University Extension Service. http://extension.msstate.edu/publications/publications/beef-cattle-water-requirements-and-source-management

Willms, W. D., Kenzie, O. R., McAllister, T. A., Colwell, D., Veira, D., Wilmshurst, J. F., Entz, T., & Olson, M. E. (2002). Effects of water quality on cattle performance. *Journal of Range Management, 55*(5), 452–460. http://dx.doi.org/10.2307/4003222

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THE UNIVERSITY OF ARIZONA
COLLEGE OF AGRICULTURE AND LIFE SCIENCES
COOPERATIVE EXTENSION

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What Well Owners Should Know about Shock Chlorination

Why it works for some well problems and not for others

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Pouring Bleach Down Your Well? Read this Publication First

Introduction

The intended purpose of shock chlorination is to reduce the levels of microorganisms that can cause illnesses (pathogens) present in water using a concentrated liquid chlorine solution. There are two places where this disinfection treatment method is typically applied: down the water well itself and/or inside water storage tanks. The practical benefits of shock chlorination are different for each application, as are the risks.

The purpose of this publication is to make well owners aware of the benefits and potential problems associated with water well shock chlorination, including when and who should do it.

A reason often cited for shock chlorinating a well is that it has tested positive for total coliform bacteria (commonly found throughout the environment), which is not a health threat in itself, and/or positive for fecal coliform bacteria, i.e. *E. coli* bacteria, which can indicate a health threat since they are more frequently associated with fecal contamination (USEPA, 2013). These tests indicate the presence or absence of harmless coliform bacteria and only imply the likely presence of potentially harmful organisms (pathogens) in the water.

Natural waters contain materials such as salts, metals, and nutrients that provide an ideal medium for many types of organisms including algae and bacteria to grow. Many organisms found in water sources are not necessary harmful when ingested and form part of a complex network of naturally occurring organisms. Unfortunately, human or animal wastes can quickly degrade natural waters including groundwater, adding pathogens such as intestinal bacteria, viruses, and even parasites. The U.S. Environmental Protection Agency (USEPA, 2013) considers that there is no safe level of any pathogen in drinking water.

Shock chlorination typically uses a strong bleach solution, which can kill most microorganisms it comes into contact. Bleach can also react with well components and naturally occurring chemicals found in groundwater aquifers with unpredictable results. For this reason, private well owners should use caution when attempting to disinfect their water well using common household bleach for shock chlorination.

Water Well Components

The four basic components of a well are a well casing (protects the bore hole) with a screen (where the water enters the well) and a drop pipe with a submersible pump (to extract

water from the aquifer). A working well has the surfaces of these components fully or partially in contact with water (submerged) most of the time. These surfaces, which are either plastic (PVC) or steel (low carbon, galvanized, or stainless steel), will interact with chemicals and microorganisms found in the soil and in the groundwater (see next section).

Typical Composition of Groundwater

Wells are designed to extract water from aquifers that contain water with varying amounts of dissolved inorganic (salts, metals, nutrients, etc.) and organic (carbon-based) chemicals, and living microorganisms such as bacteria. Many chemicals and microorganisms are naturally occurring in soils and aquifers. Common chemicals found in all natural waters include: sodium, calcium, chloride, sulfate, nitrate, phosphate, and organics made up of plant and animal residues (this group of chemicals is also known as dissolved organic matter or dissolved organic carbon). Common bacteria found in all waters include coliforms. See Extension publication AZ1578 (Artiola et al., 2012) for a detailed discussion of the types of chemicals and common contaminants present in water.

Deep groundwater aquifers are not easily impacted by human activities such as the introduction of industrial wastes, wastewaters, or direct surface recharge. Deep aquifers typically contain low concentrations of the nutrients necessary for bacteria and other organisms to grow. Therefore, normally, there are fewer living microorganisms present in deep compared to shallow aquifers since microorganisms grow slowly and compete for nutrients in this nutrient starved environment (BSIa, 2013).

Installing a well into an aquifer and then pumping water changes the delicate balance (dynamics) between naturally occurring organisms and nutrients in the groundwater. As pumping starts, the pump suction creates a funneling effect that draws water through the screen openings into the well much faster than water that moves through the rest of the aquifer. This means that any organisms attached to the well components are able to harvest more nutrients since fresh, nutrient-rich water passes by them more frequently. In addition, the opening into the aquifer (the well casing) brings oxygen directly to the well water, which also promotes the growth of oxygen-loving organisms. Thus, in this artificially created environment naturally occurring bacteria and other organisms are able to thrive (BSIa, 2013).

As microorganisms grow in numbers, they form groups and associations (consortia) that look and feel like slime on the surfaces of well components and on the insides of both pressure and storage tanks.

Slime (Biofilm) Formation

There is evidence that layers of slime begin to form quickly (hours to weeks) on surfaces exposed to moisture or submerged in water (Mittelman, 1985), see Figure 2.

The process of slime formation starts with the exposure of well components to dissolved organic chemicals that attach to their surfaces, see Figure 3.

Figure 2. Louvered well screen covered with slime within days of being installed in new water well. Photo by G. Hix, 2012.

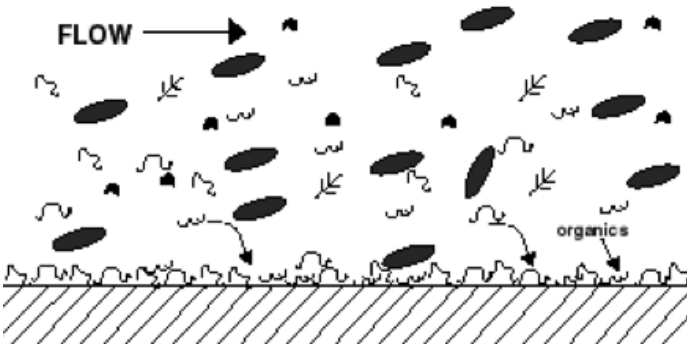

Figure 3. Water-soluble organic chemicals (strings) and bacteria (oval shapes) present in aquifer waters attach to well components. Source: Characklis & Marshall, 1990.

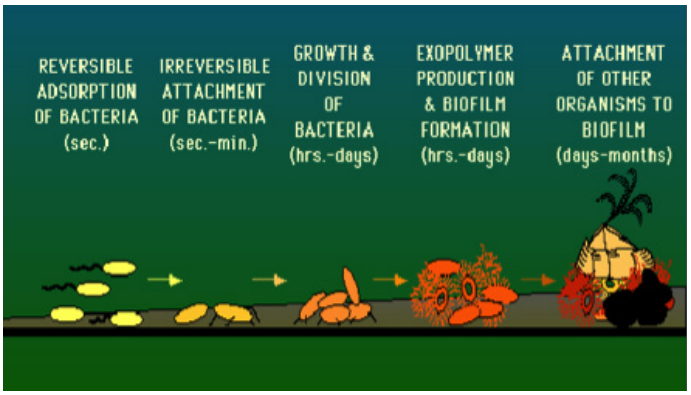

Figure 4. Steps to slime formation on wet or submerged surfaces. Source: K. Todar, U. Wisconsin.

Soon after, bacteria looking for food land on these surfaces and eventually attach themselves there permanently.

Slime is produced by anchored bacteria that excrete long strands of chained sugar molecules (polysaccharides), which form a sticky mat that covers to the surfaces of well components (BSIa, 2013). The slime is formed of layers and strands that are porous. This multistep process is shown in Figure 4.

Slime can also filter and concentrate nutrients (such as nitrate and phosphate) from the water that are used by organisms living in the slime layer to grow and multiply and coat well and water system components. Therefore, once formed, slime provides an ideal environment for many organisms, good and bad (pathogens) to exist inside wells, tanks, and even pipes used to produce, store, and transport water.

Is Slime Beneficial?

We have shown why and how slime is produced on well components and we have described how the well environment changes the natural chemical composition of aquifer water in ways that favor the rapid growth of microorganisms that attach to well surfaces. Once attached, microorganisms form consortia. As these grow, they begin to disperse bacteria that act as scouting and pioneering groups that grow in places outside the well casing and screens that are virtually impossible to reach, making them difficult to remove with shock chlorination.

Research indicates that slime (biofilm) is made up of a highly complex and collaborative group of many species of organisms (mostly bacteria) that interact in many different ways. For example, waste produced by one species may be used by another species as food. Several species of bacteria may collaborate to breakdown a food source using different methods (enzymes) for the benefit of all.

The slime itself and organisms that live inside may also filter and degrade (use as food) contaminants such as nutrients (previously discussed), organic residues, and some salts and metals, **making the water safer to drink.** However, excessive slime growth can be detrimental to well performance and components and facilitate the growth of pathogens and other unwanted organisms as shown in the next sections.

Iron, Oxygen, Corrosion, and Slime

Many Arizona aquifers often contain some dissolved oxygen and very low levels of dissolved iron. But some shallow aquifers, regularly impacted by surface recharge, have high concentrations of dissolved organic matter. These conditions can increase microbial activity, depleting oxygen quickly and increasing the levels of soluble iron in the water. When iron-rich and oxygen-poor water from the aquifer is drawn into the well, oxygen is mixed into the water, which together with the presence of iron oxidizing bacteria (see Figure 5) produces a reddish slime (hydrated iron oxides) and favors the growth of iron-loving bacteria that produce well damaging red slime, see Figure 6.

These red residues and red slime can form and deposit in storage tanks, toilets, and sinks, giving water a yellow to red color, often with a musty, swampy smell. Excessive growth (also known as biofouling) of these iron bacteria can clog pipes and shut down wells (BSIb, 2013).

When well water stagnates for several days, the oxygen dissolved in the water is depleted (microbes use it up to grow) and organic matter (in the form of microbes) accumulates. Under these conditions, if iron-reducing bacteria are present,

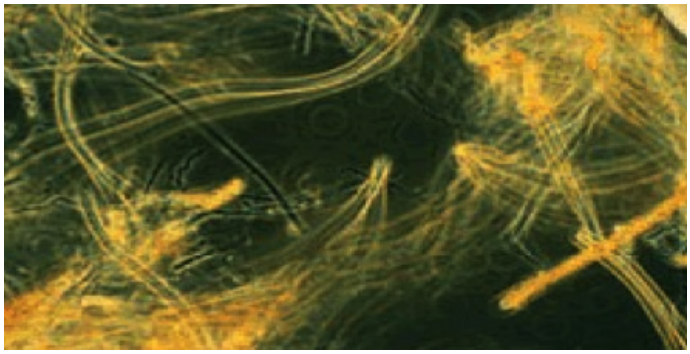

Figure 5. Example of a filament-producing bacterium (Leptohrix) that produces red slime. Source: BSIb, 2013.

Figure 6. A well pump intake screen covered with red slime and iron rust formed by bacteria living inside a pumping well. Photo by G. Hix.

they become active and start to use oxygen from the iron (rust), which re-dissolves iron in the water. When this happens, the typical red colors in the water and walls begin to disappear. If these conditions are prolonged, sulfur-reducing bacteria may begin to thrive. These bacteria also steal oxygen but from sulfates (commonly found in water) producing a highly toxic, corrosive, rotten-egg smelling gas called hydrogen sulfide. This gas dissolves in water and reacts with soluble iron producing a black residue that coats surfaces.

Research also indicates that sulfur-reducing bacteria prefer to grow deep inside the slime layers where oxygen gas levels tend to be very low (Edstrom, 2004). Thus, when these bacteria produce hydrogen sulfide, the gas can move back into an oxygen-rich zone, re-oxidize, and form sulfuric acid that can corrode iron surfaces such as metal casings and screens.

Sediments, Pathogens, Slime, and Chlorine

Although slime is mostly composed of living organisms and organic residues, it may also filter out and trap inorganic materials such as rust particles (produced during the oxidation of iron components) and aquifer materials such as sand, silt, and clay particles. Slime can trap aquifer materials as they enter the well and facilitate the formation of mineral deposits (encrustations) on the small screen slits that can progressively lower the well yield.

Figure 7. Wire wrapped well screen plugged with slime and mineral deposits. Photo by G. Hix.

Figure 8. A water well pump encrusted with iron rust tubercles. Photo by G. Hix.

Excessive accumulation of slime and mineral deposits on a well screen can reduce water flow and lower well yields as shown in Figure 7. These mineral deposits are often calcareous (calcium carbonate) in composition and are not affected or removed by shock chlorination.

When bleach (liquid chlorine--5.25% sodium hypochlorite solution) is poured down a water well, is not effective against the biomass and iron-reducing bacteria living in wells. The disinfecting power of the chlorine does not fully penetrate the outer layers of the biomass to reach the majority of the bacteria living beneath. Shock chlorination is most effective against the floating (planktonic) microbes found in well water, but these microbes represent only a very small fraction (one millionth) of the total population of bacteria living in a well.

In order to truly disinfect a water well with a chlorine solution, the biomass, iron stained coatings, and iron rust scale (tubercles) (see Figure 8) and encrustations must be physically scraped away to expose the bacteria living beneath their protective shells. Then, when the chlorine solution is applied at the proper concentration and given enough contact time, disinfection of many (not all) of the microorganisms can take place.

Sodium hypochlorite, the chlorine chemical found in bleach, when used as disinfectant, is more than 99% effective within a pH range of 5 to 7 at killing bacteria. But its effectiveness drops dramatically for water pH values greater than 7 or less than 5 (Hanson, 2001). In Arizona, groundwater pH values range from high 7 to high 8. For this reason, shock chlorination without pH adjustment is much less effective, particularly if calcium hypochlorite (solid form of chlorine) is used.

This chlorine chemical is particularly ineffective (and potentially damaging to well components) when used to disinfect wells with water that is alkaline and hard, a common characteristic of Arizona aquifers.

Any attempt to use shock chlorination inside a water well to do anything other than to obtain a negative result from a test for the presence or absence of a single colony of harmless bacteria is fruitless. Well owner shock chlorination treatment will do little or nothing to control or prevent the growth of biomass, and iron- and sulfur-reducing bacteria.

Truly effective water well treatment methods for the temporary control of coliform and iron-, and sulfur-reducing bacteria should and can only be performed when the pump is out of the well. The well can then be brushed, bailed, and disinfected using National Sanitation Foundation (NSF) approved commercial well treatment chemicals and methods applied by qualified and licensed water well contractors.

Slime Formers in Water Storage Tanks

Under certain conditions slime may also allow waterborne pathogens such as viruses and parasites to survive and even thrive in water. Excessive slime growth and warm temperatures in storage tanks, filters, and distribution pipes are ideal for the survival of dangerous amoeba parasites See Extension Publication AZ#1586 (Artiola et al., 2012). Well owners who have water storage tanks can safely disinfect them using shock chlorination, see also above publication for details on storage tank disinfection.

Shock Chlorination to Remove Slime?

All evidence suggests that shock chlorination alone will not remove all the slime from well components since slime is attached to the surfaces and must be scrubbed off. Iron- and sulfur-reducing bacteria, in particular, are very difficult to kill with shock chlorination alone since they reside deep inside the slime layers. Strong chlorine solutions, used for shock chlorination, may damage (partially oxidize) biofilms but will not kill all the organisms in them since the chlorine may not fully penetrate inside or destroy all the slime filament-like structures. Once chlorine is flushed out of the well, organisms will re-grow, often more rapidly than before (Characklis & Marshall, 1990) for several reasons, including:

a) Surviving organisms are already used (adapted) to the well environment, can start growing quicker, and produce even more slime than before.
b) Microbes attach easier to unclean surfaces (rough) than on clean surfaces.

In conclusion, shock chlorination alone may kill the planktonic bacteria and other organisms such as algae in the well water, and only damage or partially destroy parts of slime layers and organisms that reside close to the surface layers. Slime re-growth often occurs faster after shock chlorination.

Shock Chlorination and Well Water Quality

Well shock chlorination usually requires adding sufficient bleach (liquid sodium hypochlorite or powdered calcium hypochlorite) to raise the chlorine equivalent concentration inside the well to between 200 and 300 parts per million (ppm). And the chlorine chemical must be maintained in the well for 6-12 hours with the pH maintained between 5 and 7. During this time, this strong oxidant can react not only with microorganisms and other organic matter but also with rubber and plastic found in well and storage tank components, sometimes with unpredictable results.

Most of the living organisms are killed (inactivated) when they come into contact with chlorine chemicals. Their dead tissue, other animal or plant residues, and some inorganic chemicals like bromide and chloride can react with any excess chlorine present to form new chemicals called disinfection byproducts (DBPs). Some of the chemicals and groups of chemicals that can be formed include: chlorite, bromate, trihalomethanes such as chloroform, and haloacetic acids. If these disinfection byproducts are ingested regularly, they can affect the nervous system and increase the risk of cancer (USEPA, 2013).

Most public drinking water sources are chlorinated, and the USEPA regulates the levels of these chemicals in public water supplies. The formation of DBPs in chlorinated waters is difficult to predict because it depends on many things including levels of chlorine, type of organic matter present, contact time, temperature, and other water quality parameters.

When a well is purged following shock chlorination, residual chlorine and DBPs are quickly removed from the well if they are in the free water, but they are not so easily removed if they are inside any remaining slime residues. Chemicals move slowly in an out of slime because it is made up of thick, sticky strands of fiber-like chemicals, as previously discussed.

Studies have shown that DBPs can be detected in well water after it has been purged four (4) well volumes, and these chemicals can be found in well water even after no free chlorine is detected (Seiler, 2006; Walker and Newman, 2011).

Studies have also shown that shock chlorination may temporarily increase the concentrations of some metals in well water. Elevated levels of metals such as lead, copper, zinc, iron, and arsenic have been measured in well water just after chlorination. In some cases, wells had to be purged more than four (4) well volumes before metal levels returned to normal, (Seiler, 2006; WDNR, 2008; and Walker and Newman, 2011).

Figure 9. Contractor chlorinating a well. Note safety equipment and water circulation hose through the opened well seal, something most well owners cannot do themselves. Photo by G. Hix.

In conclusion, following shock chlorination, it is very important to flush residual chlorine and any toxic chemicals that may have been formed or released from the well components or aquifer materials. Shock chlorinated wells should be purged at least four (4) well volumes or until no residual chlorine is detected in the well water using a chlorine test kit, see Extension Publication #AZ1586 (Artiola et al., 2012).

Was Your Well Disinfected?

The construction and design standards for private (also called exempt) wells in Arizona require that a new well be "disinfected" before use by humans. According to Arizona Department of Water Resources (ADWR) Rule 12-15-814 any well from which the water is to be withdrawn is intended to be utilized for human consumption or culinary (cooking) purposes without prior treatment shall be disinfected by the well drilling contractor **before** removing the drill rig from the well site." However, in Arizona, a well driller may or may not install a pump in a new well, leaving this last step to a pump contractor. But, since no method of disinfection is specified in the regulations and without a pump in the well, the driller has no means to effectively disinfect a new well.

Additional confusion arises because the ADWR does not license or regulate water well pump installers. Therefore, there is no requirement for them to disinfect the pump or any other well components at any time. In short, your well may never have been disinfected.

Who Should Disinfect a Well?

Well disinfection should be done by a qualified well driller or pump contractor **at the time the well is equipped with a pump for human use.**

Owners of existing wells should remember that it is a good practice to disinfect any well that has had major maintenance (such as pump replacement, well rehabilitation procedures, or new equipment installed in the water system) with a strong chlorine solutions to kill pathogens that might be left on the surfaces of any drinking water system components during construction and/or maintenance.

There are several reasons for leaving well disinfection (using shock chlorination or other methods) to professional drillers or pump installers. These professionals:

a) Are trained to safely handle strong chlorine solutions and dispose of them offsite;
b) Adjust the well water pH as needed using strong acids and water testing equipment;
c) Use appropriate amounts of chlorine chemicals to prevent damage to well components [1]; and
d) Purge sufficient well water volumes and test residual chlorine levels in the well water.

Note that well disinfection is no guarantee that the well water will pass the fecal or total coliform tests a few days after shock chlorination, because:

a) Bacteria in the remaining slime that are not affected by the chlorine may re-grow and contaminate the well water;
b) Improperly capped/sealed wells can allow bacteria to enter the well;
c) A non-existent or compromised surface seal surrounding the upper twenty feet of well casing may allow surface water and contaminates to enter the aquifer; and
d) Contamination may be in the aquifer water, not the well itself.

What If Your Well Water Fails the Total Coliform, Fecal Coliform, or *E. coli* Tests?

One of the main reasons for collecting and performing a water quality test for the presence or absence of coliform bacteria is to satisfy homebuyer or mortgage lender requirements during the sale and transfer of real estate. While Arizona has no specific requirements for water quality of private water wells, including during the sale and transfer of real estate, a "potability" test is often a part of the terms of the sale when a private or shared water well is the source of domestic water for the home.

Laboratory results from these tests indicate that a "Positive" test result will be found in approximately 10% of the samples submitted for coliform bacteria testing, personal communication (Turner, 2013). Less than half of the samples that tested positive for coliform also test "Positive" for fecal coliform or *E.coli*. Obtaining a totally bacteria free water sample in the field is not as simple as it may seem and the accidental introduction of coliform is quite possible. See Arizona Extension Publications AZ1486f (Farrell-Poe et al., 2011) & AZ1486g (Farrell-Poe, 2010).

When a "potability" test is required for the transfer or financing of real property and the coliform test results come back "Positive," shock chlorination of the well or water storage tanks and distribution system may be what is needed to pass the test.

Well owners, buyers, estate brokers, and mortgage lenders must understand that even if the test results are "Negative" for total coliforms, this does not mean that the well water is totally safe to drink. However, if the tests are "Positive" for total and fecal coliforms, then the well water is most likely contaminated with feces and not safe to drink (USEPA, 2006)

Ultimately, **a private well owner has the sole responsibility for the quality of water produced by their well.** It is up to the well owner to insure that the well water remains free of harmful bacteria and/or other potentially harmful contaminants. Well owners that are concerned about the re-occurring presence of pathogens and/or other contaminants such as DBPs in their well water should consider home water treatment devices either as point of entry (whole house) or point of use (prior to faucet or use). These treatment options are discussed in detail in the Arizona Publication AZ1578 (Artiola et al., 2012).

Summary

Shock chlorination alone may not remove slime that quickly forms in wet and submerged surfaces of well components. Slime is made up of a complex mixture of organisms that live together benefiting from each other and improving water quality. However, excessive slime growth can clog well screens and pump intakes and can also harbor pathogens that can make well water unsafe to drink.

Arizona regulations on private wells are not clear on how wells should be disinfected and by whom.

It is a good practice to disinfect all new wells and old wells after maintenance. Proper well shock chlorination requires well equipment removal, surface scrubbing, proper chlorine dose, and water pH adjustment, followed by thorough well purging to remove disinfection byproducts and testing for residual chlorine – this should be done by qualified water well personnel (www.AzWWA.Org, www.NGWA.Org). Well owners should also clean and disinfect their water storage tanks regularly.

Well owners should also consider home water treatment devices either for the entire home (point of entry) or at the point of use to treat all or some of their well water to insure potability.

[1] Chlorine chemicals are strong oxidants: strong bleach solutions may damage plastic pipes, electric cables, and rubber diaphragms in pressure tanks.

References

Arizona Administrative Code (A.C.C.). R12-15-814. Disinfection of Wells. http://www.azsos.gov/public_services/Title_12/12-15.htm. Accessed 5-3-13.

Artiola, J.F., K. Uhlman, G. Hix. 2012. Arizona Wells: Maintaining and Troubleshooting Wells. University of Arizona Cooperative Extension Publication AZ1581.

Artiola, J.F., C. Rock, and G. Hix. 2012. Water storage tanks, disinfection, and maintenance. University of Arizona Cooperative Extension Publication AZ1586.

Artiola, J.F., K. Farrell-Poe, and J. Moxley. 2012. Arizona Know Your Water: A consumer's guide to water sources, quality, regulations and home water treatment options. University of Arizona Cooperative Extension Publication AZ1578.

BSIa. 2013. The Science Behind BioShield. http://www.berrysystemsinc.com/brochure.html. Accessed 5-1-13.

BSIb. 2013. Leptohrix http://www.berrysystemsinc.com/irb.html

Characklis, W.G.and K.C. Marshall, eds. 1990. *Biofilms*, John Wiley & Sons, Inc., New York.

Farrell-Poe, K. 2010. Obtaining a water sample for bacterial analysis. University of Arizona Cooperative Extension Publication AZ1486g.

Edstrom 2004. Biofilm: The Key to Understanding and Controlling Bacterial Growth in Automated Animal Drinking Water Systems. http://www.edstrom.com/file.aspx?DocumentId=21. Accessed 5-6-13.

Farrell-Poe, K., L. Jones-McLean, and S. McLean. 2011. Private water well components. University of Arizona Cooperative Extension Publication AZ1486b.

Farrell-Poe, K., L. Jones-McLean, and S. McLean. Well water testing and understanding the results. 2011. University of Arizona Cooperative Extension Publication AZ1486f.

Hanson, D.T. 2001. Chlorine and misinformation. *Water Well Journal*, Aug. 35-35.

Mittelman, M.W. 1985. Biological fouling of purified-water systems: Part 1, bacterial growth and replication. *Microcontamination* 3(10): 51-55, 70, October.

Seiler, R.L. 2006. Mobilization of lead and other trace elements following shock chlorination of wells. *Sci. Total Environ.*, 367:2-3, 757-768.

Todar, K. 2009. The New Microbial World. University of Wisconsin. http://textbookofbacteriology.net/themicrobialworld/homepage.html. Accessed 5-6-13.

United States Environmental Protection Agency (USEPA). 2006. Ground Water Rule. Federal Register. 40 CFR, Parts 59, 141 and 142. Vol. 7. No. 2168. Wednesday, November 8, 2006.

United States Environmental Protection Agency (USEPA). 2013. Disinfection byproducts: http://water.epa.gov/drink/contaminants/index.cfm#Byproducts. Accessed 3-3-13.

Walker, M. and J. Newman. 2011. Metals releases and disinfection byproduct formation in domestic wells following shock chlorination. *Drink. Water Eng. Sci.*, 4: 1-6.

Washington Department of Natural Resources (WDNR). 2008. Shock chlorination and temporary arsenic release. http://www.caes.uga.edu/Publications/pubDetail.cfm?pk_id=8021#Arsenic. Accessed 4-2-13.

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Impacts of on-farm water sanitation practices on microbial hygiene in poultry waterlines and efficacy of sodium hypochlorite-based product on *foodborne pathogens*

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Primary Audience: Poultry scientists and Live production managers

SUMMARY

The microbial water quality of poultry water supplies can be correlated to bird health, livability, overall performance, and human food safety. On-farm microbial evaluations were conducted to understand microbial hygiene of poultry waterlines based on water sanitation practices. With on-farm study I, 2 farms were selected: Farm A and Farm B; that did not practice water sanitation consistently during the flock grow-out period. Prevalence tests for specific pathogens- *Salmonella, E coli, Campylobacter, Listeria,* and *Staphylococcus* were performed for both farms by collecting swab samples post bird harvest from waterlines and plating in specific growth media. With the on-farm study II, 1 broiler farm with 4 barn units that treated water (Chlorine [**Cl**]= ∼ 1 ppm) during the flock grow-out period and flushed waterlines between flocks using concentrated chlorine solution (>1,000 ppm) was selected. Swab samples (1 from each barn, n = 4; each sampling occasion) were collected on 3 occasions-before flushing, after flushing, and at the end of the grow-out period (d 42) to understand biofilm growth nature in poultry waterlines. Additionally, a separate in-vitro study was conducted to understand the efficacy of a commonly used poultry drinking water sanitizer product (sodium hypochlorite, 8.25%) against specific foodborne pathogens*: Salmonella* Enteritidis (**SE**), *E. coli* and *Listeria*. Results from the on-farm study I showed that Farm A and Farm B were positive for *Listeria* and *Staphylococcus,* whereas these farms were found negative for other species tested. The findings from on-farm study II showed that biofilm reestablished (>4 $\log_{10}$ cfu/mL) in waterlines by the end of grow-out cycle despite waterline cleaning and consistent water sanitation during flock grow-out period. The in-vitro test showed that the efficacy of chlorine-based water sanitizer at the field application dose rates was affected by pathogen load in water and pathogen types. The overall results from on-farm or in-vitro studies indicated that poultry

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growers need to emphasize consistent and robust poultry drinking water sanitation practice in their farms to keep the acceptable bacterial levels in poultry water supplies or water systems.

Key words: poultry water supplies, microbial level, biofilm, sanitation

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DESCRIPTION OF PROBLEM

Water is the most critical nutrient for poultry (Bell, 2002) which also aids in body metabolism and excretion (Jafari et al., 2006). Water provides an excellent medium for various chemical reactions that are required to form meat and eggs. In comparison to other nutrients, birds consume water in large quantities. The water consumption could be double the intake of feed amount and could vary according to age and species of birds, activity level, air temperature, humidity, respiratory rate, and existing diseases and environmental conditions such as heat stress. Any increase or decrease in normal water consumption of poultry could indicate a health disorder in birds (Butcher et al., 1999). Further, numerous studies have shown the positive correlation between feed intake and water consumption (Patterson et al., 1989; Lott et al., 2003). The knowledge of providing birds with sufficient access to water is well appreciated. Water availability and quality both have significant roles in the overall health and productivity of birds. However, the role of providing safe and quality water for optimal performance is generally neglected.

The presence of bacteria, fungi, minerals, and water additives in the poultry water system and drinker lines hinder the efficient management practices intended to gain optimal performance (Oviedo, 2006). Microbial contamination in poultry drinking water compromises birds' overall health and performance (Maharjan et al., 2016). The waterline system in a poultry house can be an appropriate habitat where bacteria can thrive and pose health risks to birds. Several factors such as warm water temperature (27−30˚C) of housing environment, reduced flow of water in poultry waterlines, and supplementation of water additives like vitamins and organic acids favor the growth of bacteria. These factors can likely predispose chicks to infections during their early grow-out phase. Supplying birds with contaminated water may also aggravate litter quality and cause high production of ammonia thus impairing the performance and livability of birds. Poor water quality is responsible for decreasing the effectiveness of vaccines and medications supplied through the waterlines (Fairchild et al., 2006), thus necessitating treating poultry drinking water supplies.

In addition to improving flock performance, effective poultry drinking sanitation is an integral component of preharvest food safety that aims to minimize foodborne pathogens in poultry production system, thus protecting the consumer's health. *Salmonella, Campylobacter, Escherichia coli*, and *Listeria* are some of the foodborne pathogens of concern due to their ability to cause infection (Cook et al., 2012). Some pathogenic bacteria show resistance to a certain extent to chemical and physical agents (Lorenzeni, 2020). The acceptable microbial load in poultry water supplies for poultry drinking purpose is considered to be 1,000 CFU per milliliter, however the contamination with *E. coli*, and other foodborne pathogens are considered nonacceptable (Watkins, 2008). Biofilms, which are aggregates of bacterial cells producing adhesive film resistant to disinfectants, can grow in poultry waterlines and pose health challenges to birds (Maharjan et al., 2017). Here, we studied the impacts of water sanitation practices on poultry waterline hygiene for the presence of bacterial pathogens and the nature of bacterial biofilm growth by conducting on-farm evaluations. Growers use different chemical-based poultry sanitizers to sanitize poultry drinking water supplies and chlorine-based product is one commonly used water sanitizer. The other part of this study evaluated the efficacy of sodium hypochlorite-based product as a poultry drinker water sanitizer against foodborne pathogens- *Salmonella, E. coli,* and *Listeria*.

MATERIAL AND METHODS

On-Farm Microbial Assessment in Poultry Waterlines

Two separate on-farm evaluations were conducted to understand microbial hygiene of poultry waterlines based on water sanitation practices. On-farm study 1, 2 commercial broiler farms (Farm A and Farm B) that used well water sources were considered. Both farms performed inconsistent, or no water sanitation practices for their water supplies during the flock grow-out period. Prevalence tests for specific pathogens, *Salmonella, E. coli, and Listeria,* were conducted both the farms by collecting swab samples during downtime from waterlines (n = 4 lines, each farm) and plating it in agar plates with growth media specific to these organisms (Xylose Lysine Deoxycholate (**XLD**) agar, Eosin Methylene Blue (**EMB**), and Tryptic Soya Agar with Listeria supplements, respectively) In the on-farm study 2, a broiler farm that had 4 house units that treated drinking water supplies consistently (free chlorine (**Cl**)= ~ 1 ppm) during the flock grow-out period and flush waterlines between flocks immediately before placing chicks using concentrated chlorine solution (>1,000 ppm) was selected. Swab samples (1 from each house, n = 4; each sampling occasion) were collected on 3 occasions: before flushing (pre-flush samples), after flushing (postflush samples), and at the end of the grow-out period (d 43, end of the grow-out) to understand the incidence of bacterial biofilm build-up in waterlines. The evaluation was conducted for 2 consecutive flocks. Total bacteria count at 3 sampling occasions (pre-flush, postflush, and d 43 swab samples) were assessed by plating Petrifilm (3M Petrifilm, St. Paul, MN).

Swabbing method: Sterile sponge dipped in phosphate buffer saline (**PBS**) was utilized to swab the inside of waterlines. The sponge was held in sterile forceps and the swabbing at the end of the waterline was done pressing the sponge against the wall of waterline 4 to 6 cm deep, rotated through 360˚. The swabbed sponge was then brought back to the PBS solution, until the solution was analyzed for bacterial enumeration.

Efficacy Evaluation of Chlorine-Based Product Against Bacterial Pathogens

A series of benchtop studies were conducted to understand the efficacy of the chlorine-based product (NaOCl, 8.25%) against *Salmonella enteritidis (strain 35664), Escherichia coli (ATCC 25922), and Listeria monocytogenes (strain 7644)* mimicking the application rates of the product as commonly practiced in production farms. First, the standard culture of *Salmonella, E coli* and *Listeria* were made by subjecting to the overnight culture at 37˚C in an incubator, which the standard culture was later enumerated. A stock solution of the product was prepared by mixing 1 mL of NaOCl with 32 mL of distilled water. The test solution consisted of DI water (12.8 mL) and pathogens introduced to it at 3 different doses of standard culture (300 uL, 600 uL, and 900 uL volume) for each specific pathogen. Test solutions were prepared in triplicate. A 100 uL of stock solution of the product was treated with each test solution. The whole set of experiments was repeated at the higher dose rate (2X) of the stock solution, stock solution prepared at 2 mL of NaOCl to 32 mL DI water and introduced to similarly prepared test solutions.

Microbial sampling occasions and enumeration: After thorough vortex mixing, samples were plated (100 uL) in duplicate in specific media agar plates for each test solution - immediately (0 m), 5 m, 3 h, and 24 h post-treatment. The agar plate media used for *Salmonella, E. coli*, and *Listeria* were Xylose Lysine Deoxycholate (**XLD**) agar, Eosin Methylene Blue (**EMB**), and Tryptic Soya Agar with *Listeria* supplements, respectively. *Salmonella* and *E. coli* cultures were subjected to a 24-h incubation period at a temperature of 37˚C, while Listeria cultures were incubated at 37˚C for 48 h before enumeration.

Statistical Analysis. Results from the on-farm study I was presented as positive or negative result for pathogens tested. For the on-farm study 2 to assess the biofilm growth nature in poultry waterline, the data were combined for barns by flock, and the data was analyzed using one-way ANOVA using JMP Pro16. For the in-vitro studies, the values of bacterial enumeration were averaged for the replicates for each

inoculum size and compared for the decrease in the bacterial enumeration post adding product treatment over time. Significant means were tested using the student's *t* test for both the on-farm and in-vitro tests.

RESULTS AND DISCUSSION

On-Farm Evaluation Results

On-Farm Study I: Bacteria Prevalence. Both the farms, Farm A and Farm B, were found to be contaminated with *Listeria* and *Staphylococcus* in poultry waterlines. Neither of these farms had a prevalence of *Salmonella, E coli*, and *Campylobacter* (Table 1). *Listeria* spp. can be found in all the stages of poultry production and is common in soil, sewage, feces from birds, and surface water (Ryser and Marth, 2007; Goh et al., 2012). The favorable temperature could enhance the replication of these anaerobes in poultry water system as they can grow in temperatures of 30 to 37˚C. Numerous studies have shown the prevalence of *Listeria* in environmental samples ranges from 1.4 to 53% (Milillo et al., 2012; Petersen and Madsen, 2000; Jones et al., 2012; Schwaiger et al., 2010), demonstrates prevalence in diverse settings, including nonanthropogenic and anthropogenic environments. Studies highlight the presence of L. monocytogenes and L. innocua in various habitats, such as marine, soil, sewage, and both stagnant and running water (Luppi et al., 1988; Frances et al., 1991; MacGowan et al., 1994; Bou-m'handi et al., 2007). Several factors influence the susceptibility of *Listeria spp.* to chlorine. Different aspects such as chlorine concentration, contact time, environmental conditions- such as presence of organic matter, play a role in determining how Listeria spp. respond to chlorine (Virto et al., 2005). *Listeria* could be present in treated farms when there is an inconsistent water sanitation program in a farm. Additionally, *Listeria* is less susceptible to chlorine than *E. coli* O157:H7 (Park et al., 2004). The mechanism of lysis of pathogens such as bacteria due to chlorine is by breaking the chemical bonds in their molecules. Sodium hypochlorite (**NaOCl**) exhibits potent antimicrobial properties as an oxidizing agent (Ueno et al., 2018), facilitating electron transfer from substrates like proteins, carbohydrates, and lipids (McDonnell and Russell, 1999; Tawakoli et al., 2017). This process, occurring more rapidly and effectively at alkaline pH, leads to the disruption and cleavage of chemical bonds in biomolecules, enhancing its efficacy, particularly against bacterial spores (Almhöjd et al., 2023). Biofilm formed by bacteria offers protection against low and high temperatures and pH, high salt conditions, and low availability of nutrients, and thus it resists the action of disinfectants and antibiotics (Stoodley et al., 2013). The findings by Dahshan et al. (2016) showed the prevalence of *Listeria* to be 47.5% in broiler poultry farms in which it contributed around 10% in drinking water (70% in farm feed, 52.5% in litter, and 42.2% in chicken breasts).

Staphylococcus aureus lodges normally in the skin and upper respiratory passage of healthy and infected chickens (Devriese,1990). There are several reasons behind the resistance of *Staphylococcus* to most of the disinfectants used in production facilities. *Staphylococcus aureus* can develop resistance to disinfectants due to its ability to aggregate and form biofilms on surfaces within food facilities. Biofilm formation renders the bacteria less susceptible to biocides, sanitizers, and antimicrobials in general (Fux et al., 2004). Additionally, pathogenic foodborne bacteria, including S. aureus, can acquire resistance to antimicrobial agents and biocides through horizontal gene transfer from antimicrobial-resistant bacteria or adaptive mutation. The growth of *S. aureus* as a biofilm increases the risk of transferring antimicrobial

Table 1. Prevalence of specific pathogens in waterline swab samples of farm A and farm B that did not practice consistent water sanitation program (n = 4 lines).*

Farm	Salmonella	*E. coli*	Campylobacter	Listeria	Staphylococcus
A				+	+
B				+	+

*, absent; +, present.

resistance genes (**ARG**) and biocide resistance genes (**BRG**) to both pathogenic and non-pathogenic bacteria on food products and contact surfaces within food facility environments (Chieffi et al., 2023). The low efficacy of sodium hypochlorite at a lower dose may be due to its inability to break cell wall or membrane which prevents its absorption into bacterial cells (Acsa et al., 2021). The findings by Wanja et al. (2020) also reported that sodium hypochlorite was less effective in killing the tested bacterial pathogens (*E coli, Staphylococcus,* and *Streptococcus*) at the recommended concentration. Another factor that may contribute to the resistance of these bacteria to disinfectant is due to the biofilm formation. In the available reports from human drinking water, *Staphylococcus* spp. has the potential to adhere strongly on PVC surfaces (Simões et al., 2007). Staphylococcus aureus can survive at temperature ranging from 15˚C to 45˚C and could tolerate NaOCl concentration up to 10% (Behling et al., 2010), and proliferate rapidly at room temperature. Hygiene management in production facilities can affect the persistence of *Staphylococcus* at the farm level (Govender et al., 2019).

On-Farm Study 2: Waterline Swab Sample Evaluations. The broiler farm swab sample evaluation exhibited aerobic plate count over 4 $\log_{10}$ CFU/mL before flushing the waterlines (Figure 1). It dropped to < 1 $\log_{10}$ CFU/mL ($P < 0.05$) postflushing, while the biofilm regrowth occurred by d 43 at the end of the grow-out period with counts > 4 $\log_{10}$ CFU/mL. The warmer temperature of the water, and stagnant flow rates in poultry waterlines enhance the multiplication of bacteria and the formation of biofilms in the drinking water system (Maharjan et al., 2017). Poor disinfection ability of disinfect product against biofilm polymer matrix favors better protection of the bacteria in its surfaces despite disinfection practices (Muhterem-Uyar et al., 2015). Flushing the water system with sanitized water loosens the substances thus removing the developed biofilms. The results also showed a decrease in aerobic plate count in swab samples collected after

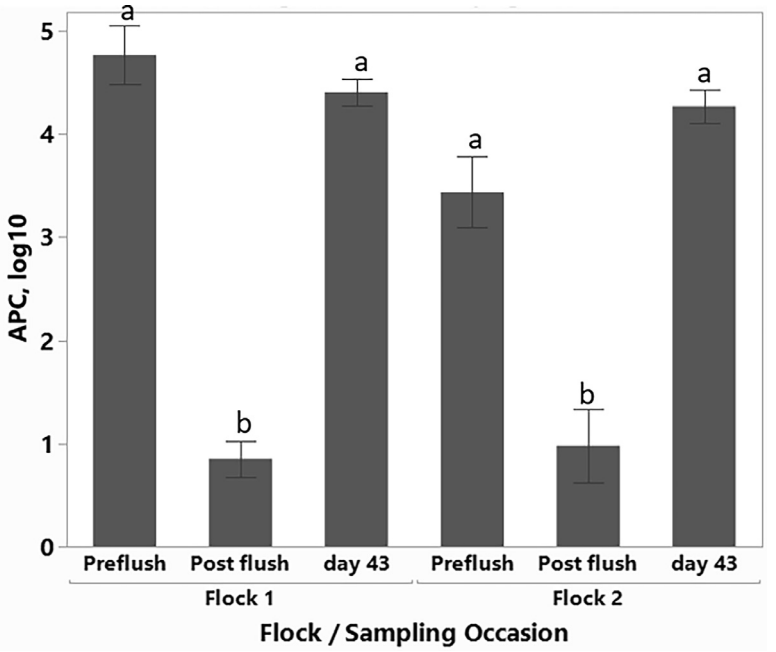

Figure 1. On-farm evaluation of bacterial swab samples in poultry waterlines performed at pre-flush and post flush (immediately before placing chicks), and at the end of the grow-out period (d 43) for 2 consecutive flocks in a treated farm. (n = 8 lines, each occasion). Different letters on top of bar within flock represents significantly differently means ($P < 0.05$).

Table 2. Bacterial enumeration in test solutions observed post sanitizer treatment (chlorine-based) at lower (1X) and higher (2X) concentration of stock solution prepared for different pathogen inoculum doses. For *Salmonella*, the experiment was conducted for 1X stock concentration, whereas for *E. coli* and *Listeria*, the experiments were conducted for 1X and 2X concentrations.*

Pathogen/ volume (μL)	Baseline count	0 min ($\log_{10}$ cfu/100 uL)	5 min	3 h	24 h
*Salmonella**					
300	6.82	6.64	2.52	0	0
600	9.37	9.09	2.56	0	0
900	9.55	9.42	4.14	0	0
E. coli					
Trial 1/1X dose					
300	6.65	6.30	6.35	6.26	5.32
600	7.02	6.69	6.69	6.68	5.99
900	7.05	6.87	6.89	6.79	6.11
Trial 2/2X dose					
300	6.43	6.14	6.27	6.08	5.77
600	6.77	6.25	6.69	6.24	5.82
900	7.0	6.06	6.91	6.79	5.96
Listeria					
Trial 1/1X dose					
300	8.14	5.86	5.65	5.49	5.28
600	8.43	6.16	6.14	6.09	5.89
900	8.68	6.44	6.38	6.36	6.25
Trial 2/2X dose					
300	6.90	5.20	5.18	4.93	5.63
600	7.08	5.74	5.69	5.20	5.76
900	7.19	5.98	5.96	5.49	5.93
		Free chlorine residual in test solution (ppm)			
1X	0.25	2	2−3	1−3	1−3
2X	0.25	4-5	6−7	5−7	4−5

*X and 2X: Stock solutions prepared at 1 mL and 2 mL of sodium hypochlorite (8.25 %) to 32 mL deionized water, respectively.

flushing the drinking water system. Watkins (2006) has also reported the effectiveness of chlorine-based sanitizers in reducing the microbial load of water. Studies have recorded the increased biofilm formation with warmer environmental temperatures of water (Kadam et al., 2013; Bonsaglia et al., 2014). Poimenidou et al. (2016) observed a temperature of 20˚C fostered more biofilm formation than at 37˚C which is the barn temperature in the latter half of bird grow-out period. Thus, monitoring regular water sanitation programs during the grow-out period should be emphasized to assess water system hygiene in production facilities.

Efficacy Evaluation of Chlorine-Based Poultry Water Sanitizer

The efficacy test exhibited the sodium-hypochlorite-based product was able to reduce the bacterial count to 0 CFU/mL post 3 h for all the volume of *Salmonella* Enteridis seeded at the tested lower dose, whereas the product was not effective for the tested doses for *E. coli* or *Listeria*. The bacterial count exceeded 5 $\log_{10}$ CFU/mL for all the volume of *E. coli* and *Listeria* post 24 h of the treatment for both the test doses at X and 2X. (Table 2). The presence of *Salmonella* up to 5 m of treatment in this experiment did not seem adequate to exert a bactericidal effect. The increasing concentration of chlorine-based disinfectants had significant effects in reducing the population of *S. enteritidis* planktonic cells under clean or dirty conditions (Byun et al., 2021). Poppe et al., (1986) reported higher counts of *Salmonella* and *Coliforms* in non-chlorinated water. Poppe (1984) reported no *Salmonella* in chlorinated water having free available chlorine not less than 0.1 ppm. The level of such concentration of

chlorine might have been sufficient to kill *Salmonella*. The application of sodium hypochlorite against *S. enteritidis* planktonic cells at 100 μg/mL for one min showed a reduction by 5.58 log CFU/mL and they were not detected when the reaction time was increased to 5 min under clean conditions, indicating increased efficacy over time and higher dose of disinfectant. It has been shown that, the inactivation of *Salmonella* was achieved by sodium hypochlorite within 5 s in the absence of organic material (Toyofuku et al., 2017). However, when fetal bovine serum (**FBS**) was present at a concentration of 0.5%, the bactericidal effects of NaOCl were completely reduced.

Maharjan et al., (2017) observed no survival of *E. coli* in test coupons after adding chlorine-based product at 24 and 48 h, which the results were not coherent with the current study. The strain of *E coli* used by Maharjan et al., (2017) was avian pathogenic, whereas in this study a different strain (*E. coli* ATCC25922) was utilized. Similar inefficacy of the product against *Listeria* tested was observed in this study. Chlorine, applied at concentrations of free chlorine residuals above 0.5 ppm, has been found to be an effective disinfectant in controlling aerobic bacteria in water within the safety levels for poultry production (Maharjan et al., 2016). Rasheed et al., (2016), observed that chlorine concentrations applied at 1.0 mg/L were optimum to inactivate *E coli* in drinking water. The free chlorine residual levels were higher in both X and 2X doses tested, therefore, showed > 1 ppm residual concentration until 24 h, however, the counts for *E coli* or *Listeria* tested did not drop significantly until 24-h time. The product efficacy could be dictated by the strain of *E coli* or *Listeria* utilized or their load in water as per the findings in this study.

CONCLUSIONS AND APPLICATIONS

1. Untreated farms could exhibit a greater susceptibility to pathogen positivity in poultry water system. Even treated farms were vulnerable to biofilm growth in poultry water lines during flock grow-out period. Therefore, waterline cleaning between flocks becomes a mandatory practice to improve the waterline hygiene before starting the next set of chicks.
2. Application of chlorine-based water sanitizer at the tested dose in-vitro did not bring significant inhibitory effect against the tested pathogens, *E coli* and *Listeria*, but did so for *Salmonella*. The efficacy of sodium hypochlorite could be dictated by pathogen type and their load in water supplies. Therefore, testing of water supplies for specific microbial types is recommended, particularly if the sub-optimal microbial water quality is suspected at the farm, so that the water sanitation program can be devised accordingly.

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DISCLOSURES

The authors declare no conflicts of interest.

REFERENCES

Acsa, I., B. L. Caroline, N. P. Njeru, and N. L. Wanjiru. 2021. Preliminary Study on disinfectant susceptibility/resistance profiles of bacteria isolated from slaughtered village free-range chickens in Nairobi. Kenya. Int. J. Microbiol. 2021:1–7.

Almhöjd, U. S., A. Lehrkinder, A. M. Roos-Jansaker, and P. Lingström. 2023. Antimicrobial efficacy of chlorine agents against selected oral pathogens. Clin. Oral Investig 27:5695–5707.

Behling, R. G., J. Eifert, M. C. Erickson, J. B. Gurtler, J. L. Kornacki, E. Line, R. Radcliff, E. T. Ryser, B. Stawick, and Z. Yan. 2010. Selected pathogens of concern to industrial food processors: infectious, toxigenic, toxico-infectious, selected emerging pathogenic bacteria. Pages 5−6 in Principles of Microbiological Troubleshooting in the Industrial Food Processing Environment. J. L. Kornacki, ed. Springer Publishing, New York.

Bell, D. D. 2002. Consumption and quality of water. Pages 411−430 in Commercial Chicken Meat and Egg Production. Springer, Boston.

Bonsaglia, E. C. R., N. C. C. Silva, A. J. Fernades, J. P. J Araujo, M. H. Tsunemi, and V. L. M. Rall. 2014. Production of biofilm by *Listeria monocytogenes* in different materials and temperatures. Food Control 35:386–391.

Bou-m'handi, N., C. Jacquet, A. El Marrakchi, and P. Martin. 2007. Phenotypic and molecular characterization of Listeria monocytogenes strains isolated from a marine environment in Morocco. Foodborne Path Dis 4:409–417.

Butcher G.D., J.P. Jacob, and F.B. Mather. 1999. Common poultry diseases. Fact Sheet PS-47 University of Florida. Accessed Oct. 2023. https://edis.ifas.ufl.edu/publication/PS044

Byun, K. H., S. H. Han, J. W. Yoon, S. H. Park, and S. D. Ha. 2021. Efficacy of chlorine-based disinfectants (sodium hypochlorite and chlorine dioxide) on *Salmonella* Enteritidis planktonic cells, biofilms on food contact surfaces and chicken skin. Food Control 123:107838.

Chieffi, D., F. Fanelli, and V. Fusco. 2023. Antimicrobial and biocide resistance in Staphylococcus aureus: genomic features, decontamination strategies, and the role of *S. aureus* complex-related species, with a focus on ready-to-eat food and food-contact surfaces. Front. Food Sci. Technol 3:1165871.

Cook, A., J. Odumeru, S. Lee, and F. Pollari. 2012. Campylobacter, *Salmonella, Listeria* monocytogenes, verotoxigenic Escherichia coli, and Escherichia coli prevalence, enumeration, and subtypes on retail chicken breasts with and without skin. J. Food Prot. 75:34–40.

Dahshan, H., A. M. A. Merwad, and T. S. Mohamed. 2016. *Listeria* species in broiler poultry farms: potential public health hazards. J. Microbiol. Biotechnol. 26:1551–1556.

Devriese, L. A. 1990. Staphylococci in healthy and diseased animals. Soc. Appl. Bacteriol. Symp. Ser. 19:71S–80S.

Fairchild, B., A. Batal, and C. Ritz. 2006. Effect of drinking water iron concentration on broiler performance. J Appl Poult Res. 15:511–517.

Frances, N., H. Hornby, and P. R. Hunter. 1991. The isolation of Listeriaspecies from fresh-water sites in Cheshire and North Wales. Epidemiol Infect 107:235–238.

Fux, C. A., S. Wilson, and P. Stoodley. 2004. Detachment characteristics and oxacillin resistance of *Staphyloccocus aureus* biofilm emboli in an in vitro catheter infection model. J. Bacteriol. 186:4486–4491.

Goh, S. G., C. H. Kuan, Y. Y. Loo, W. S. Chang, Y. L. Lye, P. Soopna, J. Y. H. Tang, Y. Nakaguchi, M. Nishibuchi, L. Afsah-Hejri, and R. Son. 2012. *Listeria* monocytogenes in retailed raw chicken meat in Malaysia. Poult. Sci. 91:2686–2690.

Govender, V., E. Madoroba, K. Magwedere, G. Fosgate, and L. Kuonza. 2019. Prevalence and risk factors contributing to antibiotic-resistant Staphylococcus aureus isolates from poultry meat products in South Africa, 2015-2016. J. S. Afr. Vet. Assoc. 90:e1–e8.

Jafari, R. A., A. Fazlara, and M. Govahi. 2006. An investigation into *Salmonella* and fecal coliform contamination of drinking water in broiler farms in Iran. Int. J. Poult. Sci. 5:491–493.

Jones, D., K. Anderson, and J. Guard. 2012. Prevalence of coliforms, *Salmonella, Listeria*, and Campylobacter associated with eggs and the environment of conventional cage and free-range egg production. Poult. Sci. 91:1195–1202.

Kadam, S. R., H. M. W. den Besten, S. van der Veen, M. H. Zwietering, R. Moezelaar, and T. Abee. 2013. Diversity assessment of *Listeria monocytogenes* biofilm formation: Impact of growth condition, serotype and strain origin. Int. J. Food Microbiol. 165:259–264.

Lorenzeni, G. 2020. Staphylococcosis in chickens. Accessed Nov. 2022. https://extension.psu.edu/staphylococcosis-in-chickens.

Lott, B. D., W. A. Dozier, J. D. Simmons, and W. B. Roush. 2003. Water flow rates in commercial broiler houses. Poult. Sci. 82:102.

Luppi, A., G. Bucci, P. Maini, and J. Rocourt. 1988. Ecological survey ofListeriain the Ferrara area (northern Italy). Zentralblatt fur Bakteriologie, Mikrobiologie, und Hygiene 269:266–275.

MacGowan, A. P., K. Bowker, J. McLauchlin, P. M. Bennett, and D. S. Reeves. 1994. The occurrence and seasonal changes in the isolation ofListeriaspp. in shop bought food stuffs, human faeces, sewage and soil from urbansources. Int J Food Microbiol 21:325–334.

Maharjan, P., S. Dey, G. Huff, W. Zhang, G. K. Phillips, and S. Watkins. 2017. Effect of chlorine treatment on inhibition of *E coli* serogroup O2 incorporation into 7-day-old biofilm on polyvinylchloride surface. Poult. Sci. 96:2862–2870.

Maharjan, P., T. Clark, C. Kuenzel, M. K. Foy, and S. Watkins. 2016. On-farm monitoring of the impact of water system sanitation on microbial levels in broiler house water supplies. J. Appl. Poult. Res. 25:266–271.

McDonnell, G., and A. D. Russell. 1999. Antiseptics and disinfectants: activity, action, and resistance. Clin. Microbiol. Rev. 12:147–179.

Milillo, S. R., E. C. Friedly, J. C. Saldivar, A. Muthaiyan, C. O'Bryan, P. G. Crandall, M. G. Johnson, and S. C. Ricke. 2012. A review of the ecology, genomics, and stress response of *Listeria* innocua and *Listeria* monocytogenes. Crit. Rev. Food Sci. Nutr. 52:712–725.

Muhterem-Uyar, M., M. Dalmasso M, A. S. Bolocan, M. Hernandez, A. E. Kapetanakou, T. Kuchta, S. G. Manios, B. Melero, J. Minarovicova, A. I. Nicolau, J. Rovira, P. N. Skandamis, K. Jordan, D. R. Lazaro, B. Stessl, and M. Wagner. 2015. Environmental sampling for *Listeria monocytogenes* control in food processing facilities reveals three contamination scenarios. Food Control 51:94–107.

Schwaiger, K., E. Schmied, and J. Bauer. 2010. Comparative analysis on antibiotic resistance characteristics of *Listeria* spp. and Enterococcus spp. isolated from laying hens and eggs in conventional and organic keeping systems in Bavaria, Germany. Zoonoses Public Health 57:171–180.

Simões, L. C., M. Simões, R. Oliveira, and M. J. Vieira. 2007. Potential of the adhesion of bacteria isolated from drinking water to materials. J. Basic Microbiol. 47:174–183.

Stoodley, P., L. Hall-Stoodley L, B. Costerton, P. DeMeo, M. Shirtliff, and E. Gawalt. 2013. Biofilms, biomaterials, and device-related infections. Pages 556−583 in Handbook of Polymer Applications in Medicine and Medical Devices. K. Modjarrad, and S. Ebnesajjad, eds. William Andrew Publishing, Oxford, UK.

Oviedo, E. O. 2006. Important factors in water quality to improve broiler performance. N. Carolina Poult. Industry Joint Area Newsletter 1:7–8.

Park, H., Y. C. Hung, and D. Chung. 2004. Effects of chlorine and pH on efficacy of electrolyzed water for inactivating Escherichia coli O157:H7 and *Listeria* monocytogenes. Int. J. Food Microbiol 91:13–18.

Patterson, P. H., M. L. Sunde, and J. L. Pimentel. 1989. Water consumption and fecal moisture of laying hens fed

wheat middlings and corn-soybean-alfalfa meal diets. Poult. Sci. 68:830–833.

Petersen, L., and M. Madsen. 2000. *Listeria* spp. in broiler flocks: recovery rates and species distribution investigated by conventional culture and the EiaFoss method. Int. J. Food Microbiol. 58:113–116.

Poimenidou, S. V., M. Chrysadakou, A. Tzakoniati, V. C. Bikouli, G. J. Nychas, and P. N. Skandamis. 2016. Variability of *Listeria monocytogenes* strains in biofilm formation on stainless steel and polystyrene materials and resistance to peracetic acid and quaternary ammonium compounds. Int. J. Food Microbiol. 237:164–171.

Poppe, C. 1984. The Effect of Chlorination of Drinking Water on *Salmonella* Infection in Poultry. M.Sc. Thesis. University of Guelph, Canada.

Poppe, C., D. A. Barnum, and W. R. Mitchell. 1986. Effect of chlorination of drinking water on experimental *Salmonella* infection in poultry. Avian Dis 30:362–369.

Rasheed, S., I. Hashmi, and L. Campos. 2016. Inactivation of Escherichia coli and *Salmonella* with Chlorine in Drinking Waters at Various pH and Temperature Levels: Chlorine inactivation of *E coli* and *Salmonella*. Pages 83 −92 in Proceedings of the Pakistan Academy of Sciences: B. Life and Environmental Sciences: *53*.

Ryser, E. T., and H. E. Marth. 2007. *Listeria*, Listeriosis, and Food Safety. 3rd ed. CRC Press, New York.

Tawakoli, P. N., K. T. Ragnarsson, D. K. Rechenberg, D. Mohn, and M. Zehnder. 2017. Effect of endodontic irrigants on biofilm matrix polysaccharides. Int. Endodontic J. 50:153–160.

Toyofuku, C., M. S. Alam, M. Yamada, M. Komura, M. Suzuki, H. Hakim, N. Sangsriratanakul, D. Shoham, and K. Takehara. 2017. Enhancement of bactericidal effects of sodium hypochlorite in chiller water with food additive grade calcium hydroxide. J. Vet. Med. Sci. 79:1019–1023.

Ueno, C. M., C. L. Mullens, J. H. Luh, and W. A. Wooden. 2018. Historical review of Dakin's solution applications. J. Plastic Reconstruct. Aesth. Surg. 71:e49–e55.

Virto, R., P. Manas, I. Alvarez, S. Condon, and J. Raso. 2005. Membrane damage and microbial inactivation by chlorine in the absence and presence of a chlorine-demanding substrate. Appl. Environ. Microbiol. 71:5022–5028.

Wanja, D. W., P. G. Mbuthia, R. M. Waruiru, L. C. Bebora, H. A. Ngowi, and P. N. Nyaga. 2020. Antibiotic and disinfectant susceptibility patterns of bacteria isolated from farmed fish in kirinyaga county, Kenya. Int. J. Microbiol. 2020:8897338.

Watkins, S. 2006. Clean water lines for flock health. Avian Advice 8:3–5. Accessed Oct. 2023. https://www.thepoultrysite.com/articles/clean-water-lines-for-flock-health.

Watkins, S. 2008. Water: Identifying and correcting challenges. Avian Advice 10:10–15. Accessed Oct. 2023. https://www.thepoultrysite.com/articles/water-identifying-and-correcting-challenges.

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Page 3: Guidelines for Canadian Drinking Water Quality: Guideline Technical Document – Chlorine

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Part II. Science and Technical Considerations

4.0 Identity, properties, and uses

4.1 Occurrence and physical properties

Chlorine is a chemical element (symbol Cl) belonging to Group 17 (formerly VIIA), with an atomic weight of 35.457 (White, 1999). In nature, chlorine is found only as chloride ion (Cl^-), with a valence of -1 (White, 1999). Chloride makes up approximately 1.9% of the mass of seawater). Most chloride salts are soluble in water, so soluble chloride is usually found in abundance only in dry areas or deep underground. Chloride ion is abundant in nature and necessary to most forms of life, including mammals. Molecular chlorine (Cl_2) does not exist naturally, but can be produced industrially by electrolysis of sodium chloride dissolved in water or by hydrogen chloride oxidation processes (Connell, 1996; White, 1999). Once produced, it is collected, purified, compressed, and cooled; it is then stored and shipped as a pressurized liquefied gas. Cl_2 solidifies at -101.5°C and boils at -34.0°C at standard atmospheric pressure (Connell, 1996; White, 1999). As chlorine gas, it is greenish yellow, 2.5 times as heavy as air (3.2 g/L at 0°C and 101.3 kPa), and extremely irritating to mucous membranes (White, 1999). Chlorine gas is considered to be slightly soluble in water: 14.6 g/L at 0°C (U.S. EPA, 1994c).

Hypochlorite salts (solid sodium hypochlorite, NaOCl) are also commonly used in drinking water treatment. NaOCl, also known as chlorine bleach, bleaching solution, or Javelle water, is commercially prepared by chlorinating aqueous sodium hydroxide solutions and reduced temperatures (IARC, 1991). For stability reasons, commercial solutions are prepared at concentrations of 5-15%. Calcium hypochlorite ($Ca(OCl)_2$), also known as chloride of lime, tropical bleach, bleaching powder, or granular bleach, is easy to handle and transport. Both $Ca(OCl)_2$ and NaOCl must be stored in cardboard fully open to prevent deterioration; NaOCl must be protected from air, light, decreases in pH, and presence of heavy metal ions (Connell, 1996), whereas $Ca(OCl)_2$ must be protected from air, organic materials, and moisture.

4.2 Uses

The major uses of chlorine are in manufacture of chlorinated organic chemicals (e.g., vinyl chloride monomer, carbon tetrachloride, perchloroethylene, 1,1,1-trichloroethane, and chlorobenzenes), non-chlorinated organic chemicals (e.g., propylene oxide and glycols), and chlorinated inorganic chemicals (e.g., sodium hypochlorite, hydrochloric acid, and hypochlorous acid). It is also widely used as a bleaching agent in manufacture of pulp and paper; in bleaching textiles and fabrics; in manufacture of polysilicon, adhesives, and pharmaceuticals; for drinking and swimming pool water disinfection; for sanitation of industrial and sewage wastes; and in degassing of ammonium chloride (Curran et al., 1991). In its liquid and solid forms, chlorine is a powerful oxidizing, bleaching, and disinfecting agent. In its gaseous and liquid forms, chlorine is commonly used to inactivate microbial pathogens found in drinking water supplies.

Only a small percentage of chlorine produced worldwide is utilized in water treatment; in United States, 6% of all chlorine produced domestically is used for purposes of treating water (White, 1999). Chlorine is most commonly used disinfectant in world for treating drinking water (WHO, 1997) owing to its effectiveness and efficiency, germicidal properties, ease of application, measurement, and control, persistence, and low cost (IARC, 1991; Connell, 1996; White, 1999). Thus, use of chlorine in treatment of drinking water plays a major role in reducing overall environmentally mediated waterborne diseases, such as typhoid fever, cholera, dysentery, and other gastrointestinal diseases, in developed countries (IARC, 1991; Burlingame, 2000). Chlorine can also be added to drinking water systems to prevent algae, fungae, and bacteria growth, to control biological growth in distribution systems, to maintain certain fixed diameter treatment plant pipe capacity, to preserve pipe capacity, to disinfect water mains, and to control tastes and odours (White, 1999; CCOHS, 2004a). When chlorine is used for drinking water treatment purposes, type of chlorine selected depends on a number of factors, including cost, availability, equipment maintenance, and ease of application. For example, chlorine gas is approximately three times less expensive than hypochlorites (White, 1999), but many times more difficult to use than hypochlorites; calcium hypochlorite can contribute to scaling problems during water treatment but is less expensive than sodium hypochlorite. While sodium hypochlorite solution is difficult to transport, it is often preferred because its easy and dedicated gives rise to maintenance problems with pumping and feeding equipment (White, 1999). Calcium hypochlorite is also commonly used for disinfection of rural and household community water supplies (WHO, 1997) and for swimming pool sanitation (CCOHS, 2004a; Wojtowicz, 2004).

Chlorine i e limite foo itive, ch ble ching gent in flo r n t rche ; it e in flo r i reg l te ccor ing to Goo Man f ct ring Pr ctice St n r (He lth C n , 1993 . Chlorine or hypochlorite lt re l o commonly e ring foo proce ing to i infect w ater pplie n control microbi l gent . For ex mple, chlorin te w ater i e in the w a hing n proce ing of re me t, po ltry, n fi h, well pro ce (CFIA, 2004, 2005, 2007 . Chlorin te w ater i l o e to nitize foo eq ipment n f cilitie ring foo proce ing (WHO, 2003; CFIA, 2004 . De pite permitte e in foo proce ing, chlorine mu t be rin e o t or otherwi e remove from cont ct with foo .

4.3 Terminology

Thi ection provi e efinition for ome relev nt term e in thi oc ment, pte from the Americ n Water Work A oci tion (AWWA, 1999; Symon et l., 2000 :

- *Chlorine residual:* the concentr tion of chlorine pecie pre ent in w ater fter the oxi nt eman h been ti fie .
- *Free chlorine:* the mo nt of chlorine pre ent in w ater i olve g (Cl_2 , hypochloro ci (HOCl , n /or hypochlorite ion (OCl^- th t i not combine with mmoni or other compo n in w ater.
- *Combined chlorine:* the m of the pecie re lting from the re ction of free chlorine with mmoni (NH_3 , incl ing monochlor mine (NH_2Cl , ichlor mine ($NHCl_2$, n trichlor mine (nitrogen trichlori e, NCl_3 .
- *Total chlorine:* ll chemic l pecie cont ining chlorine in n oxi ize t te. U lly the m of free n combine chlorine concentr tion pre ent in w ater.
- *Primary disinfection:* the pplic tion of i infect nt t the rinking w ater tre tment pl nt, with primary objective to chieve the nece ry microbi l in ctiv tion.)
- *Secondary disinfection:* the b eq ent pplic tion of i infect nt, either t the exit of the tre tment pl nt or in the i trib tion y tem, with the) objective of en ring th t i infect nt re i l i pre ent thro gho t the i trib tion y tem.

4.4 Chemistry in aqueous media

When e to w ater, chlorine g (Cl_2 i olve r pi ly n e t bli he n eq ilibri m with hypochloro ci (HOCl , ccor ing to chemic l) eq tion (1 :

$$Cl_2 + H_2O \leftrightarrows H^+ + Cl^- + HOCl \quad (1)$$

A ition of N aOCl n C (OCl 2 to w ater chieve the me e enti l oxi izing gent, HOCl, ccor ing to chemic l re ction (2 n (3 below (IARC, 1991; Connell, 1996; White, 1999 , with the only ifference being i e re ction n en pro ct :

$$NaOCl + H_2O \leftrightarrows NaOH + HOCl \quad (2)$$

$$Ca(OCl)_2 + 2H_2O \leftrightarrows Ca(OH)_2 + 2HOCl \quad (3)$$

HOCl then i oci te to neg tive hypochlorite ion (OCl^- ccor ing to chemic l eq tion (4 :

$$HOCl \leftrightarrows H^+ + OCl^- \quad (4) \qquad pK_a = 7.5$$

All of the e chemic l re ction , n th i infection effectivene , re highly epen ent pon the pH n temper t re of the q eo me i m, which etermine the extent of conver ion between the three free chlorine pecie : Cl_2, HOCl, n OCl^-. HOCl i con i ere to be more effective t microbi l in ctiv tion n omin te t lower pH level . For ex mple, t pH of 6.5 n temper t re of 0°C n 20°C, n q eo ol tion of chlorine wo l cont in bo t 95.5% n 92.4% HOCl, re pectively (4.5% n 7.6% of OCl^- ; t higher pH of 8.5, the eq ilibri m hift to 17.5% n 10.8% of HOCl (82.5% n 91.2% of OCl^- . The typic l pH r nge of rinking w ater i between 6.5 n 8.5, n chlorin tion of rinking w ater t pH level below 8 provi e maximum i infection efficiency (White, 1999; IPCS, 2000; WHO, 2004 . However, efficiency c n l o be incre e by incre ing cont ct time, concentr tion, or temper t re (U.S. EPA, 1999b, 2007 .

4.5 Application to drinking water treatment

Thi G i eline Technic l Doc ment foc e on the he lth effect rel te to expo re to chlorine in rinking w ater pplie . It oe not review the benefit or the proce e of chlorin tion, nor oe it e the he lth ri k rel te to expo re to by-pro ct forme re lt of the chlorin tion proce .

4.5.1 Chlorine in water treatment

Drinking w ter o rce re often cont min te with v riety of p thogenic org ni ms, incl ing enteric vir e , b cteri , n protozo , th t co l be re pon ible for o tbre k of w aterborne i e e (White, 1999 . Chlorine c n be e both primary)n econ ry i infect nt. The prim ry p rpo e of chlorin ting rinking w ater i i infection, thro gh the e tr ction or in ctiv tion of p thogenic org ni ms pre ent (Connell, 1996 . The U.S. Center for Di e e Control n Prevention (U.S. CDC, 1999 h ve i entifie the control of infectio i e e thro gh cle n w ater n improve nit tion one of the 10 gre t p blic he lth chievement of the 20th cent ry. The intro ction of rinking w ater tre tment e rly in the 20th cent ry, incl ing i infection with chlorine, h yiel e r tic re ction in the r te of illne n e th from w aterborne p thogen (C tler n Miller, 2005 .

The mech ni ms by which chlorine in ctiv te microbiologic l org ni ms re not f lly n er too . Chlorine i r ption of the cell membr ne eems to be f n ment l event le ing to the in ctiv tion of the org ni ms. Thi i r ption may incl e btle event ch nco pling of the electron ch in n incre e to membr ne perme bility re lt of ifference in electric l ch rge between the i infect nt n the org ni m. Thi le to enzyme in ctiv tion or rele e of vit l cell l r con tit ent n , ltimately, cell e th (Connell, 1996; White, 1999; Virto et l., 2005 .

Other chemic re ct s ccurr g the w ater w i ffect the mou t f ch r e requ red f r d s fect . I rg c c mp u ds, such s mmo , r , d ma g ese, w i re ct r p d y w th free v b e ch r e, where s tur rg c c mp u ds, such s humic d fu v c cds d g mater , may re ct s w ly. Theref re, ch r e s ge er y dded excess f the dema d f r d s fect thr ugh ut the d str but system (IP S, 2000; Sym s et ., 2000).

4.5.2 Primary disinfection

Pr mary d s fect s the pp c t f d s fect t the dr k g w ater tre tme t p t, w th pr mary bject ve t ch eve the ecess ry micr b ct v t . The eff c cy f d s fect us g ch r e c be pred cted b sed k wledge f the res du free ch r e c ce tr t , temper ture, pH, d c t ct t me. Th s re t sh p s c mmo y referred t s the T c cept d s used by pub c dr k g w ater supp ers s e t f r e sur g dequ te ct v t f rg sms dur g d s fect . T s the pr duct f the res du c ce tr t f d s fect t ( ), me sured mg/L t the ut et f the c t ct ch mber, d the d s fect t c t ct t me (T), me sured mi utes. The T v ues requ red t ch eve the ecess ry ct v t w i depe d the micr rg sm t rgeted, pH, d temper ture. More f rmat T v ues typ c y requ red t ct v te *E. coli* (He th d , 2006), e ter c v ruses (He th d , 2004), d cert pr t z (*e.g., Giardia*) (He th d , 2004b)

Other f ct rs th t may f ue ce d s fect eff c e cy c ude c t ct ch mber des g , dequ te mix g, d prese ce f su ght (U.S. EPA, 1999 ; Wh te, 1999).

4.5.3 Secondary disinfection

Sec d ry d s fect may be pp ed t the tre ted w ater s t e ves the tre tme t p t r t rech r t p ts thr ugh ut the d str but system, t tr duce d ma t ch r e res du the dr k g w ater d str but system. Over , ch r e res du pr v des tw oma be ef ts:

1. It c mit the gr wth f b f m w th the d str but system d ts ss c ted t ste d d ur pr b ems (Le hev er, 1998; Trusse , 1999; Wh te, 1999).
2. A r p d dr p d s fect t res du may pr v de mmed te d c t f tre tme t pr cess ma fu ct r bre k the tegr ty f the d str but system (Le hev er, 1998; H s, 1999; He th d , 2006 , 2006b).

A ch r e res du may s reduce the r sk f w despre d micr b g c c t mi t the eve t f trus t the d str but system. H wever, th s pr tect ve effect depe ds he v y the mag tude f the eve t d the suscept b ty f the c t mi t g micr rg sms t ch r e. Veget t ve b cter p th ge s such s *E.coli* O157:H7 re re d y ct v ted by ch r e res du s, where s ch r e-res st t rg sms such s *Giardia* d *Cryptosporidium spp*. w ou d be expected t rema u ffected (H s, 1999; P yme t, 1999; AwwaRF, 2005). I ge er , w ater e v g tre tme t p t sh u d be tested d y f r b th ch r e res du d turb d ty d sh u d be tested t e st week y f r t t c f rms, t mo t r per t dequ cy, d f r *E. coli*, t c f rm the micr b g c s fety f the supp y (He th d , 2006 , 2006b). I the d str but system, the prese ce f dequ te ch r e res du s sh u d be c f rmed whe s mp g f r t t c f rms d *E. coli.*

G ve the per t be ef ts f sec d ry d s fect , per t rs sh u d str ve t ma t ch r e res du thr ugh ut the system t c tr regr wth d t pr v de d c t f system tegr ty. It s rec g zed th t th s may be d ff cu t w-f w re s such s de d-e ds r extreme p rts f the d str but system d th t t may e d t the ge er t f u ccept b e eve s f DBPs d c u d requ re the mp eme t t f ppr pr te c tr str teg es.

d d t bt ed 2005 fr m 3590 dr k g w ater f c t es c ted e pr v ces d terr t r es d c te th t s d um hyp ch r te s the most c mmo d s fect t used f r d s fect 78% f the p ts, where s 19% used ch r e g s, 1.4% used c c um hyp ch r te, d ess th 0.5% pp ed ter t ve d s fect ts. D t pr v ded by s x pr v ces d terr t r es d c te th t typ c eve s f free ch r e d dr k g w ater systems r ge fr m 0.4 t 2.0 mg/L e v g the tre tme t p t, fr m 0.4 t 1.2 mg/L t termed te p ts the d str but system, d fr m 0.04 t 0.8 mg/L t the f r e d f the d str but system.

Requ reme ts f r ch r e res du c ce tr t s re determi ed by the resp s b e uth r ty d may v ry mo gst the pr v ces d terr t r es. Most jur sd ct s spec fy mi mum eve f free ch r e res du th t sh u d be pp ed t the tre tme t p t d/ r detect b e w th the d str but system. I most pr v ces d terr t r es, h gher ch r e res du s c be permitted by the regu t ry uth r ty, s deemed ecess ry c se-by-c se b s s. The U.S. E v r me t Pr tect Age cy (EPA) Surf ce Water Tre tme t Ru e requ res mi mum d s fect t res du f 0.2 C mg/L f r w ter e ter g the d str but system d th t detect b e eve be ma t ed thr ugh ut the d str but system (U.S. EPA, 2002).

The Wor d He th Org z t (WHO) h s suggested th t, f r re s w th tt e r sk f ch er r re ted utbre ks, free ch r e res du r ge f 0.2-0.5 mg/L be ma t ed t p ts the supp y (WHO, 1997). I ge er , free ch r e res du f 0.2 mg/L s c s dered mi mum eve f r the c tr f b cter regr wth the d str but system (Le hev er et ., 1996).

I s me w ter systems, ch r e s c mb ed w th mmo w th the te t t f rm mo ch r mi e, wh ch s used s sec d ry d s fect t. More f rmat ch r mi es c be f u d the c rresp d g Gu de e Tech c D cume t (He th d , 1996)

4.5.4 Formation of chlorinated disinfection by-products

S me f the tur rg c matter the tre ted w ater h s the p te t t re ct w th ch r e t f rm DBPs t the p t d w th the d str but system. The types d structures f DBPs re c mp ex d re fu ct f w ater qu ty d tre tme t c d t s (IP S, 2000). The most c mmo DBPs prese t ch r ted w aters c ude tr h meth es (THMs) d h ge ted cet c c ds (HAAs). Bec use e ev ted eve s f DBPs may h ve dverse effects he th (WHO, 1995; U.S. EPA, 1999 ; He th d , 2000; IP S, 2000), every eff rt sh u d be made t ma t the r

concentration a o a rea ona y achieva e, thout compromi ing the effectivene of di infection. Thi can e done u ing trategie uch a precur or contro and remova or app ication of a ternative/modified di infection practice (IPCS, 000), inc uding optimization of the treatment proce .

Thi document doe not di cu the hea th effect of expo ure to the variou di infection y-product . Other document have een deve oped for pecific CDBP , inc uding Guide ine Technica Document for THMs (Hea th Canada, 006c), ch oramine (Hea th Canada, 1996), ch orite/ch orate (Hea th Canada, 008a) and HAA (Hea th Canada, 008), a ea a Guidance document for ch ora hydrate (Hea th Canada, 008c). In addition, exten ive revie are avai a e in the cientific iterature (Cantor, 1997; IPCS, 000; Vi anueva et a ., 003).

4.5.5 Taste and odour considerations

Whi e ch orination can he p improve ta te and odour through the reaction thi organic materia and iron (Conne , 1996), it can a o generate ch orinou f avour cau ed y the pre ence of the di infectant it e f or y the occurrence of other CDBP formed y the reaction thi other compound in the tar. For examp e, the reaction of ch orine thi certain nitrogen compound (e.g., amino acid , ammonium, urea) pre ent in ource tar may ead to the formation of trong- me ing compound uch a a dehyde , nitri e , and ome ch oramine , ich can cau e pronounced ch orinou ta te and odour , ometime even at very o eve . Ch oropheno , re u ting from the reaction of ch orine thi pheno ic compound , can e formed at the p ant or in the di tri ution y tem and can impart ta te and odour to the tar.

In a Nationa Toxico ogy Program (NTP) rodent tudy (NTP, 199), aver ion to the ta te of ch orine ademon trated to e do e re ated (U.S. EPA, 1994a). WHO (1997) noted that aver ion to the ta te of ch orine in drinking tar cou d ead human popu ation to reject a ource of tar that i actua y afe to drink. WHO (004) a o indicated an increa ed ri k of unaccepta i ity at free ch orine re idua et een 0.6 and 1.0 mg/L. The Au tra ian NHMRC (004) reported an odour thre ho d of 0.6 mg/L for ch orine in drinking tar, emrea a tudy y A RF(a 004) ugge ted thre ho d a o a 0.05-0.1 mg/L. It i c ear that there i a de variа i ity of ta te and odour thre ho d in the popu ation, depending on individua en itivitie . Ho ver, a urvey conducted in U.S. and Canadian drinking tar p ant found that ch orine a ti the dominant cau e of odour comp aint and a ignificant cau e of ta te comp aint from con umer (Suffet et a ., 1996).

A recent cro -country urvey of 1750 peop e in the United State examined pu ic perception of ch orinou f avour in tap tar and found that the majority of con umer re ati fied thi their municipa tap tar. Ho ver, ta te aoften the cau e of higher con umer di ati faction, and the mo t common off-f avour reported in wdrinking ter a"ch orinou " (15.5% for ta te, 14.8% for odour) (A RF,a 004). In thi tudy, average thre ho d en itivity to the ta te of free ch orine a0.8 mg/L, thi 46% una e to detect free ch orine at the highe t re idua concentration (~1 mg/L) in their hou eho d tap tar.

Suffet et a . (1996) indicated that it i po i e that ch orine odour pro em s are produced y other compound and are not a re u t of the di infection proce . A though con umer may report a ta te or odour pro em a "ch orinou ," it hou d e noted that they may e confu ing ch orinou ta te and odour thi ta te and odour from CDBP (A RF,a 004). Thu , in many ca e , it i difficu t to accurate y identify con umer ' ta te and odour pro em s a ed o e y on their de cription of the ta te and odour .

5.0 Exposure

Becau e ch orine i not ta e in the environment, expo ure i not expected to e ignificant, and there are fe data avai a e.

5.1 Air

There are no data avai a e on concentration of ch orine in indoor air; ho ver, ga eou ch orine i e timated to e at o eve in ambient air: 1-3.7 mg/m^3 (0.344-1. 7 ppm) (U.S. EPA, 1994 ; WHO, 004).

5.2 Drinking water

Human expo ure to ch orine re u t primari y from the inge tion of free ch orine pre ent in treated drinking tar (U.S. EPA, 1994a; WHO, 004). Vaporization of ch orine at o concentration into the air from drinking tar i not con idered to e re evant (U.S. EPA, 1994 ; IPCS, 000). Simi ar y, the amount of ch orine re ea ed from di ute odium hypoch orite o ution into the air under norma u age condition i not con idered ignificant (CCOHS, 004c).

5.3 Food

Ch orine and hypoch orite a t o ution are common y u ed during food proce ing to di infect tar upp ie and contro micro ia agent . Fre h produce i permitted to e ed thi ch orinated n tar containing free ch orine re idua eve et een and 7 mg/L (or 100-150 mg tota ch orine/L); exce amount of n tar mu t e ater removed from the produce (CFIA, 005). Ch orinated tar i a o u ed in red meat, pou try, and fi h proce ing. Water in contact thi eef carca e i permitted to contain a maximum tota avai a e ch orine eve of 0 mg/L or a maximum eve of 10 mg/L for tota avai a e ch orine a hypoch orou acid (CFIA, 004). Beef mu t then e fo o de y a rin e thi pota e tar or a imi ar appropriate mea ure to en ure that re idue re u ting from treatment are neg igi e (CFIA, 004). Pou try carca e and part are a o permitted to e dipped, prayed, or ed thi tar containing 0-50 mg tota avai a e ch orine/L (CFIA, 004) or up to 10 mg/L for tota avai a e ch orine a hypoch orou acid, provided that treatment i fo o de y a rin e thi pota e tar. In fi h proce ing, re idua ch orine may not exceed 10 mg/L n the tar icome into direct contact thi fi h; ho ver, higher concentration may e u ed for anitation, provided that the tar doe not come into direct contact thi fi h (CFIA, 007).

No data ex t o hlo ne e due n ood. Howeve , due to the wate olub l ty and h gh ea t v ty, hlo ne and hypo hlo te alt a e not expe ted to a umulate o b o on ent ate n the ood ha n (ATS R, 2002, 2007; UNEP, 2003). The e o e, the e no ea on to expe t that e due above natu ally o u ng ba kg ound level would be ound n ood (U.S. EPA, 1999a).

5.4 Swimming pools and hot tubs

Chlo ne and hypo hlo te alt a e al o u ed o the d n e t on o w imming pool and hot tub . Tho e who w im o u e a hot tub equently ould have g eate de mal and po bly nhalat on expo u e to hlo ne and C BP (U.S. EPA, 1994a). Howeve , o the pu po e o th do ument, hlo ne expo u e om w imming pool and hot tub w ll not be evaluated.

6.0 Analytical methods

The U.S. EPA ha app oved eve al method , ba ed on olo met (P), ampe omet , odomet , and y ngaldaz ne method , o the dete minat on o ee, total, and omb ned hlo ne n d nk ng wate (Table 1).

The P olo met method o e dual hlo ne the mo t w idely u ed to dete mine ee and total hlo ne. The ampe omet t t at on te hn que equ e a h ghe deg ee o k ll and a e than the olo met method. The odomet method le en t ve than the ampe omet method but u table o mea u ng total hlo ne on ent at on h ghe than 1 mg/L. The y ngaldaz ne method a olo met / pe t ophotomet method pe o the analy o ee hlo ne.

Othe method n lude Standa d Method 4500-Cl B p opo ed by the Ame an Publ Health A o at on, whe e the min mum dete table on ent at on app ox mately 0.04 mg/L. Fo th method, a d t t at on (pH 4) p e e ed, be au e ome o ms o omb ned hlo ne do not ea t at no mal d nk ng wate pH ond t on (APHA et al., 2005). In add t on, method app oved by the Inte nat onal O gan zat on o Standa d zat on (ISO, 2006) o the dete minat on o ee hlo ne and total hlo ne n lude ISO 7393-1 (1985) (t t met), ISO 7393-2 (1985) (olo met), and ISO 7393-3 (1990) (odomet t t at on).

Table 1: U.S. EPA-approved analytical methods for chlorine

Methodology>	Method [a]	Chlorine residual measured (MDC)	Comments
DPD colorimetric D	SM 4500-Cl G [1] D	F ee, omb ned, total D (0.010 mg/L)	Inte e en e : ox d zed mangane e; h gh D o gan ontent
	EPA 330.5 [3] D	Total (0.2-4 mg/L)	
DPD ferrous D	SM 4500-Cl F [2]	F ee, omb ned, total (0.018 mg/L) D	Inte e en e : ox d zed mangane e and oppe ; omb ned hlo ne o > 0.5 mg/L an g ve h gh [Cl]
	EPA 330.4 [3] D	Total (NA)	
Amperometric D	SM 4500-Cl [1]	ee, omb ned, total (NA)	Inte e en e : hlo amine an g ve h gh [Cl]; ve y low tempe atu e equ e long t t at on t me; p e en e o oppe and lve an au e ele t ode to mal un t on; mangane e, on, and n t te an be min mized by a d at on
	ASTM 1253-03 [4] D	Total (NA) D	
	EPA 330.1 [3]	Total (NA)	
	EPA 330.2 [3] D	Total (NA)	
Low-level amperometric	SM 4500-Cl E [1] D	Total (0.010 mg/L)	Cannot d e ent ate between ee and omb ned hlo ne
Iodometric electrode	SM 4500-Cl I [1]	Total (> 1 mg/L)	Inte e en e : mangane e and othe ox dant
	EPA 330.3 [3] D	Total (> 1 mg/L)	

FACTS, F ee ava lable hlo ne te t; M C, Min mum dete table on ent at on; NA, Not ava lable

a D Sou e a e a ollow s:

1 D Standa d Method (APHA et al., 2005).

2 D Standa d Method (APHA et al., 1998).

3 D U.S. EPA method a e ava lable o download at www.nemi.gov/.

4 D ASTM Inte nat onal (ASTM, 2006).

Methodolog 0	**ethod** [a]	**hlo ne es dual measu ed (MD) C** 0	**omments**
S ngaldaz ne 0 **(FA TS)**	SM 45 -Cl H 0 [1] 0	Free (.1 m 0 g/L)	Interferences: None reported 0

FACTS, Free available chlorine test; MDC, Minimum detectable concentration; NA, Not available

a 0 Sources are as follows:

1 0 Standard Methods (APHA et al., 2 5).

2 0 Standard Methods (APHA et al., 1998).

3 0 U.S. EPA methods are available for download at www.nemi.gov/.

4 0 ASTM International (ASTM, 2 6).

Because chlorine is not stable in water, the chlorine content of samples will decrease with time. Therefore, chlorine should be determined immediately after sampling (APHA et al., 2 5); for samples collected in the distribution system, it is preferable to conduct the analysis in the field using a field test kit (Harp, 2 2). Field test kits are based on the DPD colorimetric method for measuring free or total chlorine in water. The use of automatic colorimeters eliminates the human error associated with colour matching. The visual comparators for free and total chlorine include colour cube (range .1-2.5 mg/L) and colour disc (-3.5 mg/L). Pocket colorimetry kits may allow the determination of free chlorine (. 2-2. mg/L) or total chlorine (-4.5 mg/L), whereas spectrophotometers may allow chlorine analysis in the range .1-1 mg/L, depending on the model. A digital titrator based on the DPD-FAS (ferrous ammonium sulphate) method can also be used for field determination of chlorine in the concentration range . 1-3. mg/L. When using the DPD colorimetric test, it is important to ensure that field staff are well trained to do both free and total chlorine measurements. This ensures that false positive results are not inadvertently reported if there is a monochloramine residual present (Pon, 2 8). A monochloramine residual is due to the presence of ammonia in either the supply (naturally occurring) or the distribution system (naturally occurring or use of chloramine).

Special analysers are often used to control the feed rate of chlorine and monitor chlorine residuals online. The analysers use amperometric titration, colorimetric, or oxidation-reduction potential probe methods.

Free and combined forms of chlorine may be present simultaneously in chlorinated water. Chloramines are the combined forms resulting from the 0 reaction of chlorine with naturally occurring ammonia or ammonia added as part of the water treatment strategy. Total chlorine is the combination of free and combined chlorine.

7.0 Treatment technology

7.1 Municipal scale

The amounts of chlorine used have to be integrated in the overall optimization of the water treatment process. Removal of contaminants that increase the chlorine demand, including precursors of CDBPs, will reduce both the quantity of chlorine added to the water and the production of CDBPs.

Control of the chlorine dosage in drinking water requires effective control of the feed rate of chlorine. Computerized control systems have been developed to determine the amount of chlorine that needs to be applied for a given water by combining inputs from several measurements, including flow and residual level in treated water. This is usually known as compound loop control (MWH, 2 5). Proper design, operation optimization, and equipment maintenance are the critical points identified for an efficient chlorination process, both at the treatment plant and in the drinking water distribution system.

Some water treatment facilities that include a "superchlorination" treatment step will subsequently require the dechlorination of the water to a system-specific operational range before distribution (White, 1999). This is usually done by the addition of chemicals, such as sulphur dioxide or sodium bisulphite, to the water.

Chemicals used for chlorination and dechlorination should be certified as meeting NSF International (NSF)/American National Standards Institute (ANSI) Standard 6 : Drinking Water Treatment Chemicals -- Health Effects, which is the recognized health effects standard for chemicals used to treat drinking water and includes certification criteria for chlorine, calcium hypochlorite, sodium hypochlorite, and dechlorination chemicals.

7.2 Residential scale

Generally, it is not necessary to use drinking water treatment devices with municipally treated water. The use of residential-scale treatment devices on municipally treated water is based primarily on individual choice. In cases where an individual household obtains its drinking water from a private well, there may be circumstances where chlorination of the well is warranted. In such cases, monitoring of the chlorine residual would also be 0

recommended. e h C n d s rong y recommends h chemic s used for ch orin ion be cer ified s mee ing NSF/ANSI S nd rd 60: rinking Wa er Tre men Chemic s -- e h Effec s. This is he recognized he h effec s s nd rd for chemic s used o re drinking w a er.

Priv e residen i drinking w a er re men devices may be n op ion for reducing ch orine concen r ions in drinking w a er if he consumer finds he s e objec ion b e. isinfec ion of we wi h ch orine for emergency or regu r main en nce requires higher ch orine concen r ions h n he dos ge used for rou ine disinfec ion. owever, re men devices h reduce ch orine re no in ended o be used o remove high concen r ions of ch orine.

A he residen i sc e, he w a er re men echno ogy for he reduc ion of ch orine is dsorp ion on o c iv ed c rbon, nd he pp ic b e s nd rd is NSF/ANSI S nd rd 42: rinking Wa er Tre men Uni s -- Aes he ic Effec s. This s nd rd es b ishes minimum requiremen s for ma eri s, design nd cons ruc ion, nd performance of drinking w a er re men sys ems h re designed o reduce specific es he ic-re ed (s e nd odour) con min n s (NSF/ANSI, 2002). For drinking w a er re men device o be cer ified o NSF/ANSI S nd rd 42, he device shou d reduce he concen r ion of ch orine in w a er by minimum of 50% from n inf uen concen r ion of 2 mg/L.

Tre men devices ose heir remov c p ci y hrough us ge nd ime nd need o be main ined or rep ced. Consumers shou d verify he expec ed ongevi y of he componen s in heir drinking w a er re men device in he manuf c urer's recommend ions nd service hem when required.

e h C n d does no recommend specific br nds of drinking w a er re men devices, bu i s rong y recommends h consumers ook for mark or be indic ing h he device or componen s h ve been cer ified by n ccredi ed cer ific ion body s mee ing he ppropri e NSF/ANSI drinking w er ma eri s s nd rds. These s nd rds h ve been designed o s fegu rd drinking w a er by he ping o ensure he ma eri s fe y nd performance of produc s h come in o con c wi h drinking w a er. Cer ific ion org niz ions provide ssur nce h produc conforms o pp ic b e s nd rds nd mus be ccredi ed by he S nd rds Counci of C n d (SCC). In C n d , he fo owing org niz ions h ve been ccredi ed by he SCC o cer ify drinking w a er devices nd ma eri s s mee ing NSF/ANSI s nd rds:

- C n di n S nd rds Associ ion In ern ion
- NSF In ern ion
- Wa er Qu i y Associ ion
- Underwri ers L bor ories Inc.
- Qu i y Audi ing Ins i u e
- Associ ion of P umbing & Mech nic Offici s. An up- o-d e is of ccredi ed cer ific ion org niz ions c n be ob ined from he S nd rds Counci of C n d .

8.0 Kinetics and metabolism

Ph rmacokine ic s udies of C_2, OC , nd OC - h ve been performed using r dio be ed ch orine compounds in r s nd re summarized be ow. owever, bec use ch orine mo ecu es re so re c ive in bio ogic sys ems, he resu s may ref ec he presence of me bo ic re c ion by-produc s nd o her ch orin ed compounds more so h n he specific oxicokine ics of free ch orine, due o he oxid ive re c ivi y of he v rious compounds (U.S. EPA, 1994 , 1994c).

8.1 Absorption

In number of s udies, b ood s mp es were co ec ed from ma e r s dminis ered r dio be ed OC by g v ge (Abde -R hman e ., 1982 , 1983, 1984). These s udies demons r ed h $O^{36}C$ w as quick y bsorbed hroughou he body ow doses. The r e cons n for bsorp ion of $O^{36}C$ w as de ermined o be 0.157 ± 0.001/hour, nd he h f- ife of bsorp ion w as 4.42 ± 1.31 hours (Abde -R hman e ., 1982). In no her s udy of f s ed nd non-f s ed Spr gue- w ey r s, or bsorp ion r e cons n s for $O^{36}C$ were 0.322/hour nd 0.316/hour for f s ed nd non-f s ed r s, respec ive y, where s bsorp ion h f- ives were 2.2 hours for bo h groups (Abde -R hman e ., 1983). I h s been pos u ed h he r nge of differences in bsorp ion of ^{36} ref ec s he endency for ch orine species o re c wi h org nic ma eri in he b ood or g s roin es in r c of non-f s ed nima s o form diverse ch orine compounds (U.S. EPA, 1994c).

8.2 Metabolism

The oxidizing po en i of ch orine w as observed in he g s roin es in r c of roden s. In pre imin ry experimen wi h no qu n i ive n y ic resu s given, six ma e Spr gue- w ey r s were given 56 mg of N aOC so u ion (p 7.9) by g v ge, equiv en o 140 mg/kg body weigh (bw) (Mink e ., 1983). Af er 1 hour, ei her rich oro ce ic cid (TCA) or dich oro ce ic cid (CA) w as de ec ed in he s omachs of six dosed r s nd in cer in p sma s mp es. Ch oroform w as de ec ed in he s omachs of r s, bu on y in he p sma of one r . ich oro ce oni ri e (CAN) w as so found in D he gu con en s of wo ou of hree nonf s ed r s, bu nei her he f s ed r s nor ny p sma s mp es con ined CAN. e ec ion of TCA (de ec ion imi 1.3 µg/mL) nd CA (de ec ion imi 0.3 µg/mL) in bo h f s ed nd nonf s ed r s indic ed h *in vivo* forma ion of hese ch orin ed ce ic cids w as no jus dependen on he in er c ions of N aOC wi h foreign org nic ma eri in he gu (Mink e ., 1983), hough he mech nism of c ion for forma ion of hese compounds h s no been de ermined. I h s been pos u ed h he bu k of he ch orin ed by-produc s formed in he g s roin es in r c remain s higher mo ecu r weigh produc s, which may h ve i e oxico ogic signific nce (IPCS, 2000).

8.3 Distribution and excretion

Administe ed d ses O^{36}Cl we e und t be quickly dist ibuted th ugh ut the b dy at l w d ses. A te 72 h u s, the highest c ncent ati ns ^{36}Cl we e und in plasma, at .77% the initial d se, and dec easing d ses we e und in the st mach, testes, lung, kidney, du denum, spleen, live , 0 b ne ma w, ca cass, and skin. The l west c ncent ati n was und in the ileum, at .14% (Abdel-Rahman et al., 1982a). In an the metab lism study in asted and n n- asted Sp ague-Dawley ats, a te 96 h u s, ^{36} l was dist ibuted at highest levels in the plasma, at 1.92 µg/g, ll wed by wh le bl d, at 1.59 µg/g, with the l west levels und in adip se tissue, at . 9 µg/g (Abdel-Rahman et al., 1983). Plasma c ntained ^{36} l activity 1.24% the administe ed d se, and packed cells had an activity .29%. The peak plasma level ^{36} l eached 1 .7 µg/mL at 4 h u s in n n- asted ats, whe eas the peak ^{36} l plasma level was btained in 2 h u s the asted ats at a c ncent ati n 7.9 µg/mL (Abdel-Rahman et al., 1983).

A study the subcellula dist ibuti n ^{36} l c mp unds und that 75% t tal ^{36} l activity was ec ve ed in the cyt s l, with 2.5% in the mic s mal, 1.5% in the nuclea , and < .1% in the mit ch nd ial acti ns, espectively. It appea s that a high pe centage t tal ^{36} l is l sely b und t the e yth cyte memb ane exchangeable with chl ide in saline (Abdel-Rahman et al., 1983).

Bl d samples ll wing gavage d sage O^{36}Cl t ats demonst ated that a te 72 h u s, the calculated ate c nstant O^{36}Cl eliminati n m plasma was . 9 ± . 1/ h u , and the hal -li e eliminati n was 77. ± 8.8 h u s (Abdel-Rahman et al., 1982a). In a late study, the eliminati n hal -lives we e dete mined t be 44.1 h u s and 88.5 h u s asted and n n- asted ats, espectively, whe eas eliminati n ate c nstants we e . 16/h u and . 8/ h u asted and n n- asted ats, espectively (Abdel-Rahman et al., 1983).

Radi labelled OCl administe ed t asted ats appea ed t be c nve ted t and eliminated c mpletely in the m chl ide. The maj ute exc eti n was via u ine, as app ximately th ee-qua te s the eliminated O^{36}Cl (21% the initial d se) in the m chl ide i n was ec ve ed in u ine, whe eas ne-qua te (7% the initial d se) was ec ve ed in aeces (Abdel-Rahman et al., 1982a). In an the study, within the i st 24-h u pe i d, 7. 5% the administe ed d se was exc eted in u ine and 7.45% was exc eted via the intestinal ute; by 96 h u s, this p p ti n had inc eased t 36% in u ine and 15% in aeces (Abdel-Rahman et al., 1983). ^{36} l c mp unds did n t appea t be eliminated via expi ed ai (Abdel-Rahman et al., 1982a).

9.0 Health effects

9.1 Effects in humans

Accidental ingesti n c mme cial s dium hyp chl ite bleach (5.25% 52 5 mg/L) is ne the most c mmon p is ning events in y ung child en. Intenti nal ingesti n has als been ep ted equently in adults. P is nings have esulted in va i us deg ees t xicity, including muc sal i itati n, nausea, v miting, dia h ea, c sive inju y t the es phagus and gast intestinal t act, acid sis, and even death (IPCS, 1997), alth ugh these e ects appea t be due mainly t additi nal chemicals p esent the ext eme alkalinity the p duct (well, 1991; IPCS, 1997). The lethal d se s dium hyp chl ite in humans has been ep ted t be ab ut 2 mL a s luti n c ntaining 31 5 -63 mg/L, alth ugh su vival patients wh swall wed up t 1 L a 5.25% (c esp nding t 52 5 mg/L) s luti n and ab ut 5 mL a 1 % (c esp nding t 1 mg/L) s dium hyp chl ite s luti n has been ep ted (Raci ppiet al., 1994). Even in the case misuse, chl ine bleach has nly slight t xicity and i itati n p tential, and ec ve y is ten apid and eve sible (Raci ppi et al., 1994; Babl et al., 1998).

Typical c ncent ati ns ee chl ine in d inking wate a e gene ally less than 1 mg/L, but humans have c nsumed hype chl inated wate sh t pe i ds time at levels as high as 5 mg/L with n appa ent adve se e ects (U.S. EPA, 1994c). An ea ly anecd tal ep t n ted that n adve se health e ects we e bse ved when 15 milita y pe s nnel c nsumed wate with chl ine levels 5 mg/L du ing a pe i d wate main disin ecti n (Muegge, 1956). Milita y pe s nnel have als been ep ted t d ink wate c ntaining up t 32 mg chl ine/L seve al months with n ill e ects (Aust alian NHMRC, 2 4). Muegge (1956) als n ted that a my pe s nnel d inking wate c ntaining chl ine at c ncent ati ns g eate than 9 mg/L expe ienced momenta y c nst icti n the th at and i itati n the mouth and th at (U.S. EPA, 1994c). The t xicity chl ine at levels n mally und in d inking wate appea s t be elatively l w (C t uv and Regelski, 1989; WHO, 1995), and humans appea t t le ate highly chl inated well 0 wate (Muegge, 1956).

In a clinical study, physical and bi chemical pa amete s we e measu ed in 1 healthy male v luntee s a te they d ank inc easing c ncent ati ns chl ine in wate , anging m .1 t 24. mg/L, 18 days. N ot eatment- elated t xicity health e ects we e bse ved (Lubbe s et al., 1982). A subsequent study in 6 men demonst ated min but statistically signi icant changes in selected bl d and bi chemical pa amete s; h weve , wing t the sh t du ati n the study and ising d se t le ance, the changes we e n t necessa ily clinical imp tance (Lubbe s and Bianchine, 1984).

In a study by Wones et al. (1993), test g ups men and w omen c nsumed 1.5 L chl inated (2 mg/L) wate (p 8.) daily 4 weeks. Small dec eases in t ii d thy nine (T3) and thy xine (T4) in the chl inated wate g up we e b de line signi icance in men nly, but we e judged n t meaning ul by the auth s, as levels thy id-stimulating h mone did n t change. Ove all, the auth s c ncluded that the e was n signi icant impact n lipid thy id metab lism in healthy adults ingesting 2 mg chl ine/L in d inking wate (Wones et al., 1993).

igh c ncent ati ns chl ine, such as undiluted s dium hyp chl ite bleach, have been ep ted t be seve ely de mally i itating in humans (Nix n et al., 1975), alth ugh this has been st ngly linked t the high alkalinity the s luti n (stynek et al., 199) athe than the p esence chl ine itsel . abets et al. (1986) ep ted a ew cases de matitis in the m d y, ed, itchy, c acked skin ll wing exp su e t h useh ld bleaching agents c ntaining s dium hyp chl ite. Alth ugh ep ts alle gic c ntact hype sensitivity t hyp chl ite exist (e.g., Eun et al., 1984; Salphale and Shen i, 2 3), these sensitive individuals a e ten p edisp sed t alle gies (stynek et al., 1989; Raci ppi et al., 1994; Salphale and Shen i, 2 3; CCOHS, 2 4b, 2 4c). Despite the equent use s dium hyp chl ite in bleaching agents, de mal sensitizati n t hyp chl ite salts is expected t be quite a e even negligible (St tts, 198 ; abets et al., 1986; Raci ppi et al., 1994).

The genera a i n is n ex ec ed s n ane s y deve derma a ergies he w eve s f ch rine bserved in drinking wa er. N o b ished re r s were f nd regarding derma r c ar irri a i n f wing ba hing r sh wering wi h ch rina ed a wa er. F r hermore, n adverse derma effec s fr m ex s re ch rine in swimming s have been re r ed; f r swimming s, minimum free ch rine c ncen ra i ns be ween 1 and mg/L (PMRA, 1999; Q ueens and Hea h, 2004; U.S. CDC, 2005; WHO, 2006) and a ch rine c ncen ra i ns 10 mg/L (Ci y f Sydney, 1996) are rec mmended. In addi i n, n inf rma i n is avai ab e n any en ia sys emic xici y ha can be ca sed by ex s re ch rine via he derma r e (UNEP, 200).

Adverse hea h effec s fr m inha a i n f high c ncen ra i ns f ch rine gas, inc ding br nch s asm (wheezing, hr a irri a i n, and hy xia), have been we d c men ed (IPCS, 1982). Af er mi d ex s res, c inica symp ms s a y res ve wi hin 6 h rs (Deschamps e a ., 1994; Sex n and Pr nchik, 1998; UNEP, 200). As wi h derma hy ersensi ivi y, adverse reac i ns f wing inha a i n r inges i n f sma amo n s f ch rine end cc r in h se wi h ch rine a ergies r as hma (Penny, 198 ; P s, 1996). There is n h man r ab ra ry anima inf rma i n avai ab e regarding hea h effec s fr m sh r - erm r ng- erm inha a i n ex s re mis s fr m s di m hy ch ri e s i ns (CCOHS, 2004c). H wever, nder n rma c ndi i ns f se, he inha a i n f mis s r va rs fr m b each s i ns r her di e ch rine s i ns is n ex ec ed be significan r res in any hea h effec s in he genera a i n (Raci i e a ., 1994; CCOHS, 2004c).

E idemi gica s dies have n ed an ass cia i n be ween he se f ch rine as a drinking wa er disinfec an and ng- erm hea h effec s, inc ding increased risks f r cancer and her hea h effec s (AwwaRF, 1991; IPCS, 2000; Arb ck e e a ., 2002). H wever, hese s dies have examined n y he br ad ex s re ch rina ed wa er, and genera y inks have been made be ween hea h effec s and ex s re CDBPs ra her han ex s re free ch rine resid a s. The hea h effec s f CDBPs are f en indirec y inked ch rine, since ch rine reac s wi h rganic rec rs rs f rm CDBPs. 3 H wever, his d es n indica e ha here are any hea h effec s direc y ca sed by free ch rine. There have n been any e idemi gica s dies ha have s ecifica y examined free ch rine c ncen ra i ns in wa er and ng- erm hea h effec s in he h man a i n (CCOHS, 2004c).

9.2 Effects on laboratory animals and *in vitro* test systems

9.2.1 Acute toxicity

The ra LD_{50} f r ch rine, in he f rm f ca ci m hy ch ri e, is 850 mg/kg bw in ra s (NIOSH, 1984; WHO, 200) and 880 mg/kg bw in mice (U.S. EPA, 1994a).

Fas ed S rag e-Daw ley ra s were adminis ered 4 mL f s di m hy ch ri e a ei her 200 r 1000 mg/L by gavage (Sc y e a ., 1985). Ana ysis f s mach f ids f rmed wi hin 10 min es sh wed ha deriva ives f ch ramines were r d ced in he s mach when ch rina ed wa er was adminis ered in c nj nc i n wi h amine c mp nds. In addi i n, very w eve s f ch rina ed ni r gen c mp nds were f nd in b d asma as s n as 0 min es a er. In a s bseq en s dy, Sc y e a . (1986) rea ed ra s mach f id *in vitro* wi h 100, 200, 400, 600, 800, 1000, r 1200 mg ch rine/L. A c ncen ra i ns be ween 200 and 1000 mg/L, rganic N-ch ramines were iden ified. H wever, he a h rs ca i ned ha he high d sage eve s adminis ered c d be verwhe ming exis ing mechanisms ha w o d red ce r deac iva e ch rine r ch ramines a wer c ncen ra i ns in he s mach *in vivo*.

Ma e S rag e-Daw ley ra s were adminis ered -mL sing e d ses f hy ch r s acid a 0, 10, 20, r 40 mg/L (a r xima e y 0, 0.19, 0. 8, r 0.75 mg/kg bw) by gavage, and b d samp es were b ained a 15, 0, 60, and 120 min es f wing adminis ra i n. B d g a hi ne (GSH) meas remen s decreased 0 min es af er adminis ra i n in he 10 and 40 mg/L d se gr s; a 60 min es, maximum decreases in b d GSH cc rred in a rea men gr s, b b d GSH was wi hin he c n r range af er 2 h rs. Simi ar y, b d haemo ysis (smo ic fragi i y) increased significan y in a gr s wi hin 15 min es, b re rned n rma wi hin an h r (Abde -Rahman e a ., 1984).

N oh rmona effec s were bserved in S rag e-Daw ley ra s in ba ed wi h s di m hy ch ri e, c n aining he eq iva en f 1% (10 000 mg/L) free ch rine, af er 1 week f bserva i n (V g e a ., 1982). Reversib e effec s n he iver were bserved in ad ma e S rag e-Daw ley ra s given 5 mL f s di m hy ch ri e s i n c n aining 10 000 mg free ch rine/L (Chang e a ., 1981). An increase f ng-chain y nsa ra ed fa y acids was bserved in he iver; af er 10 days, here were n bvi s differences be ween iver riacy g ycer eve s in rea ed gr s and h se f c n r s.

9.2.2 Short-term exposure

Gr s f six fema e C57BL/6 mice were adminis ered s di m hy ch ri e in drinking wa er a eve s f 7.5, 15, r 0 mg/L (a r xima e y 1.5, 2.9, r 5.7 mg/kg bw/day) f r 2 weeks (French e a ., 1998). N oc nsis en differences were bserved in mesen eric ymph n de, ymph cy e r ifera i n, s een r hymus weigh , an ib dy i res, r n mber f an ib dyf rming ce s be ween rea ed mice and c n r mice. S me inc nsis en increases in base ine s een ymph cy e r ifera i n and T ce mi gen res nse were bserved, b hese did n a ear be d se re a ed. The s dy was re ea ed wi h an ex ended d ra i n f 6 weeks, and he ex erimen a c me was iden ica . The a h rs c nc ded ha nei her C⁻ n r ch rina ed by r d c s f rmed in he g adverse y affec immune f nc i n and ha he immune sys em d es n a ear be a sensi ive arge f r C⁻ xici y.

C ndie and Bercz (1985) demons ra ed ha monkeys rea ed wi h 125 mg/L (10 mg/kg bw/day) ch rina ed drinking wa er f r 6 weeks did n ex erience changes in ser m T4 eve s.

In an imm n xici y s dy, ma e S rag e-Daw ley ra s were ex sed s di m hy ch ri e in drinking wa er a c ncen ra i ns f 5, 15, r 0 mg/L fr m weaning 12 weeks f age. S a is ica y significan changes in s een weigh and de ayed- y e hy ersensi ivi y reac i ns were bserved in ra s a he highes d se. Ra s in he mid- and high-d se gr s demons ra ed de ayed macr hage xida ive me ab ism, as we as e eva ed r s ag andin E r d c i n. I was hy hesized ha direc cy xici y immune sys em ce s (via xida ive s ress- y e mechanisms) migh be

responsib e or i ne e ects. The a thors conc ded that mo e ma crophage nction co d be a ected by s bchronic expos re to ch orine-based disin ectants, b t on y at re ative y high doses, and that ch orine-based disin ectants were genera y not partic ar y strong i nodepressants (Exon et a ., 19 7).

Drinking water with ch orine concentrations o 0, 25, 100, 175, and 250 g /L at pH 9.4 was ad nistered to Cr :CDBR Sprag e-Daw ey rats (10 per sex per gro p) or 90 days (Danie et a ., 1990). Statistica y signi icant decreased water cons mp tion was observed in a dose-dependent ma nner, ike y d e to taste aversion. A tho gh mo changes were observed in organ weight and hi to ogica and c inica para ters, these appeared to be sporadic. No treat nt-re ated e ects were seen pon gross and mi croscopic observation. The a thors estab ished a no-observed-adverse-e ect 8 eve (NOAEL) o 250 g /L (24.9 g /kg bw per day o mo es, 16.7 g /kg bw per day o ra es), the highest dose tested in this st dy.

Ch orine was ad nistered via drinking water to B6C3F1 i ce or 90 days, at concentrations o 12.5, 25, 50, 100, and 200 g /L (Danie et a ., 1991). One ma e in the highest dose gro p died towards the end o the st dy, necropsy revea ing gi d congestion o the ng and bronch s, which co d not be identi ied as treat nt-re ated. Decreased water cons mp tion was observed in both sexes, with statistica signi icance in ma es at the two highest doses. So e changes were observed in organ weights and ser enzy es, b t the a thors attrib ted observed changes to decreased water and n trient cons mp tion and a tered e ectro yte ba ance, since there were no overt c inica signs o toxicity and no detectab e treat nt-re ated histopatho ogies. No gross or mi croscopic esions were observed. It was conc ded that drinking water disin ectants s ch as ch orine ind ce mi d, non-speci ic toxicity via indirect mecha nisms -- or exa e, thro gh n tritiona de iciencies--rather than by direct toxico ogica e ects on speci ic organs or tiss es. A NOAEL o 50 g /L (10-12 g /kg bw per day) was estab ished, based on red ction in heart weight in ma es and other decreased organ weights in ma es at 100 g /L (Danie et a ., 1991).

In a pre i inary s bchronic toxicity st dy (Hasegawa et a ., 19 6), 120 F344 rats (10 per sex per gro p) were exposed to sodi hypoch orite concentrations o 0, 250, 500, 1000, 2000, and 4000 g /L in disti ed water or 13 weeks. Body weight gain was decreased in a gro ps, b t the decrease was statistica y signi icant on y in the two highest dose gro ps in ma es and the highest dose gro p in ma es. Sc e o the high-dose rats were ema ciated. No decrease in water cons mp tion or aversion to the taste o water was reported by the a thors. Abso te ng, iver, and sp een weights in ma es and sa ivary g and, ngs, heart, and brain weights o ma es were signi icant y ower than contro s. No histo ogica changes were evident; biochemi ca exa ination showed signs o s ight cha ge to the iver in the two highest dose gro ps o both sexes. A ong-ter o ow- p experi nt did not indicate any notab e di erences in previo s res ts obtained. The a thors conc ded that despite the ack o c ear gross or histopatho ogica changes in organs or s rviva rates, a sodi hypoch orite dose greater than 1000 g /L was s ggestive o s bchronic toxicity to the rat, based on decreases in body weight.

In a s bchronic toxicity test, F344 rats ingested ch orine at concentrations o 500, 1000, 2000, or 4000 g /L in drinking water or 92 days. A trend o decreased water cons mp tion with increasing ch orine dose was observed in both ma es and fema es, and body weight in ma es at the highest dose was decreased co p ared with contro s. A ew organ weights were decreased at the highest dose, inc ding thy s and ng in both ma es and fema es, iver and sp een in ma es, and heart, brain, and sa ivary g ands in fema es. The a thors noted that no mea rkab e patho ogica changes were observed (F r kawa et a ., 19 0).

Ch orinated drinking water was ad nistered to 210 a e CR1:CD1 i ce or 120 days at concentrations p to 30 g /L. A ter 4 ho rs, i ce previo s y i nized with sheep erythrocytes and receiving the 30 g /L water treat nt showed a s ight, b t not statistica y signi icant, increase in oot pad thickness (a test or de ayed-type hypersensitivity) co p ared with other i nized treat nt gro ps. No other di erences in i ne response were observed, and the a thors conc ded that hyperch orinated drinking water did not appear to signi icant y a ect the *in vivo* i ne nction o i ce (Hena nn et a ., 19 2).

Revis et a . (19 6) exposed white rabbits or 3 o nths to ch orine in drinking water at a concentration o 15 g /L (pH 6.5 or .5) and ad nistered in conj nction with a diet o 300 µg iodide/kg (s icient) and 950 µg iodide/kg (high) in a red ced-ca ci diet (previo s st dies had shown dietary ca ci to enhance the e ect o drinking water disin ectants on para cho estero , T3, and T4). No statistica y signi icant changes in ree or bo nd para iodide *in vivo* were observed, and ch orine did not appear to signi icant y a ter para iodide eve s at ow iodide treat nt eve s (Revis et a ., 19 6).

The se a thors cond cted a s bseq ent st dy in New Zea and White rabbits exposed to 0, 0.5, 2, 6, or 15 g ch orine/L in drinking water or 9 mo nths, a ong with both a mo a and a ca ci - de icient diet. Liver cho estero eve s were increased in the two highest dose gro ps, and iver trig ycerides were signi icant y increased in the highest dose gro p. Under mi croscopic exa ination, there was an increased appearance o ipid drop ets in the hepatocytes o treated rabbits. The presence o ca ci did not signi icant y a ter the e ect o ch orine on ipid me tabo ism. The a thors s ggested that ch orine a ects the excretion o cho estero ro the iver, a tho gh the mecha nism is not known (Revis et a ., 1990).

The se a thors cond cted another si ar st dy in rabbits given 0, 0.5, 2, 6, or 15 g ch orine/L in drinking water or 9 mo nths and o nd no statistica y signi icant e ects on para cho estero or T4 eve s. When e ects were observed, they did not appear to be dose dependent. The a thors s ggested that there is no ir evidence o a ca sative ink between ch orinated drinking water and e evated para cho estero or T4 eve s (Ho dsworth et a ., 1990).

9.2.3 Dermal effects

Five ma e ICR i ce (CD-1 strain) had hair mo ved and abdo e ns sprayed with 0.2 mL o 0.525% (5250 g /L) sodi hypoch orite b each eight 8 mi e s per day or 2 consec tive days. A ter treat nt, skin had a dry appearance, with scattered brown cr sty patches. Tiss e changes were mo derate, with a ew areas showing mo re severe changes. The a thors post ated that the high pH ma y have been responsib e or mo e o the

observed e e s ess e al., 1991).

Robinson e al. 1986) rea ed Sen ar mi e wi h a single solu ion o 1 mg/L hypo hlorous a id p 6.5) and sodium hypo hlori e p 8.5) by whole-body exposure and examined hem on days 1, 2, 3, 4, 5, 8, 1 , and 12 ollowing rea men . The maximum response was observed on he 8 h day a er hypo hlorous a id rea men bu waned as ime passed, al hough i was s ill above baseline on he las day o examina ion. Trea men wi h sodium hypo hlori e OCl! ion) also aused in reases in skin hi kness, bu he maximum in rease o 18.2 µm o urred on day 1 . Resul s demons ra e ha brie exposures o high on en ra ions o hypo hlorous a id and sodium hypo hlori e resul in dermal hyperplas i responses in mi e, al hough he in rease in hi kness o he epidermal layer or OCl! was only 4 % o ha o hypo hlorous a id, hus indi a ing ha hypo hlorous a id is a more po en hyperplasiogeni ompound.

Robinson e al. 1986) also rea ed emale Sen ar mi e dermally wi h solu ions o 1, 1 , 1 , 3 , or 1 mg hypo hlorous a id/L p 6.5) by whole-body exposure or a 1 -minu e period or 4 days. In addi ion, OCl! was es ed a 1 mg/L p 8.5). Skin hi kness was assessed he day a er he las rea men . Morphologi al hanges observed were hi kened epidermis and elonga ed basal ells. The maximum epidermal hi kness o 38.7 µm was observed ollowing 1 mg hypo hlorous a id/L rea men , ompared wi h 13.8 µm in on rols. In reased doses were linked o in reased hi knesses, al hough he wo low-dose rea men groups o 1 and 1 mg/L were similar o on rols. owever, or 1 mg OCl!/L rea men , epidermal hi kness in reased o only 25 µm, even hough orresponding ell oun s rom o her rea men s indi a ed ha he hi kness should be grea er. ypo hlorous a id did appear o be hyperplasiogeni ; however, repea ed high-dose appli a ions did no appear o be par i ularly e e ive a main aining he maximum response, whi h may indi a e ei her an adap ive response hrough ine e ive pene ra ion o he hyperplas i skin or oxi i y o he sur a e layer o ells, ausing more rapid loss wi h subsequen rea men s.

9.2.4 Long-term exposure and carcinogenicity

Male Sprague-Dawley ra s were adminis ered hypo hlorous a id a , 1, 1 , or 1 mg/L daily in drinking wa er or 1 year, wi h blood GS and osmo i ragili y measuremen s aken a 2, 3, 4, 6, 1 , and 12 mon hs Abdel-Rahman e al., 1984). A er 1 and 12 mon hs o rea men , GS de reased in higher-dose groups ompared wi h on rols, whi h was hough o be due o possible impa s on he oxida ion-redu ion y le by he GS -dependen sys em. In erim examina ion a 3 mon hs revealed s a is i ally signi i an de reases in red blood ell oun and haema o ri per en age in he high-dose group, bu his was no observed a a la er ime period. De reases in osmos i ragili y values were observed a 6 mon hs o rea men , bu may have been due o lower- han-normal haemolysis o urring in he on rols. No signi i an hloro orm on en ra ions were ound in blood during he 1 year o rea men ; here ore, i does no appear ha T Ms are ormed as a resul o rea ions be ween hlorina ed wa er and organi ma erial in he body. The s udy resul s revealed ha hanges in haema ologi al parame ers were in onsis en and did no indi a e a dose-response pa ern IRIS, 1994).

Groups o 5 male ra s were adminis ered on en ra ions o . 5% and .1% 5 and 1 mg/L) sodium hypo hlori e in drinking wa er, and groups o 5 emale ra s were adminis ered .1% and .2% 1 and 2 mg/L) sodium hypo hlori e in drinking wa er, or 2 years asegawa e al., 1986; Kurokawa e al., 1986). Animals were observed daily; body weigh was measured weekly during he irs 6 weeks and hen every 4 weeks un il he end o he experimen . Ra s o bo h sexes showed a dose-rela ed redu ion in body weigh gain. No de reases in wa er onsump ion were no ed. Rela ive liver weigh s were similar o on rols, bu absolu e liver weigh s were signi i an ly lower in rea ed groups. S a is i ally signi i an de reases in male brain and hear weigh s a he high dose were observed; in emales, de reased salivary gland weigh s a bo h dose levels and in kidneys a he highes dose were observed. Sporadi hanges in blood parame ers o urred bu did no appear o be dose rela ed. No rea men -rela ed in rease in non-neoplas i lesions was observed; in a , non-neoplas i lesions were de reased. Tumours were ound o o ur in a number o organs, bu no s a is i ally signi i an di eren e be ween rea ed groups and on rols was observed or any ype o umour.

Mos o he umours ound were repor ed o be hose ha o ur as ommon spon aneous umours in F344 ra s. The au hors on luded ha sodium hypo hlori e was no a ar inogen in F344 ra s and ha all dose levels had no ar inogeni e e . owever, body weigh redu ion was sugges ive o hroni oxi i y, wi h emales a he highes dose weighing up o 2 % less han on rols a er 2 years o rea men asegawa e al., 1986).

Groups o 5 male and 5 emale mi e were adminis ered sodium hypo hlori e a , 5 , or 1 mg/L in drinking wa er or 2 years Kurokawa e al., 1986). Survival ra es did no di er be ween he rea ed and on rol groups. Dose-rela ed redu ions in body weigh gain o urred and were par i ularly eviden in he high-dose group. Tumours were observed in a number o organs, in luding hyperplas i nodules and hepa o ellular ar inomas in livers o all males. owever, similar o ra s, none o hese were s a is i ally signi i an or appeared o be dose rela ed. The au hors on luded ha sodium hypo hlori e was no a ar inogen in mi e.

Chlorina ed wa er a on en ra ions o , 7 , 14 , and 275 mg/L was given o F344/N ra s or 2 years NTP, 1992). Doses were equivalen o , 4.2, 7.3, and 13.6 mg/kg bw per day or male ra s and , 4.2, 7.8, and 14.4 mg/kg bw per day or emale ra s IRIS, 1994). Groups o 1 ra s were sa ri i ed a weeks 14, 66, and 1 4 o he s udy. There was eviden e o a doserela ed de rease in wa er onsump ion in bo h sexes, bu no di eren es in survival ra es were observed. Mean body weigh s were be ween 5% and 8% less han in on rols hroughou he s udy U.S. EPA, 1994), bu here were no biologi ally signi i an di eren es in organ weigh s or organ o body weigh ra ios. No haema ologi al hanges or lini al indings a ribu able o rea men were observed. The in iden e o mononu lear ell MNC) leukaemia in mid-dose, bu no high-dose, emales was signi i an ly grea er han in on rols, a ording o he li e able es p = . 14). owever, he in iden e o MNC leukaemia in he on rol group 16%) was less han ha in his ori al on rols 25%), sugges ing ha he marginal in rease observed may have been spurious and no due o rea men . Fur hermore, here was no lear dose-response rela ionship or redu ed la en y eviden in hose re eiving lower doses o hlorina ed wa er. O her neoplas i lesions were 0

observed e d ey, pa creas, oral cav y, a d splee , bu ese were o co s dered o be rela ed o e co sump o of c lor a ed wa er. No gross or microscop c les o s were a r bu able o c lor a ed wa er co sump o . Overall, e au ors repor ed a ev de ce was wea suppor of a assoc a o be wee MNC leu aemia female ra s a d co sump o of c lor a ed wa er.

C lor a ed wa er was also g ve o B6C F1 mice for 2 years a co ce ra o s of 0, 70, 140, a d 275 mg/L (NTP, 1992). Doses were equ vale o 0, 8, 15, a d 24 mg/ g bw per day for male mice a d 0, 1, 1 , a d 22 mg/ g bw per day for female mice (WHO, 200). Groups of 10 mice were sacr f ced a wee s 15, 66, a d 104 of e s udy. Surv val ra es amo g rea ed mice were o d ffere from co rols, bu wa er co sump o was decreased bo sexes. Mea body we g s were w i 10% of co rols roug ou e s udy; owever, a e er m evalua o of 66 wee s, body we g s of g - dose male mice were s a s cally s g f ca ly lower a co rols. T ere were o b olog cally s g f ca d ffere ces orga we g s or orga o body we g ra os be wee rea ed a d co rol groups; lower bra a d l ver we g s were oug o be due o e lower body we g s observed. No al era o s aema ology or cl cal c emis ry were observed, a d o gross or microscop c les o s were a r bu able o c lor a ed wa er.

Groups of 50 male a d 50 female Sprague-Dawley ra s were exposed o sod um ypoc lor e co ce ra o s of 0, 100, 500, a d 750 mg/L dr g wa er (Soffr e al., 1997). A mals were allowed o l ve ou e e re l fespa , w i e dea of e las a mal a 151 wee s. Mea da ly wa er co sump o was decreased rea ed a mals a dose-rela ed fas o . Body we g s were sl g ly decreased ose rea ed w i e g es dose of c lor e; s was more ev de males. S a s cal s g f ca ce was o g ve s s udy. No o eoplas c c a ges were observed by gross spec o or s olog cal exami a o . Tumour developme was o dose-rela ed, a d ere was a grea er a expec ed crease umours a e lowes dose. I creased c de ces of lymp omas a d leu aemias were observed female ra s; owever, ese were o dose rela ed, a d e c de ce of leu aemias was u usually low e co rol groups. Males a e g es a d lowes doses ad creased c de ces of rela vely rare s omac umours (squamous cell carc oma of e fores omac a d le omyosarcomas); e lowes -dose females, ree rare lu g ade omas were observed (Soffr e al., 1997). Al oug umour c de ce was o dose rela ed, e au ors sugges ed fur er researc exami g e o coge c r s s rela ed o e c lor a o of dr g wa er.

Dermal appl ca o of 10% (100 000 mg/L) sod um ypoc lor e comb a o w i 4- roqu ol e 1-ox de o ddN female mice was performed. Appl ca o of ypoc lor e self, 60 appl ca o s over 00 days, d d o duce s umours. However, s umours were duced 9/ 2 (4 mal g a , 5 be g) mice follow i g 45 appl ca o s of sod um ypoc lor e g ve af er subma fes a o al doses of 4- roqu ol e 1-ox de, a d o e lymp a c leu aemia was observed. T e au ors o ed, owever, a seemed u l ely a sod um ypoc lor e co s u es a prac cal carc oge c azard (Haya su e al., 1971).

Kuro awa e al. (1984) exami ed bo promo er a d comple e carc oge c proper es of sod um ypoc lor e female Se car mice. I e promo o s ud es, e dorsal s of 20 mice was s aved pr or o s gle op cal appl ca o of ace o e (co rol) or 20 mol d me ylbe za race e (a or) 0.2 mL ace o e. O ne wee la er, 0.2 mL of a solu o of 1% (10 000 mg/L) ypoc lor e solu o d ssolved ace o e was appl ed; e appl ca o was repea ed a e ra e of w ice per wee for 1 year. T e umber a d d ame er of all s umours were recorded wee ly, a d body we g was recorded mo ly. T e c de ce of s umours was o s a s cally d ffere from co rols. I e comple e carc oge c y s es s, sod um ypoc lor e was op cally appl ed alo e (d ssolved ace o e) for 51 wee s (Kuro awa e al., 1984). No e of e mice developed umours w i e 1-year per od, a d o ep dermal yperplas a was see . Overall, sod um ypoc lor e d d o appear o be e er a promo er or a comple e carc oge .

9.2.5 Mutagenicity/genotoxicity

I a *in vitro* Ames assay, sod um ypoc lor e was fou d o be marg ally pos ve for reverse mu a o . T e umber of mu a s ob a ed was var able a d was o dose rela ed. I a repea ed es , sod um ypoc lor e was mu age c for *S. typhimurium* s ra TA 15 0, bu o for s ra TA 15 8 (Wlod ows a d Rose ra z, 1975). Al er a vely, a b oassay exami g b o of a *E. coli* s ra def c e polymerase I, was observed a sod um ypoc lor e co s s e ly b ed pol A_1^- s ra (Rose ra z, 197 ; Rose ra z e al., 1976) bu d d o duce mu a o e Ames assay (Rose ra z e al., 1976).

A me abol c ac va o sys em w i ra l ver microsome frac o plus cofac ors (S9 mix) was appl ed o *in vitro* c romosomal aberra o es s. Sod um ypoc lor e es ed pos ve (10-19.9%) for c romosomal aberra o es s o ac va o w i S9 mix (Ma suo a e al., 1979). Bo calc um ypoc lor e a d sod um ypoc lor e es ed pos ve a o er Ames es , as well as c romosomal aberra o es s C ese ams er cells (Is da e e al., 1984). Calc um ypoc lor e demo s ra ed a g freque cy of cells w i exc a ge- ype aberra o s (per u dose, mg/mL), w hc ge erally s ow carc oge c po e al a mals.

A micro ucleus es s x ddY mice was ega ve for bo sod um ypoc lor e a d calc um ypoc lor e (Hayas e al., 1988). I a o er micro ucleus es , ere were o b olog cally s g f ca d ffere ces observed, a d o s g f ca d ffere ces c romosomal aberra o s were observed for c lor e a pH 6.5 or 8.5 (Me er e al., 1985).

T e SOS C romo es , a *in vitro* assay s ow i g pr mary damage o *E. coli*, was ega ve for sod um ypoc lor e. T e Ames fluc ua o es , a *in vitro* es de ec g po mu a o s o S. *typhimurium*, was also ega ve for sod um ypoc lor e (Le Cur eux e al., 199). T e au ors co cluded a o de ec able mu a o was duced by sod um ypoc lor e follow i g ese wo es s. A ew tmicro ucleus es , for *in vivo* clas oge c effec s o per p eral blood ery rocy es of amp b a ew tlarvae, was pos ve for sod um ypoc lor e (Gau er e al., 1989). However, e au ors d d o rule ou e poss b l y a c lor a ed compou ds mig be par ally respo s ble for e observed effec s (Gau er e al., 1989).

A es for u sc eduled DNA sy es s was performed, us g cul ured mammal a cells o assess *in vitro* ge o ox c y of 12.6% sod um ypoc lor e solu o Syr a ams er embryo cells. Sod um ypoc lor e d d o appear o duce u sc eduled DNA sy es s (Hamaguc a d Tsu su , 2000).

Mutageni it te t a e often p elimina eening te t , and po itive e ult do not alwa o elate with long-te m *in vivo* toxi it (I hidate et al , 1984) Gene all , the above te t indi ate that neithe hlo ine no odium h po hlo ite i on ide ed to be genotoxi

9.2.6 Reproductive and developmental toxicity

Seve al tudie have demon t ated that hlo ine ha few effe t on ep odu tive o developmental health in odent D inking wate ontaining f ee hlo ine on ent ation of 100 mg/L wa given dail ove the enti e lifetime of 236 BD II at , in even on e utive gene ation Chlo ine wa epo ted to be well tole ated, and the e we e no adve e effe t on fe tilit , life pan, g owth patte n, haematolog , o hi tolog of the live , pleen, kidne , o othe o gan The in iden e of malignant tumou wa identi al in expe imental and ont ol g oup of at (D u ke , 1968)

Two t ain of mi e, C3H/HeJ and C57BL/6J, we e u ed to te t the ep odu tive effe t of a id hlo ine-t eated wate fo 6 month T eated wate ontained 10-13 mg e idual hlo ine/L a idified to a pH of 2 5, whe ea tap wate (hlo ine e idual un pe ified) at a pH of 9 6 wa u ed a a ont ol One hund ed and ixt -eight pai ing of C3H/HeJ mi e (1 male and 1 female) o u ed, along with 168 mating of C57BL/6J mi e (45 pai ing of 1 male and 1 female, and 123 t io mating of 1 male with 2 female) In the C3H/HeJ t ain, the total numbe of mi e bo n, total numbe weaned, and both the numbe bo n pe dam and the numbe weaned pe dam we e tati ti all ignifi antl g eate in the t eated wate g oup than in the ont ol g oup In the C57BL/6J t ain, the pe entage weaned in the ont ol g oup wa lightl g eate than that in the t eatment g oup if onl pai ed mating we e on ide ed; howeve , if the t pe of mating wa di ega ded, ep odu tive pe fo man e in the t eated g oup ex eeded that in the ont ol g oup in eve pa amete The numbe of C3H/HeJ mi e weaned wa 5 7% g eate in the t eated g oup than in the ont ol g oup; the numbe of C57BL/6J mi e weaned wa 17 5% g eate in the t eated g oup than in the ont ol g oup The autho noted, the efo e, that hlo ine o h d o hlo i a id t eatment of wate wa not det imental to ep odu tive out ome in mi e (Le , 1968)

In a tud b Che noff et al (1979), app oximatel 500 CD-1 mi e we e allowed to on ume di tilled (ont ol) o muni ipal (t eated) wate fo a minimum of 2 week du ing a limatization and then we e b ed at va iou inte val ove a pe iod of 8 month Level of hlo ine in the t eated wate we e not pe ified Anal i of fetal pa amete indi ated no ignifi ant t eatment- elated effe t in numbe of implant , mo talit , weight, o deg ee of o ifi ation Howeve , an in ea ed in iden e of fetal upe nume a ib in the tap wate g oup wa ob e ved ($p < 0.005$) The autho noted that on ide able va iation in the pa amete tudied in both ont ol and tap wate g oup indi ated that flu tuation we e andom and not elated to wate qualit Vi e al anal i howed no ignifi ant diffe en e in eithe the t pe o o u en e of te atologi al effe t No wate - elated diffe en e in fetal mo talit o o u en e of malfo mation we e ob e ved (Che noff et al , 1979)

Female Sp ague-Dawle at , ix pe g oup, we e admini te ed 0, 1, 10, o 100 mg h po hlo ou a id/L in d inking wate fo 2 5 month p io to in emination (da 0) T eatment ontinued du ing ge tation until da 20, when at we e a ifi ed and live and dead fetu e a well a e o ption we e noted Individual fetal weight we e e o ded, and g o examination fo malfo mation we e made Fetu e we e al o examined fo keletal anomalie and oft-ti ue defe t All fetu e we e viable and no mal in exte nal appea an e, with no g o abno malitie Two ea l e o ption ite we e di ove ed in one female in the high-do e g oup, but thi wa not ignifi antl diffe ent f om ont ol No ignifi ant effe t on fetu weight we e ob e ved Although the high-do e g oup had highe pe entage of both keletal and oft-ti ue defe t , the e we e no tati ti all ignifi ant diffe en e The numbe of anomalie in ont ol and low/mid-do e g oup wa imila , with the low-do e g oup a tuall howing lowe pe entage of total defe t ompa ed with the ont ol The autho on luded that h po hlo ou a id i not emb otoxi o te atogeni , and hlo ine in d inking wate at the on ent ation noted i elativel ha mle to the at when fed to p egnant dams (Abdel-Rahman et al , 1982b)

In a te t fo pe m-head abno malitie in B6C3F1 mi e, a ignifi ant do e- elated in ea e wa ob e ved fo the 100 and 200 mg/L do e (equivalent to 1 6 and 4 0 mg/kg bw pe da) of OCl! (pH 8 5) at a lag pe iod of 3 week following 5 da of gavage do ing (Meie et al , 1985) At the highe t do e of 400 mg/L (8 0 mg/kg bw pe da), the in iden e of pe m-head abno malitie wa ignifi antl in ea ed ompa ed with ont ol , but it wa not fu the in ea ed above that found with the 200 mg/L do e Howeve , h po hlo ou a id (pH 6 5) did not p odu e an pe m-head abno malitie at an do e, no we e the e on i tent po itive e ult fo OCl^- at lag pe iod of 1 and 5 week Effe t on ep odu tive out ome and geneti damage we e not a e ed in thi tud (CCOHS, 2004)

Long-Evan at we e admini te ed h po hlo ou a id b gavage at 0, 1, 2, o 5 mg/kg bw fo 56 da p io to b eeding and th oughout a 10-da b eeding pe iod fo male ; and fo 14 da p io to b eeding and th oughout b eeding, ge tation, and la tation (until pup we e weaned 21 da following pa tu ition) fo female Following b eeding, male we e given a g o ne op of the whole ep odu tive t a t and we e evaluated fo pe m mo pholog Dams we e ob e ved fo fe tilit , length of ge tation, bod weight gain, mate nal behaviou , and ep odu tive t a t evaluation Litte we e evaluated fo viabilit , litte ize, da of e e opening, bod weight gain, and g o exte nal abno malitie Re ult indi ated no lini al ign of toxi it , haematologi al hange , o bod weight dep e ion, even at the highe t do e up to 76 da of expo u e The ob e ved fe tilit ate and vaginal paten (exual matu ation) we e no mal in female ; in litte , da of e e opening, litte u vival, litte ize, and pup weight we e al o no mal In male at , pe m motilit , p og e ive movement, and abno mal pe m mo pholog we e ompa able a o all g oup No hi topathologi al le ion we e ob e ved in an of the at It wa noted that hlo ine demon t ated no te atogeni effe t (Ca lton et al , 1986)

9.2.7 Mode of action

The pha ma okineti and mode of a tion of f ee hlo ine on the human bod a e not full unde tood Re ea he have po tulated that an ob e ved effe t on biologi al tems e ult not f om f ee hlo ine it elf, but athe f om hlo inated o gani fo med in the bod due to it ea tivit (Mink et al , 1983; Meie et al , 1985; Exon et al , 1987; U S EPA, 1994a, 1994) The e hlo inated ompound appea to fo m in the ga t ointe tinal t a t (Mink et al , 1983); howeve , the me hani m of fo mation ha not been dete mined Although CDBP appea to be fo med in the ga t ointe tinal

tract, it ha o tulat d that th ulk of th m r mai a high r mol cular w ight roduct , which may hav littl toxicological ig ifica c (IPCS, 2). Ba d o th availa l tudi , th toxicity of fr chlori i dri ki g wat r i low, a d ff ct o rv d, if a y, a ar to tra i t a d r v r i l .

10.0 Classification and assessment

Th I t r atio al Ag cy for R arch o Ca c r (IARC, 1991) ha cla ifi d hy ochlorit alt i Grou 3, ot cla ifia l a to carci og icity to huma , du to i ad quat vid c i x rime tal a imal a d o data i huma . Similarly, th U.S. EPA (1994a) ha cla ifi d chlori i Grou D, ot cla ifia l a to huma carci og icity. Ba d o th availa l data, H alth Ca ada cla ifi chlori , i th form of hy ochlorit io or hy ochlorou acid, i Cla IV D, u lik ly to carci og ic to huma (data from id miological tudi ar i ad quat to a carci og icity; th r i o vid c of carci og icity i w ll-d ig d a d ro rly co duct d carci og icity ioa ay i two ci of a imal). Thi cla ificatio i a d o th H alth Ca ada (1994) crit ria for cla ificatio of carci og icity.

Th NTP (1992) tudy co clud d that th r wa quivocal (margi al) vid c of carci og ic activity i f mal rat a d o a i cr a i th i cid c of MNC l uka mia a d that th r wa o vid c of carci og ic activity of chlori at d wat r i mal rat , mal mic , or f mal mic . Although a i cr a d i cid c of MNC l uka mia i f mal rat wa o rv d, th r wa o vid c of a do -r o r latio hi a d o vid c of a t mporal r latio hi tw e i cr a i g do a d i cid c of tumour . I additio , it wa ot d that MNC l uka mia ha a high o ta ou rat of occurr c i f mal F344 rat a d that th l v l r ort d i th NTP tudy w r withi th hi torical co trol ra g of i cid c for th x a d trai of rat (U.S. EPA, 1994a). Th U.S. EPA (1994a) tat d that th i cid c of MNC l uka mia i f mal rat ca ot ol ly attri ut d to x o ur to chlori i dri ki g wat r, ut rath r may r fl ct th high ackgrou d rat of MNC l uka mia i th t t ci . Oth r lo g-t rm tudi hav ot fou d a y adv r ff ct r ulti g from i g tio of chlori i dri ki g wat r (Ha gawa t al., 1986; Kurokawa t al., 1986) a d thu u ort th fi di g of th NTP tudy (IRIS, 1994).

A d cr a i a imal ody w ight, at time accompa i d y a d cr a i c rtai orga w ight , ha o rv d i a umb r of tudi . Author hav ugg t d that thi r o i li k d to d cr a d wat r co umptio y th rod t (Ha gawa t al., 1986; Kurokawa t al., 1986; Da i l t al., 1991; NTP, 1992; Soffritti t al., 1997), mo t lik ly du to av r io to th ta t of high l v l of chlori i dri ki g wat r (Da i l t al., 1991; IRIS, 1994). Da i l t al. (1991) ugg t d that i ad quat wat r co umptio r ult i alt r d l ctrolyt ala c a d utritio al d fici ci , which i tur ca aff ct ody a d orga w ight . Ha gawa t al. (1986) d mo trat d that o c tr atme t with chlori at d dri ki g wat r wa t rmi at d, rod t how d ra id a d r marka l ody w ight gai , thu ugg ti g that a y ff ct o ody w ight ar i d d tra i t a d r v r i l .

I huma , th g ral o ulatio i ot x ct d to x ri c d rmal ff ct or to o ta ou ly d v lo d rmal all rgi to chlori i dri ki g wat r, articularly at th low l v l o rv d. Th r hav o u li h d r ort of d rmal or ocular irritatio i huma followi g athi g or how ri g with chlori at d ta wat r. I additio , o i formatio i availa l o a y ot tial y t mic toxicity that ca cau d y x o ur to chlori via th d rmal rout (UNEP, 2 3).

10.1 Aesthetic considerations

Whil chlori atio ca h l improv ta t a d odour through th r actio with orga ic mat rial a d iro (Co ll, 1996), it ca al o g rat chlori ou flavour cau d y th 0 r c of th di i f cta t it lf or y th occurr c of oth r chlori at d y- roduct form d y th r actio with oth r com ou d i th wat r. A urv y of U.S. a d Ca adia dri ki g wat r la t fou d that chlori wa th domi a t cau of odour com lai t a d a ig ifica t cau of ta t complai t from co umer (Suff t t al., 1996).

WHO (1997) ot d that av r io to th ta t of chlori i dri ki g wat r could l ad huma o ulatio to r j ct a ourc of wat r that i actually af to dri k. Th r ort d thr hold ra g for u acc ta ility vary from a low a . 5- .1 mg/L (AwwaRF, 2 4) to tw e .6 a d 1. mg/L (WHO, 2 4). H c , co umer acc ta ility of ta t a d odour hould a co id ratio i th d liv ry of dri ki g wat r co tai i g fr chlori .

11.0 Rationale

Th u of chlori i th tr atme t of dri ki g wat r ha virtually limi at d wat r or di a , cau chlori ca kill or i activat mo t microorga i ms commo ly fou d i wat r. Th majority of dri ki g wat r tr atme t la t i Ca ada u ome form of chlori to di i f ct dri ki g wat r: to tr at th wat r dir ctly i th tr atme t la t a d/or to mai tai a chlori r idual i th di tri utio y t m to r v t act rial r growth. 0 H alth ri k from chlori or from a y of it di i f ctio y- roduct ar much low r tha th ri k from co umi g wat r that ha ot ad quat ly di i f ct d.

H alth- a d valu hav ta li h d i oth r cou tri or y i t r atio al orga izatio . WHO a d Au tralia hav ta li h d ta dard or guid li valu for chlori of 5 mg/L i dri ki g wat r, wh r a th U.S. EPA ha t a maximum r idual di i f cta t l v l of 4 mg/L for chlori . Th h alth- a d valu hav all d riv d from th ame NTP (1992) tudy. Th ta dard or guid li ar co id r d to co rvativ i c th r w r o docume t d h alth ff ct i that tudy, or a y oth r availa l tudi . A tru NOAEL wa ot id tifi d i th NTP tudy du to a th tic co id ratio limiti g th high t do t t d i rod t .

Ba d o th lack of toxicity o rv d i rod t tudi , th F d ral-Provi cial- T rritorial Committ o Dri ki g Wat r ha d m d that th r i o d to ta li h a guid li for chlori i dri ki g wat r. Th Committ ha al o d t rmi d that a a th tic o j ctiv i ot c ary, i c l v l commo ly fou d i dri ki g wat r ar withi a acc ta l ra g for ta t a d odour a d rot ctio of co umer from micro ial h alth ri k i aramou t. 0

Where chlor e e a a r k g water fecta t, t recomme e that t co ce trat o be etermi e o a y tem- pec f c ba to e re effect ve e of fect o a ma te a ce of a appropr ate re al, wh le mi miz g by-pro ct format o a ae thet c co cer .

12. References

Ab el-Rahma , M.S., Co r , D., a B ll, R.J. (1982a) Metabol m a pharmacok et c of alter ate r k g water fecta t . E v ro . Health Per pect., 46: 19-23.

Ab el-Rahma , M.S., Berar , M.R., a B ll, R.J. (1982b) Effect of chlor e a mo ochlorami e r k g water o the evelop g rat fet . J. Appl. Tox col., 2(3): 156-159.

Ab el-Rahma , M.S., Wal ro , D.M., a B ll, R.J. (1983) A comparat ve k et c t y of mo ochlorami e a hypochloro ac rat. J. Appl. Tox col., 3(4): 175-179.

Ab el-Rahma , M.S., S h, D.H., a B ll, R.J. (1984) Pharmaco y amic a tox c ty of chlor e r k g water the rat. J. Appl. Tox col., 4(2): 82-86.

APHA, AWWA, a WEF (1998) Sta ar metho for the exami at o of water a wa tewater. 20th e t o . Amer ca P bl c Health A oc at o , Amer ca Water Work A oc at o , a Water E v ro me t Fe erat o , Wa h gto , DC.

APHA, AWWA, a WEF (2005) Sta ar metho for the exami at o of water a wa tewater. 21 t e t o . Amer ca P bl c Health A oc at o , Amer ca Water Work A oc at o , a Water E v ro me t Fe erat o , Wa h gto , DC.

Arb ckle, T .E., Hr ey, S.E., K ra er, S.W ., N uckol , J.R ., R char o , S.D., S ger, P., Me ola, P., Do , L., We el, C., A hley, D.L., Froe e, K.L., Pegram, R.A., Sch ltz, I.R., Re f, J., Bacha , A.M., Be o t, F.M., Ly berg, M., Poo le, C., a Waller, K. (2002) A e g expo re ep emiolog c t e to fect o by-pro ct r k g water: report from a ter at o al work hop. E v ro . Health Per pect., 110(S ppl. 1): 53-60.

ASTM (2006) Sta ar te t metho for re al chlor e water. I : A al book of ASTM ta ar . Vol. 11.01. ASTM I ter at o al, W e t Co hohocke , PA (ASTM D1253-03).

ATSDR (2002) ToxFAQ sfor calc m hypochlor te/ o m hypochlor te. Age cy for Tox c S b ta ce a D ea e Reg try, P bl c Health Serv ce, U.S. Departme t of Health a H ma Serv ce , Atla ta, GA ; acce e Febr ary 10, 2006).

ATSDR (2007) ToxFAQ sfor chlor e. Age cy for Tox c S b ta ce a D ea e Reg try, P bl c Health Serv ce, U.S. Departme t of Health a H ma Serv ce , Atla ta, GA; acce e J e 13, 2007).

A tral a NHMRC (2004) A tral a r k g water g el e : Fact heet -- orga c chemical . Nat o al H ealth a Me cal Re earch Co c l, Gover me t of A tral a; acce e September 24, 2007).

AWWA (1999) Water q al ty & treatme t: a ha book of commu ty water ppl e . 5th e t o . P bl he for the Amer ca Water Work A oc at o by McGraw-H ll, New York, NY.

AwwaRF (1991) Health effect of fecta t a fect o by-pro ct . Amer ca Water Work A oc at o Re earch Fo at o , De ver, CO.

AwwaRF (2004) P bl c percept o of tap water chlor o flavor. Amer ca Water Work A oc at o Re earch Fo at o , De ver, CO (Report 1P-5.75C-90980F-3/04-CM).

AwwaRF (2005) Impact of tr b t o y tem water q al ty o fect o eff cacy. Amer ca Water Work A oc at o Re earch Fo at o , De ver, CO (Report 91094).

Babl, F., Khar ch, E.S., a Woolf, A. (1998) A rway e ema follow i g ho ehol bleach ge t o . Am. J. Emerg. Me ., 16: 514-516.

B ll, R.J. (2000) Dr k g water fect o . I : L ppma , M. (e .). E v ro me tal tox ca t : h ma expo re a the r health effect . Wiley-I ter c e ce, New York, NY.

Ca tor, K.P. (1997) Dr k g water a ca cer. Ca cer Ca e Co trol, 8(3): 292-308.

Carlto , B.D., Barlett, P., Ba ara , A., Coll g, K., O s , I., a Smith, M.K. (1986) Repro ct ve effect of alter at ve fecta t . E v ro . Health Per pect., 69: 237-241.

CCOHS (2004a) CHEMINFO Chemical Prof le: Calc m hypochlor te. Ca a a Ce tre for Occ pat o al Health a Safety, Hamilto , O rtar o (www. tox.org/ ataba k/ oc me t /chemical/calhypoc/c e100.htm; acce e Febr ary 10, 2006).

CCOHS (2004b) CHEMINFO Chemical Prof le : Chlor e. Ca a a Ce tre for Occ pat o al Health a Safety, Hamilto , O rtar o; acce e Febr ary 10, 2006).

CCOHS (2004c) CHEM INFO Chemical Prof le: So m hypochlor te ol t o . Ca a a Ce tre for Occ pat o al Health a Safety, Hamilto , O tar o; acce e Febr ary 10, 2006).

CFIA (2004) Meat hyg e e ma al of proce re : Meat hyg e e rect ve . Ca a a Foo I pect o Age cy; acce e Febr ary 10, 2006).

CFIA (2005) Co e of pract ce for mi mally proce e rea y-to-eat vegetable . Ca a a Foo I pect o Age cy; acce e J e 16, 2008).

CFIA (2007) Chapter 5, S bject 1. Fac l ty Compl a ce Req reme t . F h I pect o Reg lat o , Sche le I a II. Fac l t e I pect o Ma al. Ca a a Foo I pect o Age cy; acce e September 24, 2007)

Chang, J , ogt, C R , un, G Y , and un, A Y (1981) Effects of acute and nistration of chlorinated water on liver lipids Lipids, 16(5): 336 340

Chernoff, N , Rogers, E , Carver, B , Kavlock, R , and Gray, E (1979) Fetotoxic potential of nicipal drinking water in the o use Teratology, 19: 165 169

City of ydney (1996) Public swi ng pool and spa pool guidelines Depart nt of ealth New outh Wales; accessed June 28, 2006)

Condie, L W. and B ercz, J P (1985) Target organ effects of disinfectants and their by products In: Jo lley, R L , Bull, R J , Davis, W.P , Katz, , Roberts, M. , and Jacobs, A (eds) Proceedings of the Fifth Conference on Water Chlorination, June 1984 ol ume 5 Lewis Publishers, Chelsea, MI m

Connell, G F (1996) W ater disinfection series: The chlorination/chlorami nation handbook Am erican Water Works Association, Denver, CO .

Cotruvo, J A and Regelski, M. (1989) Issues in developing national pri ry drinking water regulations for disinfection and disinfection by products In: m Calabrese, E J , Gilbert, C E , and Pastides, (eds) afe Drinking Water Act Am nd nts, regulations and standards Lewis Publishers, Chelsea, MI pp 57 69

Curlin, L C , Bo rajou, T , and ansson, C B (1991) Alkali and chlorine products: chlorine In: Kirk Ot mer encyclopedia of chemi cal technology 4th edition ol 1 John Wiley and ons, New York, NY

Cutler, D and Miller, G (2005) The role of public health im p rovements in health advances: the twentieth century United tates Demo graphy, 42(1): 1 2 2

Daniel, F B , Condie, L W., Robinson, M., tober, J A , York, R G , Olson, G R , and Wang, R (1990) Com p arative subchronic toxicity studies of three disinfectants J A Water Works Assoc , 82: 61 69

Daniel, F B , Ringhand, P , Robinson, M., tober, J A , Olson, G R , and Page, N P (1991) Com p arative subchronic toxicity of chlorine and mo nochloramine in the B6C3F1 o use J A Water Works Assoc , 83(11): 68 75

Deschamp s, D , oler, P , Rosenberg, N , Baud, F , and Gervais, P (199 4) Persistent asthma after inhalation of mi xture of sodium hypochlorite and hydrochloric acid Chest, 105: 1895 1896

Druckey, (196 8) [Chlorinated drinking water, toxicity tests involving seven generations of rats] Food Cosmet Toxicol , 6: 147 154 (in Germa n)

Eun, C , Lee, A Y , and Lee, Y (1984) odium hypochlorite dermati tis Contact Dermati tis, 11(1): 45

Exon, J , Koller, L D , O'Reilly, C A , and Bercz, J P (1987) I nortoxicologic evaluation of chlorine based drinking water disinfectants, sodium hypochlorite and mo nochloramine Toxicology, 44(3): 257 269

French, A , Copeland, C B , Andrews, D L , Wilhams , W.C , Riddle, M.M., and Luebke, R W. (1998) Evaluation of the potential i nortoxicity of chlorinated drinking water in mi ce Toxicology, 125: 53 58

Furukawa, F , Kurata, Y , Kokubo, T , Takahashi, M , and Nakadate, M (1980) Oral acute and subchronic toxicity studies for sodium hypochlorite in F344 rat Bull Natl Inst yg ci , 98: 62 69

Gauthier, L , Levi, Y , and Jaylet, A (1989) Evaluation of the clastogenicity of water treated with sodium hypochlorite or mo nochloramine using a m mi cronucleus test in newt larvae (Pleurodeles waltl) Mutagenesis, 4: 170 173

aas, C N (1999) Benefits of using a disinfectant residual J A Water Works Assoc , 91(1): 65 69

abets, J M W., Geursen Reitsma , A M., tolz, E , and van Joost, T (198 6) ensitization to sodium hypochlorite causing hand dermati titis Contact Dermati titis, 15: 140 142

mai guchi, F and Tsutsui, T (2000) Assessme nt of genotoxicity of dental antiseptics: ability of phenol, guaiacol, p phenolsulfonic acid, sodium hypochlorite, p chlorophenol, cresol or formaldehyde to induce unscheduled DNA synthesis in cultured yrian hamster embryo cells Jpn J Pharmacol , 83(3): 273 276

arp, D L (2002) Current technology of chlorine analysis for water and wastewater ach Com p any, Loveland, CO (Technical Informa tion eries, Booklet 17; accessed February 23, 2006)

asegawa, R , Takahashi, M., Kokubo, T , Furukawa, F , Toyoda, K , ato, , Kurokawa, Y , and ayashi, Y (1986) Carcinogenicity study of sodium hypochlorite in F344 rats Food Chem Toxicol , 24(12): 1295 1302

ayashi, M , Kishi, M., ofuni, T , and Ishidate, M. (1988) M icronucleus tests in mi ce on 39 food additives and eight mi scellaneous chemi cals Food Chem Toxicol , 26(6): 487 500

ayatsu, , oshino, , and Kawazoe, Y (1971) Potential co carcinogenicity of sodium hypochlorite Nature (London), 233: 295

ealth Canada (1993) Food and Drug Regulations; accessed February 10, 2006)

ealth Canada (1994) Appendix B Criteria for classification of carcinogenicity In: Canadian Environme ntal Protection Act hu man health risk assessme nt for priority substances Minister of upply and ervices Canada, Ottawa, Ontario (Catalogue No En40 215/41E)

ealth Canada (1996) Guidelines for Canadian drinking water quality: upporting docume nt: Chlorami nes; accessed February 10, 2006)

ealth Canada (2000) Chlorinated disinfection by products Prepared for the Chlorinated Disinfection By product Task Group

Health Ca a a 4a) Gui eli es for Ca a ia ri ki g water quality: Supporti g ocue t: E teric viruses; accesse October 1 , 6).

Health Ca a a 4b) Gui eli es for Ca a ia ri ki g water quality: Supporti g ocue t: Protozoa: Giar ia a Cryptospori iu, accesse October 4, 6).

Health Ca a a 6a) Gui eli es for Ca a ia ri ki g water quality: Gui eli e Tech ical Docue t: Escherichia coli; accesse October 1 , 6).

Health Ca a a 6b) Gui eli es for Ca a ia ri ki g water quality: Gui eli e Tech ical Docue t-- Total colifos ; accesse Dece ber 19, 6).

Health Ca a a 6c) Gui eli es for Ca a ia ri ki g water quality: Gui eli e Tech ical Docume t: Trihalo e tha es; accesse February 1 , 6).

Health Ca a a 8a) Gui eli es for Ca a ia Dri ki g W ater Quality: Gui eli e Tech ical Docu t: Chlo rite a Chlorate. W ater Quality a Health Bureau, Healthy E viro e ts a Co sue r Safety Bra ch, Health Ca a a, Ottawa, O tario.

Health Ca a a 8b) Gui eli es for Ca a ia Dri ki g Water Quality: Gui eli e Tech ical Docue t - Haloacetic aci s. Water Quality a Health Bureau, Healthy E viro e ts a Co sue r Safety Bra ch, Health Ca a a, Ottawa, O tario.

Health Ca a a 8c) Gui a ce for Chloral Hy rate i Dri ki g Water. Water, Air a Cli ate Cha ge Bureau, Healthy E viro e ts a Co sue r Safety Bra ch, Health Ca a a, Ottawa, O tario.

Hea , L.M., White, W.J., a La g, C.M. 198) Prolo ge exposure to aci , chlori e, or tetracycli e i the ri ki g water: effects o elaye -type hyperse sitivity, hea ggluti atio titers, a reticuloe othelial cleara ce rates i i ce. Lab. Ai Sci., 3 : 6 3-6 8.

Hess, J.A., M oli ari, J.A., Gleaso , M.J., a Re ecki, C. 1991) Epi ea l toxicity of isi fecta ts. J. De t., 4: 51-56.

Hol sworth, G., McCauley, P., a Revis, N.W. 199) Lo g-term effects of chlori e-co tai i g isi fecta ts o phos a levels o f cholesterol a thyroxi e i rabbits a pigeo s. I : Jolley, R.L., B ull, R.J., Davis, W .P., Katz, S., Roberts, M.H., a Jacobs, V.A. e s.). Procee i gs of the Sixth Co fere ce o Water Chlori atio , 1987. Vo ue 6. Lewis Publishers, Chelsea, MI.

Hosty ek, J.J., Patrick, E., You ger, B., a Maibach, H.I. 1989) Hypochlorite se sitivity i a . Co tact Dea titis, : 3 -37.

Hosty ek, J.J., Wilhe J K .-P., Cua, A.B ., a M aibach, H.I. 199) Irritatio factors of so iu hypochlorite solutio s i hu a ski . Co tact Dea titis, 3 : 316-3 4.

Howell, J.M. 1991) Alkali ity of o -i ustrial clea i g pro ucts a the likelihoo of pro uci g sig ifica t esophageal bur s. J. E rg. M e ., 9 6): 56 -56 .

IARC 1991) Chlori ate ri ki g water, chlori ate by-pro ucts, sce other haloge ate o p ou s, cobalt a cobalt o p ou s. I ter atio al Age cy for Research o Ca cer, Lyo , Fra ce IARC Mo ographs o the Evaluatio of Carci oge ic Risks to Hu a s, Vo ue 5).

IPCS 198) Chlori e a hy roge chlori e. I ter atio al Progra o Che ical Safety, Worl Health Orga ization , Ge eva, Switzerla E viro e tal Health Criteria 1).

IPCS 1997) So iu hypochlorite. I ter atio al Progra o Che ical Safety, Worl Health Orga ization , Ge eva, Switzerla Poiso s I for a tio Mo ograph 495; accesse February 1 , 6).

IPCS) Disi fecta ts a isi fecta t by-pro ucts. I ter atio al Progra o Che ical Safety, Worl Health Orga ization , Ge eva, Switzerla E viro e tal Health Criteria 16).

IRIS 1994) Chlori e CASRN 778 -5 -5). I tegrate Risk I for a tio Syste ; U.S.E viro e tal Protectio Age cy www.epa.gov/iris/subst/ 4 5.ht accesse February 1 , 6).

Ishi ate, M., Sofu i, T., Yoshikawa, K., Hayashi, M., Nohi , T., Sawa a, M ., a Matsuoka, A. 1984) Pri ary u tage icity scree i g of foo a itives curre tly use i Japa . Foo Che . Toxicol., 8): 6 3-636.

ISO 6) List of ICS fiel s: 13. 6 .5 - Exa ia atio of water for che ical substa ces. I ter atio al Orga ization for Sta ar ization ; accesse February 3, 6).

Kurokawa, Y., Tak u ra, N., Matsushi a , Y., Ha zawa, T., a Hayashi, Y. 1984) Stu ies o the pro o ti g a o p lete carci oge ic activities of sce oxi izi g che icals i ski carci oge esis. Ca cer Lett., 4 3): 99-3 4.

Kurokawa, Y., Takaya a , S., Ko ishi, Y ., Hiasa, Y., Asahi a, S., Takahashi, M ., Maekawa, A., a Hayashi, Y . 1986) Lo g-ter *in vivo* carci oge icity tests of potassiu bro a te, so iu hypochlorite a so iu chlorite co ucte i Japa . E viro . Health Perspect., 69: 1- 35.

LeChevallier, M.W. 1998) Be efits of re p loyi g a isi fecta t resi ual i istrib utio syste s . Water Supply, 16: 61-73.

LeChevallier, M.W., Welch, N.J., a S th, D .B. 1996) Full-scale stu ies of factors relate to colifor regrowth i ri ki g water. Appl. E viro . Microbiol., 6 7): 1- 11.

Le Curieux, F., Marzi , D., a Erb, F. 1993) Co p ariso of three short-ter assays: results o seve che icals. Pote tial co tributio to the co trol of water ge otoxicity. M utat. Res., 319 3): 3- 36.

Les, E.P. 1968) Effect of aci ifie -chlori ate water o repro uctio i C3H/HeJ a C57BL/6J i ce. Lab. Ai Care, 18: 1 - 13.

Lubbers, d Bi chi e, (1984) Effects of the cute risi g dose dmi istr tio of chlori e dio ide, chlor te d chlorite to ormal he lthy dult male volu teers E p P thol T o icol O rcol , 5: 215-228

Lubbers, , Ch u , S , d Bi chi e, (1982) Co trolled cli ic l ev lu tio s of chlori e dio ide, chlorite d chlor te i ma E viro He lth Perspect , 46: 57-62

Matsuok , A , H y shi, M , d Ishid te, M (1979) Chromosomal berr tio tests o 29 chemic ls combi ed with S9 mi *in vitro* Mut t es , 66: 277-290

Meier, , Bull, , Stober, A , d Cimi o, M.C (1985) Ev lu tio of chemic ls used for dri ki g w ater disi fectio for productio of chromosomal d mage d sperm-he d b ormalities i mice E viro Mut ge , 7: 201-211

Mi k, F L , Colema , W.E , Mu ch, W., K ylor, W.H , d i gh d, H P (1983) I vivo formatio of h loge ted re ctio products followi g peror l sodium hypochlorite Bull E viro Co t m. To icol , 30: 394-399

Muegge, O (195 6) Physiologic l effects of he vily chlori ted dri ki g w ater Am. Water Works Assoc , 48: 1507-1509 [cited i U S EPA, 1994c]

MWH (2005) Water tre tme t pri ciples d desig 2 d editio Mo tgomery Watso H rz , revised by Critte de oh Wiley & So s, Hoboke , NJ

NIOSH (1984) egistry of to ic effects of chemic l subst ces N atio l I stitute for Occup tio l S fety d He lth, W shi gto , DC

Ni o , G A , Tyso , C A , d Wertz, W C (1975) I terspecies comp riso s of ski irrit cy To icol Appl Ph rmacol , 31: 481-490

NSF/ANSI (2002) St d rd 42: Dri ki g w ater tre tme t u its--Aesthetic effects NSF I ter tio l d Americ N atio l St d rds I stitute NSF I ter tio l, A Arbor, MI

NTP (1992) To icology d c rci oge esis studies of chlori ted w ater (CAS os: 7782-50-5 d 7681-52-9) d chlor mi ted w ater (CAS o 10599-90-3) i F344/N r ts d B6C3F1 mice (dri ki g w ater studies) N atio l To icology Progr m, N atio l I stitutes of He lth, U S Dep rtme t of He lth d Huma Services, ese rch Tri gle P rk, NC (NTP T 392)

P yme t, P (1999) Poor effic cy of residu l chlori e disi fect t i dri ki g w ater to i ctiv te w aterbor e p thoge s i distributio systems C Microbiol , 45: 709-715 x

Pe y, P T (1983) Swimmi g pool wheezi g B r Med , 287: 461-462

PMRA (1999) Devices for use i swimmi g pools d sp s [brochure] Pest Ma geme t egul tory Age cy, He lth C d , Ott w a, O rt rio (www.pmr - rl gc c /e glish/pdf/p otes/pool_devices-e pdf; ccessed u e 28, 2006)

Po , (2008) Do You e lly H ve Free Chlori e esidu l? Opflow, 34(6): 24-27

Potts, (1996) F ctors ssoci ted with respir tory problems i swimmers Spo rts Med , 21(4): 256-261

Quee sl d He lth (2004) Quee sl d He lth swimmi g d sp pool w ater qu lity d oper tio l guideli es (October 2004) Commu ic ble Dise ses U it, Public He lth Services, Quee sl d He lth, Quee sl d Gover me t; ccessed u e 28, 2006)

cioppi, F , D sk lero s, P A , Besbelli, N , Borges, A , Der emaeker, C , M g li i, S I , Marti ez Arriet , , Pulce, C , uggero e, M.L , d Vl chos, P (1994) Household ble ches b sed o sodium hypochlorite: review of cute to icology d poiso Food Chem. To icol , 32(9): 845-861

evis, N , McC uley, P , d Holdsworth, G (1986) el tio ship of diet ry iodide d dri ki g w ater disi fect ts to thyroid fu ctio i e perime t l imals E viro He lth Perspect , 69: 243-248

evis, N.W , Holdsworth, G , d McC uley, P (1990) Effect of dri ki g w ater co t i i g chlori e d mo ochlor mi e o cholesterol d triglyceride levels i the liver of the pigeo d r bbit I : olley, L , B ull, , D vis, W.P , K tz, S , oberts, M.H , d cobs, V A (eds) Proceedi gs of the Si th Co fere ce o Water Chlori tio , 1987 Volume 6 Lewis Publishers, Chelse , MI

obi so , M., Bull, , Sch mer, M., d Lo g, E (1986) Epidermal hyperpl si i mouse ski followi g tre tme t with lter tive dri ki g w ater disi fect ts E viro He lth Perspect , 69: 293-300

ose kr z, H S (1973) Sodium hypochlorite d sodium perbor te: prefere ti l i hibitors of DNA polymer sedeficie t b cteri M ut t es , 21: 171-174 x

ose kr z, H S , Gutter, B , d Speck, W.T (1976) Mut ge icity d DNA-modifyi g ctivity: comp riso of two microbi l ss ys M ut t es , 41: 61-70

S lph le, P S d She oi, S D (2003) Co t ct se sitivity to c lcium hypochlorite Co t ct Dermatitis, 48 : 162

Scully, F E , Mazi , K E , So e shi e, D E , d D iel, F B (1985) e ctio s of hypochlorite d org ic Nchlor mi es i stomach fluid I : olley, L , Bull, , D vis, W.P , K tz, S , oberts, M.H , d cobs, V A (eds) Proceedi gs of the Fifth Co fere ce o Water Chlori tio , u e 1984 Volume 5 Lewis Publishers, Chelse , MI

Scully, F E , Mazi , K E , So e shi e, D E , d Kopfler, F (1986) Qu tit tio d ide tific tio of org ic Nchlor mi es formed i stomach fluid o i gestio of queous hypochlorite E viro He lth Perspect , 69: 259-265 x

Se to , D d Pro chik, D (1998) Chlori e i h l tio : the big picture To icol Cli To icol , 36(1-2): 87-93

Soffritti, , poggi, F , L nzi, A , and toni, G (1997) R su ts of ong t rm carcinog nicity studi s of ch orin in rats Ann N Y Acad Sci , 837: 189 208

Stotts, J (1980) P anning, conduct and int rpr tation of human pr dictiv s nsitization patch t sts In: Dri , V A and Lazar, P (d s) Curr nt conc pts - in cutan ous toxico ogy Acad mic Pr ss, N w York, N Y

Suff t, I H , Corado, A , Chou, D , Guir , c , and utt rworth, (1996) Tast and odor surv y J Am. Wat r Works Assoc , 88(4): 168 181

Symons, J , rad y, L C , and C v and, T C (ds) (2000) Th drinking wat r dictionary American Wat r Works Association, D nv r, CO .

Truss , R R (1999) Saf guarding distribution syst m int grity J Am. Wat r Works Assoc , 91(1): 46 54

UNEP (2003) Initia ass ssment profi : Ch orin Scr ning Information Datas t for High Vo ume Ch mica s (SIDS), Unit d Nations Environment Programm

U S CDC (1999) T n gr at pub ic h a th achi v ments Unit d Stat s, 1990 1999 U S C nt rs for D is as Contro and Pr v ntion rbid o rta o Wk y R p , 48(12): 241 243

U S CDC (2005) Your disinf ction t am: ch orin & pH Division of Parasitic Dis as s, U S C nt rs for Dis as Contro and Pr v ntion (www.cdc gov/h a thyswimming/ph_ch orin htm; acc ss d Jun 28, 2006)

U S EPA (1994a) Nationa Primary Drinking Wat r R gu ations Disinf ctants and Disinf ction yproducts Fina Ru U S Environmenta Prot ction Ag ncy F d R gist , 59, Ju y 29

U S EPA (1994b) Ch mica summary for ch orin Offic of Po ution Pr v ntion and Toxics, U S Environmenta Prot ction Ag ncy (www. pa gov/ch mfact/s_ch ori txt; acc ss d F bruary 10, 2006)

U S EPA (1994c) Drinking wat r crit ria document for ch orin , hypoch orous acid and hypoch orit ion (draft) Offic of Drinking W at r, U S Environm nta Prot ction Ag ncy, Cincinnati, OH

U S EPA (1999a) R r gistration igibi ity d cision (RED): Ch orin gas Offic of Pr v ntion, P sticid s and Toxic Substanc s, U S Environmenta Prot ction Ag ncy, Washington, DC; acc ss d S pt mb r 24, 2007)

U S EPA (1999b) 2 Disinf ctant us in wat r tr atment In: EPA guidanc manua : A t rnativ disinf ctants and oxidants Offic of Wat r, U S Environm nta Prot ction Ag ncy, Washington, DC Apri (EPA 815 R 99 014; acc ss d rch 16, 2008)

U S EPA (2002) Nationa Primary Drinking Wat r R gu ations 40 CFR Part 141: 436 438

U S EPA (2007) EPA simu tan ous comp ianc guidanc manua for th Long T rm 2 and Stag 2 D P Ru s Offic of Wat r, U S Environmenta Prot ction Ag ncy, Washington, DC rch (EPA 815 R 07 017; acc ss d rch 16, 2008)

Vi anu va, C , F rnand z, F , ats, Na, Grima t, J O , and Kog vinas, (2003) ta ana ysis of studi s on individua consumption of ch orinat d drinking wat r and b add r canc r [corr ct d] J Epid mio Community H a th, 57(3): 166 173

Virto, R , anas, P , A var z, I , Condon, S , and Raso, I (2005) mbran damag and microbia inactivation by ch orin in th abs nc and pr s nc of a ch orin d manding substrat App Environ crobioi 71(9): 5022 5028

Vogt, C R , Kapi a, S , Chang, J S , and Sun, A Y (1982) Eff ct of acut administration of ch orinat d wat r on hypotha amic nor pin phrin cont nt In: A baigés, J (d) Ana ytica t chniqu s in nvironmenta ch mistry 2: Proc dings of th S cond Int rnationa Congr ss, arc ona, Spain, Nov mb r 1981 P rgamon Pr ss, N w York, NY

Whit , G C (1999) Handbook of ch orination and a t rnativ disinf ctants 4th dition John Wi y & Sons, N w York, NY

WHO (1995) Disinf ctants and disinf ction by products In: WHO s minar pack for drinking wat r qua ity W or d H a th Organization, G n va, Switz r and (www.who int/wat r_sanitation_h a th/dwq/ n/S04 pdf; acc ss d F bruary 10, 2006)

WHO (1997) Disinf ction In: WHO s minar pack for drinking wat r qua ity Wor d H a th Organization, G n va, Switz r and (www.who int/wat r_sanitation_h a th/dwq/S13 pdf; acc ss d F bruary 10, 2006)

WHO (2003) Ch orin in drinking wat r ackground document for d v opment of WHO *Guidelines for Drinkingwater Quality* Wor d H a th Organization, G n va, Switz r and

WHO (2004) Guid in s for drinking wat r qua ity 3rd dition Wor d H a th Organization, G n va, Switz r and; acc ss d F bruary 22, 2006)

WHO (2006) Guid in s for saf r cr ational wat r nvironments Vo ume 2: Swimming poo s and simi ar nvironments Wor d H a th Organization, G n va, Switz r and; acc ss d Octob r 4, 2006)

Wlodkowski, T J and Ros nkranz, H S (1975) tag nicity of sodium hypoch orit for *Salmonella typhimurium* . tat R s , 31: 39 42

Wojtowicz, J A (2004) Dich orin monoxid , hypoch orous acid, and hypoch orit s In: Kirk Othmer ncyc op dia of ch mica t chno ogy, Vo um 8 - Fifth dition John Wi y & Sons, N w York, NY

Won s, R G , D ck, C C , Stad r, , Roark, S , Hogg, E , and Fr man, L A (1993) Lack of ff ct of drinking wat r ch orin on ipid and thyroid metabo ism in h a thy humans Environ H a th P rsp ct , 99: 375 381

Appendix A i f acr nym s

ANSI
American ional Standards Institute

ASTM
ASTM International (formerly American Society for Testing and Materials)

AWWA
American Water Works Association

bw
body weight

CDBPs
chlorinated disinfection by-products

DCA
dichloroacetic acid

DCAN
dichloroacetonitrile

DNA
deoxyribonucleic acid

DPD
, diethyl-p-phenylenediamine N

FACTS
free available chlorine test

FAS
ferrous ammonium sulphate

GSH
glutathione

HAAs
halogenated acetic acids

IARC
International Agency for Research on Cancer

ISO
International Organization for Standardization

LD_{50}
median lethal dose

MDC
minimum detectable concentration

MNC
mononuclear cell

NA
not available

NOAEL
no-observed-adverse-effect level

NSF
$ International

NTP
ional Toxicology Program (United States)

ppm
parts per million

SCC
Standards Council of Canada N

SM
Standard Method

triiodothyronine

4
thyroxine

CA
trichloro cetic cid

HMs
trih lometh nes

U.S. C C
United St tes Centers for Dise se Control nd Prevention

U.S. EPA
United St tes Environment l Protection A ency

WHO
World He lth Or niz tion

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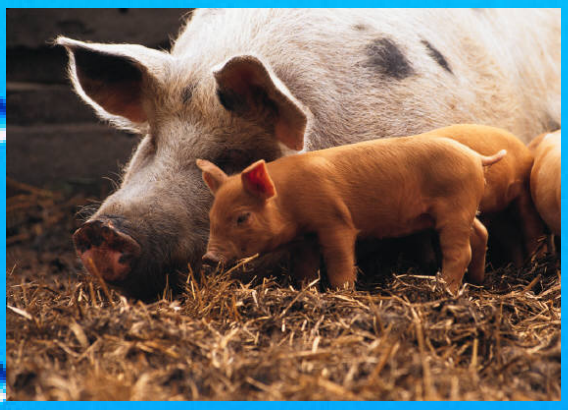
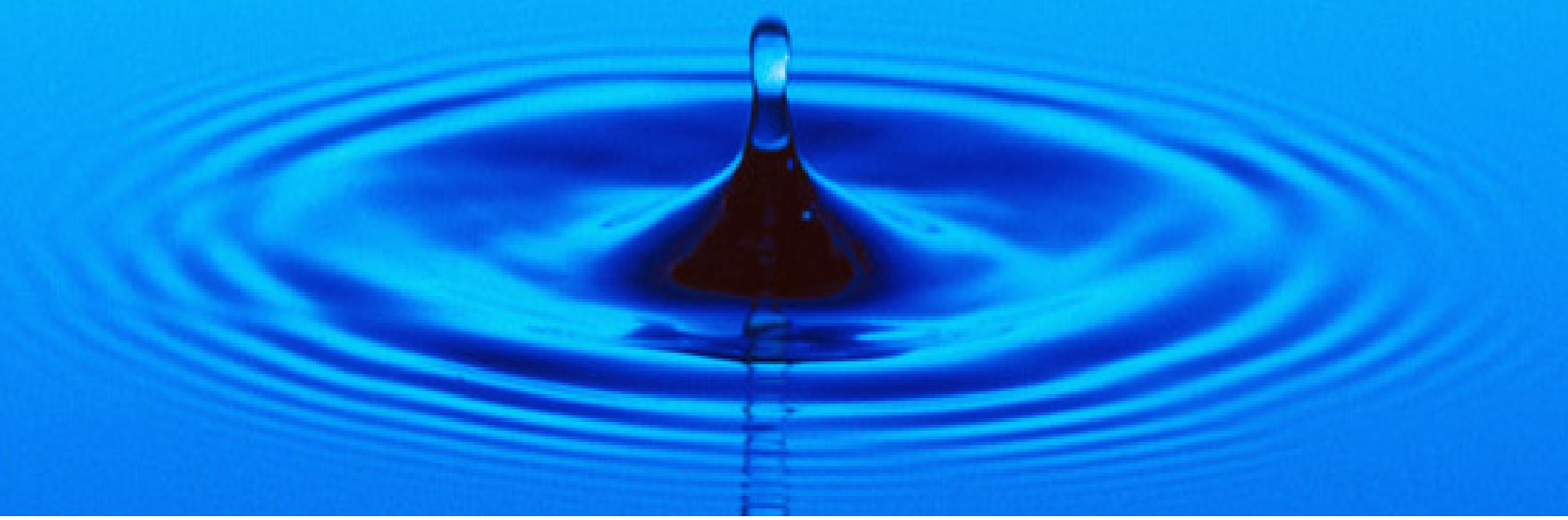
Livestock Water Quality

A Field Guide for Cattle, Horses, Poultry and Swine





Agriculture and Agri-Food Canada
Agriculture et Agroalimentaire Canada
University of Saskatchewan
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Livestock Water Quality

A Field Guide for Cattle, Horses, Poultry, and Swine

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Disclaimer

Foreword

The ultimate objective of the work undertaken here was to arrange an assembly of information on water quality issues and contaminants using various original research papers, textbooks, and other reputable sources, into one concise, and easy-to-interpret manual. This manual is intended to provide fundamental information to livestock and water quality specialists and other professionals on a wide range of water quality parameters and related physiological and/or toxicological effects. Many producers may also find the information useful in identifying problems and symptoms relating to water quality.

While preparing this document, a deliberate attempt was made to minimize the "excessive scientific" content, while focusing on factual interpretation of the knowledge in the context of practical applicability of the information. However, it is not uncommon that different scientific sources discussing seemingly the same water quality issues provided divergent results. Therefore, it is important to understand that data comparability may be a major problem in evaluation of water quality. In particular, it may be difficult to determine what is correct and what is incorrect, especially with the "experts" often disagreeing. In this context, the user of this guide should to be aware of a broad range of conflicting results or differing expert opinions. It is likely important to note cases where this occurs so that it is clear that the author felt the controversy worthy of mention.

While compiling the information for this guide, the author did not simply report the existing discrepancies, but rather, attempted to resolve conflicting information in the context of the overall knowledge of physiology, biochemistry, nutrition, and toxicology.

Although an effort was made to provide comprehensive interpretation of water quality data, it is important to understand the complex nature of biological responses of animals, in particular those that are genetically selected for high production traits. In this context, it is imperative that the high metabolic demand associated with constantly increasing production goals is taken into consideration in assessment of water quality standards, especially in the face of the increasing complexity of water contaminants.

There is a noticeable insufficiency of recent information on many aspects of water quality issues in contemporary livestock selected for superior performance characteristics. Without comparative research using today's high performance genetics, interpretation of water quality data is problematic at minimum.

No doubt, the success of Canadian livestock production depends on the availability of good quality water. However, in many areas where the livestock industry is prominent, water quality is poor, or at best marginally tolerable. It is important to understand that, at present, the elimination of all undesirable effects associated with water contaminants is not realistic under most circumstances. Therefore, a substantial effort has been made in this guide to emphasize the management of potential risks to livestock associated with water problems encountered under common field conditions.

Health effects of water contaminants are an important issue, but in reality, the economic success of the modern Canadian livestock industry is predominantly based on animal performance. The key elements of utmost importance, in terms of economic success in any sector of the contemporary livestock industry in Canada, are based on four fundamental parameters i.e. growth rate, feed conversion ratio, reproductive success, and product quality. Any of these parameters can be affected by water contaminants at a very subtle, sub-clinical, metabolic level.

Contributions

This work is the result of the collective efforts of several dedicated people. Mr. Larry Braul, PFRA, Agriculture and Agri-Food Canada, played a critical role as coordinator. He also prepared and compiled information on water types or conditions relevant to water contaminants in Saskatchewan, served as a reviewer of various drafts, and editor of the final version. Mr. Bob Klemmer, Saskatchewan Ministry of Agriculture, prepared background information relevant to feed and dietary components, and played a key role as reviewer and technical editor. Ms. Erin Zoski, PFRA, Agriculture and Agri-Food Canada, served as a technical editor, and prepared the document for printing.

Acknowledgements

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Thanks are also extended to Dr. Terry Hanley and Saskatchewan Watershed Authority for allowing the editors to use their water quality data base for Saskatchewan farms and communities.

TABLES

FIGURES

1. INTRODUCTION

1.1 The Significance of Water Quality in Livestock

Water is an essential nutrient which is involved in all basic physiological functions of the body. However, it is important to note that water, relative to other nutrients, is consumed in considerably larger quantities. Therefore, water availability and quality are extremely important for animal health and productivity. Limiting water availability to livestock will depress production rapidly and severely, and poor quality drinking water is often a factor limiting intake. Considering that water is consumed in large quantities, if water is poor quality, there is an increased risk that water contaminants could reach a level that may be harmful.

The water requirement and intake in livestock may vary depending on species and breeds of livestock, animal status, production mode, environment or climate in which livestock are raised. All these variables are directly or indirectly relevant to several aspects of water metabolism and physiology. In this context, it is necessary to understand water quality issues from the perspective of water intake physiology.

1.2 Brief Overview of Water Physiology

In order to maintain a physiological balance of water, most animals have to drink every few days to survive, and at least every other day to be productive. However, with regard to highly producing animals, provision of a large amount of clean, fresh water is essential.

The requirement for water is influenced by numerous factors such as the animal's activity, air temperature, humidity, respiratory rate, water intake, feed consumption, and several physiological factors such as age, reproductive status (e.g. dry, pregnant, lactating), milk production and many other factors.

1.2.1 Water Intake Physiology

***Gains and Losses of Water**:* The vast majority of water required by animals is obtained by drinking water. Intake of liquid dietary components containing high levels of water such as milk, by-products from the dairy industry, sugar industry by-products, liquid distiller grain by-products, etc. may fulfil a significant proportion of daily water requirement. Animals can obtain a substantial amount of water by eating feedstuffs containing high levels of moisture (e.g. lush pasture). Metabolic water is acquired in the oxidation of various dietary constituents (although feed itself may be limiting at times). Limited amounts of water can be supplied by absorbing water through the skin.

Water Turnover and Body Water Pool: Water is lost mostly through feces and urine, in respiration from the lungs and as sweat. There is a strong correlation between metabolic rate and body water turnover. Water turnover can be expressed in relation to

the size of the body pool rather than to body weight. For practical purposes, the body water pool is taken as 70% of live weight.

Metabolic rate and water turnover are higher in young and highly productive animals, and lower in older or less productive animals. However, water turnover may vary considerably depending on species specific physiological characteristics. For instance, in comparison to cattle, sheep and goats are more economical with water, turning it over at a rate of only 50-60% that of cattle in the same environment.

The greatest metabolic and physiological strain is placed on highly producing animals during lactation. Efforts of synthesis increase both energy and water consumption rates by 40-60%.

1.2.2 Water Quality Issues in the Context of Drinking Behaviour

Drinking is a vital part of the daily activities of livestock, particularly in the summer. Given a choice, cattle would prefer to drink water with moderate temperatures, rather than very cold or hot water, but overall, the temperature of drinking water has only a slight effect on drinking behaviour and animal performance. Observations on the behaviour of cattle in the field indicate that cattle having access to fresh water will consume more forage.

1.2.3 Water as a Coolant

Water metabolism is essential to the maintenance of body temperature. Ruminants such as sheep, goats and cattle dissipate internal and absorbed heat by evaporation of body water. The economy of water use is a desirable feature for livestock in arid or semi arid regions, but other factors such as food intake or growth rate may also be important. In animals exposed to heat there is an increase in water consumption.

1.2.4 Water Quality

The key properties that must be taken into consideration while assessing water quality for livestock include:

- sensory (organoleptic) attributes such as odour and taste,
- physiochemical properties (pH, total dissolved solids, hardness),
- chemical composition
    - toxic compounds (heavy metals, pesticides, herbicides, hydrocarbons, etc),
    - excess minerals or compounds such as nitrates, sodium sulphates,
    - biological contaminants (bacteria, algae, viruses).

The most common water quality problems affecting livestock production include high concentrations of minerals, sulphates, nitrates and nitrites, bacterial contamination,

heavy growth of blue-green algae and chemical contamination associated with agricultural and industrial activities.

As the adverse effects of water contaminants are directly related to the amount consumed, the greatest impact of water contaminants to livestock is often observed during hot weather when large volumes of water are consumed, and in particular when animals are fed low moisture feed.

River water is generally considered safer than pond or well water, because a large body of free flowing water provides more opportunities for natural biological decontamination processes. Nitrates may build up in well water by leaching of manure down through the soil or along the casing of a poorly constructed well. However, high nitrate water levels may come from other nitrogen sources, such as crop fertilizers. Water nitrate levels may fluctuate widely in surface water, but they are generally highest following wet periods and lowest during dry periods of the year.

Water quality may have significant impacts on an animal's production and health, therefore water for livestock should be tested periodically.

Water Sampling and Testing: Water for livestock should be tested periodically, in order to avoid problems that potentially may arise from poor water quality. Possible problems with water contamination can occur at the source (inherent factors) or at the level of watering device (acquired factors). Occasionally it may be necessary to distinguish the cause of contamination, and therefore a water samples representative of the source and watering container or device should be used for analysis.

It is important to stress that water quality may change over time, and therefore one should not rely on past analysis. Water testing should be done routinely, preferably every year, or at least every 2 years under normal circumstances, whereas any unusual situation such as changes in water smell, clarity, taste, or changes in animals eating or drinking habits, loss of performance, or health problems should immediately trigger the need for water testing.

Analysis should be done by an accredited laboratory. Producers should consult with their veterinarian or livestock specialist for assistance in selecting a laboratory. The scope of analytical objectives for water contaminants may vary depending on specific location or circumstances. Although this guide may provide basic information and tools for interpretation of water quality requirements, in more complex situations it is advisable that producers seek assistance in selecting more specific tests and interpreting the results.

An example of water analysis results for livestock water quality purposes under most common circumstances is presented in Table 1.1

Table 1.2 Example of water test results detailing tested parameters and their concentration.

Sample Details/Parameters	Result	Qualifier	D.L.	Units	Extracted	Analyzed
L111346-1 WELL WATER						
Sample Date:						
Matrix: WATER						
Basic Livestock Suitability						
Iron (Fe)-Extractable	10.1		0.005	mg/L	23-MAY-03	23-MAY-03
Chloride (Cl)	7		1	mg/L	26-MAY-03	26-MAY-03
Nitrate	<1		1	mg/L	29-MAY-03	29-MAY-03
pH and Conductivity						
TDS (Calculated from EC)	1660		1	mg/L	23-MAY-03	23-MAY-03
pH	7.2		0.1	pH	23-MAY-03	23-MAY-03
Conductivity (EC)	2600		0.2	uS/cm	23-MAY-03	23-MAY-03
ICP Cations and Hardness						
Calcium (Ca)	357		1	mg/L	26-MAY-03	26-MAY-03
Potassium (K)	12		1	mg/L	26-MAY-03	26-MAY-03
Magnesium (Mg)	180		1	mg/L	26-MAY-03	26-MAY-03
Sodium (Na)	79		1	mg/L	26-MAY-03	26-MAY-03
Sulfate (SO4)	1190		0.5	mg/L	26-MAY-03	26-MAY-03
SAR	0.9		0.1	SAR	26-MAY-03	26-MAY-03
Hardness (CaCO3 equivalent)	1630		1	mg/L	26-MAY-03	26-MAY-03

1.3 Understanding Water Quality Problems

The Federal government provides *CCME – Canadian Environmental Quality Guidelines* which is a set of non-binding recommended limits for a variety of parameters that affect water quality for humans, irrigation, recreation, and livestock.

With respect to livestock, there is plenty of information on water quality requirements, but very few practical solutions to deal with problems. For instance, according to the Canadian Guidelines, only high quality water should be available to livestock. In reality, it is often not practically possible to reach the official goals/guidelines due to unavailability of good quality water.

It will be some time before economically acceptable technology for water purification, at a scale required by the livestock industry, is developed. Therefore, in the present situation utmost attention should be focused on development of strategies for the management of current problems.

Identification of water contaminants is an essential component in the management of the associated problems. Most certainly, from the perspective of water quality specialists or veterinarians, knowledge on how to recognize the various problems associated with water contaminants is essential for the rapid detection of problems and effective management of the adverse effects. However, livestock producers should also have a basic understanding of possible adverse effects associated with water contaminants.

1.4 Management of Water Quality Problems

In the situation where water for livestock contains contaminants, water treatment should be recommended. However, if this is not practical, management of the potential risk associated with water must be approached from a local perspective with thorough consideration of any other contributing risk factors (feed, environment, etc.).

Intake of many elements that are excessive in water can be effectively managed through appropriate ration formulation. Thus, a solid understanding of the specific regional issues of water quality for livestock is important.

The problem of water contaminants in livestock should be recognized as early as possible, and definitively before the signs of adverse health effects are showing. Both producers and water specialists ought to be trained on how to recognize subtle adverse effects on growth rate, feed conversion ratio, reproductive success, milk yield, and product quality.

The importance of interactions of water contaminants with factors such as production mode or the nutritional and physiological status of the animal must be fully appreciated. In order to understand and recognize subtle problems resulting from water quality in livestock, it is important to understand how water contaminants affect physiological and biochemical parameters.

The current water quality guidelines provide recommendations of values for each contaminant. However, it is important to stress, that in view of the current knowledge, the effects of individual water contaminants cannot be deliberated as a “stand alone” problem, but rather must be considered in the context of complex interactions with other dietary and/or environmental variables with a strong analytical emphasis on the potential adverse effects resulting from:

- cumulative effects
- additive effects
- synergistic effects

Further, it is important to understand that the risk of adverse effects associated with any particular individual contaminant in the water should not be dismissed based exclusively on a perceived safe concentration in water. This is because if the same factor is also present in the feedstuffs, along with the water contribution, the cumulative content of this contaminant may exceed the threshold and trigger metabolic or even toxic effects.

In order to provide a solution to the many problems that may be associated with a wide range of water contaminants, the current approach to management of water quality issues in livestock must take into consideration direct effects of water contaminants, as well as their interactions with other dietary components.

1.4.1 Importance of Water Intake: When evaluating the impact of water contaminants, it is important to consider water intake. From management of water quality problems, it seems obvious that when water intake increases, intake of any contaminant present in this water is increased in the same proportion, yet the impact of water intake is frequently underestimated in many popular publications. Therefore, it is important to remember that daily water intake varies widely depending on class of livestock, animal activity, and environmental temperature, and is greatly influenced by physiological variables including: 1) production parameters, 2) developmental stage, 3) age, 4) physiological status, and 5) nutritional status. It has to be stressed that these variables are of enormous importance in terms of susceptibility to adverse reactions.

1.5 Effects of water quality on feed and water intake

Several water quality parameters such as pH, salinity, odour, taste etc., may affect palatability. Contaminants in water may affect intake of both water and feed, but the responses may vary depending on specific metabolic features of animals.

For instance, high sulphate levels in water significantly decreased water intake in cattle (Weeth and Hunter, 1971; Grout *et al.,* 2006). Reduction of TDS in water from about 4,400 to 440 mg/L resulted in increased water intake and feed intake (Challis *et al.,* 1987). If water quality affects feed intake, reduced feed consumption may affect performance (Weeth and Capps, 1972; Loneragan,*et al.,* 2001). Moreover, the specific features of sulphur metabolism in ruminants may result in a wide range of metabolic effects associated with high levels of sulphate in drinking water (for details see section on sulphur).

On the other hand, in animals that do not metabolize water contaminants such as sulphate, the responses may be completely different. For example in weanling pigs

offered high TDS and sulphate drinking water, the intake of water actually increased (Maenz *et al.,* 1994), and no overt metabolic effects were observed.

Horses are more sensitive to some specific aspects of water quality. Although the risk of direct health effects associated with water contaminants is relatively low, water quality may have a tremendous impact on water palatability, and water intake by horses may decrease substantially when water is poorly palatable. Inadequate water intake may increase the risk of intestinal impactions and colic. Further, dehydration may be detrimental to the horse's health, and deficiency of water may result in death.

1.6 Water Quality Guidelines

Water quality guidelines are developed to allow assessment of the acceptability of water for the specific purposes. The Canadian Council of Resource and Environment Ministers developed extensive guidelines for livestock in 1987 (CCREM, 1987) based on the existing guidelines from other countries or from provinces. As additional scientific information became available, many of the livestock guidelines were revised, the last revision occurring in 2005.

The existing CCME water quality guidelines are developed only for the protection of the animal and do not address potential accumulation of contaminants that may be passed on to consumers through milk or meat. Accumulation of the contaminant from other sources, such as feed is sometimes addressed, often with the addition of a safety factor of about five times. The variability in sensitivity for different species and life stages is addressed by basing the livestock drinking water quality guidelines on the most sensitive species at its most sensitive life stage (i.e. to safeguard animal health). An uncertainty factor is often applied based on the quality and extent of the data. Antagonistic or synergistic aspects between various contaminants are rarely addressed as these factors complicate an already complex and challenging guideline derivation. Succinctly stated, synergistic effects of multiple contaminants in water, feed and environmental exposure, is not well understood. For more information on the derivation of the CCME water quality guidelines for livestock, refer to the "Protocols for Deriving Water Quality Guidelines for the Protection of Agricultural Water Uses (Irrigation and Livestock Water) published in the Canadian Environmental Quality Guidelines by the Canadian Council of Ministers of the Environment, 1999 (http://documents.ccme.ca/download/en/131/)

The water quality guidelines for livestock drinking water must be approached with an understanding of the challenges in identifying a single value for each contaminant and the factors that are applicable for specific situations. For instance, on the assumption that most guidelines are conservative, a mature bull, in a cool environment with high moisture feed will likely tolerate water sulphate at a much higher concentration than specified by the guidelines. On the other hand, for a young calf grazing on dry grass in extremely hot weather, the CCME guidelines for contaminants such as sulphate or nitrate may exceed tolerance levels, especially if sulphur or nitrate contributions from

feed are already marginally high (for more details see sections on sulphate and nitrates respectively).

The goal of a guideline in livestock drinking water is to ensure that concentrations of contaminants less than the guideline will ensure no significant health or production effect. Where data is sparse or lacking, guidelines may be based on protocols used for assessment of drinking water standards for humans. Application of these protocols for derivation of the livestock water quality guidelines results in values that are often excessively conservative.

Many provinces rely on Federal livestock drinking water guidelines, and in some cases these guidelines are used to approve the development of intensive livestock operations. While much is not fully understood about the complex nature of water quality on animal health and livestock food products (meat, dairy), water quality is clearly a critical input factor in livestock production and must not be taken for granted. Conversely, guidelines that may be too conservative could have an impact on the cost of production, and unnecessarily negatively impact the sector. Provincial governments need to be cautious in using CCME guidelines in a regulatory fashion, as the acceptable concentration of a contaminant is very situational. As knowledge improves, both regulators and the livestock sector will be able to make better decisions regarding the acceptability of water for specific applications.

Decisions to improve poor quality source waters used for livestock drinking water by using water treatment devices or procedures should be based on economics combined with a better understanding of water related factors and how these may impact animal health, animal production, and product quality. Such an approach will allow improved decision-making, healthier animal populations, reduced risk management in livestock production, and better market potential for a safe and healthy food product.

The present document provides additional information to enhance the understanding of factors that may play a role in the evaluation of the livestock drinking water guideline value for a specific situation. Over time, it is expected that the CCME guidelines will be refined as new scientific information becomes available.

The following table summarizes the 2005 CCME guidelines for substances other than pesticides.

Table 1.2 CCME (2005) Livestock Guidelines for Selected Constituents

(for complete table see Appendix A)

Water Contaminant *	CCME Guideline (mg/L)	Date Introduced or Revised
Arsenic	0.025	1997
Cadmium	0.08	1996
Calcium	1000	1987
Cyanobacteria	Avoid heavy growths	1987
Chloride	None	
Chromium	0.05	1997
Cobalt	1.0	1987
Coliforms, fecal**	None	
Coliforms, total**	None	
Colour***	Narrative	1999
Copper	0.5 to 5.0	1987
Cyanide	None	
Fluoride	1 to 2	1987
Hardness	None	
Hydrogen Sulphide	None	
Iron	None	
Lead	0.1	1987
Magnesium	None	
Manganese	None	
Mercury	0.003	1987
Molybdenum	0.5	1987
Nickel	1.0	1987
Nitrate + Nitrite	100	1987
Nitrate nitrogen	23	1987
Nitrite	10	1987
Nitrite nitrogen	3.0	1987
Potassium	None	
Selenium	0.05	1987
Silver	None	
Sodium	None	
Sulphate	1000	1987
TDS	3000	1987
Uranium	0.2	1987
Vanadium	0.1	1987
Zinc	50	1987

Source: CCME Canadian Water Quality Guidelines for the Protection of Agricultural Water Uses – Summary Table – Update October 2005

* CCME factsheets exist for arsenic, cadmium, chromium and colour. See Canadian Guidelines for the Protection of Agricultural Water Uses – Arsenic, 1999; Cadmium, 1999; chromium, 1999; Colour, 1999 (http://documents.ccme.ca/)

** CCREM 1987 suggests that only high quality water should be provided to intensive livestock operations*** Narrative suggests a guideline similar to that for humans which is an aesthetic objective of 15 TCU. See Canadian Guidelines for the Protection of Agricultural Water Uses – Colour, 1999 (http://documents.ccme.ca/download/en/114/) for more information

2. MICROBIOLOGICAL CONTAMINANTS

2.1 Cyanobacteria

Natural toxins originating from cyanobacteria (blue-green algae) are a primary concern in drinking water for livestock. Toxigenic species occur in at least 18 genera. Cyanobacteria are known to produce acute hepatotoxins, cytotoxins, neurotoxins, and toxins causing gastrointestinal disturbance.

Cyanobacteria may grow in surface waters of freshwater lakes and rivers throughout the year, but are typically very prevalent during the summer months when they may bloom and pose a risk to livestock. Evidence is emerging that the number of incidences of cyanobacterial blooms has been increasing in recent years. It has been hypothesized that one of the reasons for the apparent increase is a corresponding increase in the load of nutrients such as nitrogen and phosphorus in the water (Chambers *et al.,* 1997).

Cyanobacteria in drinking water sources are an important health issue in both humans and animals (Chorus, 2001). Livestock deaths have been attributed to cyanobacterial toxins (Puschner *et al.,* 1998). The problems occur across Canada, but are particularly prevalent in the Prairies where cyanobacterial poisoning has resulted in a number of livestock deaths (Manitoba Environment, 1998).

In Saskatchewan many cattle die every year from drinking water containing toxins. According to Peterson (2000), it is highly likely that fatalities in livestock are greatly under-reported because there is lack of expertise in accurately recognizing cyanobacterial poisoning.

Stagnant waters or those with decreased rate of flow may encourage the growth of cyanobacteria. Heavy algae growth occurs most commonly during summer and fall in shallow, calm water rich in organic nutrients. Water bodies that are protected from the wind and those without aeration are prone to producing prolific cyanobacterial growth. Microcystin and an alkaloid hepatotoxin are considered to be the major toxic agents. There are several other species of algae that contain a variety of toxins.

Heavy cyanobacteria growth does not necessarily mean high levels of toxin. The trigger for cyanobacteria to produce toxins is not completely understood. If the cyanobacteria growth is not of the *Microcystis* species, there is a low probability of having high toxin levels.

Identification of cyanobacteria and especially the *Microcystis* species is difficult. An expert can identify the various species under a microscope, however, in the field one can only determine whether the bloom is filamentous (stringy) or planktonic. Filamentous algae are easily removed from water by hand whereas planktonic algae/cyanobacteria are single celled and will slip through your fingers. No toxin-producing cyanobacteria is of the filamentous type. Some laboratories provide determination of the algae species and the Saskatchewan Provincial Laboratory was

providing a test for Microcystin LR in 2008. More information is available in the publication "*Algae, Cyanobacteria and Water Quality*" available from AAFC-PFRA Water Quality Division.

Cyanotoxin toxicological tests clearly demonstrate that these toxins have adverse health effects. There is plenty of information available regarding acute toxicity associated with cyanotoxins in livestock, but the levels of toxins causing sub-clinical problems in livestock are poorly characterized. Only a few toxicological trials attempted to determine safe levels of intake of cyanobacterial cells or toxins for domestic animals, and the research is fragmented and the findings are inconclusive. Table 2.1 provides guidelines extrapolated from known toxic effects at Lowest Observed Adverse Effect Level (LOAEL).

Table 2.2 Guideline for calculated tolerance levels (No Observed Effect Level) of microcystin LR toxicity equivalents and number of cell of *Microcystis aeruginosa.*

Livestock Category	Body weight (kg)	Peak water intake L/day)	Calculated Total Toxin Level (µg/L)	Equivalent Cell Number (cells/mL)
Cattle	800	85	4.2	21000
Sheep	100	11.5	3.9	19500
Pigs	110	15	16.3	81500
Chicken	2.8	0.4	3.1	15500
Horse	600	70	2.3	11500

Adopted from ANZECC 2000.

Considering that some cyanotoxins can induce severe injury to the liver, it is very likely that even sub-clinical effects can be of toxicological significance. In view of the possibility of liver damage, even at a sub-clinical level, adverse effects of other water born contaminants may be exacerbated, because liver is the primary organ responsible for detoxification of any ingested toxins.

The potential adverse effects associated with long term, low level exposure to cyanotoxins are poorly understood, but the problem of such exposure is not a trivial issue, because cyanotoxins in water may persist long after the bacteria has died out, particularly when cyanobacteria are killed with the help of algaecides.

Management Options: It is recommended that water contaminated with cyanobacteria should be avoided until the level of toxins is determined or until the water is treated and toxins are allowed to dissipate.

The prevention of cyanobacterial blooms is a more cost effective means of reducing risk of toxicity than the typical water treatment process. Reducing the growth potential of cyanobacteria, by lowering nutrient availability, should be the primary goal for reducing the risks associated with cyanobacterial blooms (Downing *et al.,* 2001).

A common approach to eliminating blooms is the use of chemical algaecides. Some references suggest that copper sulphate added to pond water up to a concentration of 1 ppm (1 mg/L) has been used successfully to kill algae blooms, but will probably be harmful to other types of aquatic life. AAFC-PFRA recommends a lower dosage, from 0.06 to 0.25 mg/L based on the surface area of the water body. Treatment at the beginning of the bloom at a low dosage is more effective than later treatment as it allows the zooplankton to populate and assist in control of algae and cyanobacteria.

It has to be remembered that a sudden release of toxins can occur when cyanobacterial blooms die. Hence, the risk of toxicity may not be effectively eliminated using chemical algaecides, and in fact the risk of exposure to toxin may increase if the application is introduced at the wrong time.

For more information on chemical treatment of water refer to the publication “Copper Treatments for Dugouts” available from the AAFC-PFRA Water Quality Division.

2.2 Pathogens: Bacteria, Protozoa, Viruses

A variety of microbial pathogens can be transmitted to livestock from drinking water sources contaminated by a wide assortment of causative factors. The risk of contamination is greatest in surface waters (dams, lakes, dugouts, etc) that are directly accessible by stock, or, that receive runoff or drainage from intensive livestock operations or human waste.

Historically, the incidence of groundwater contamination by pathogens, particularly deep wells, has generally been considered to be low. However, in recent years, agricultural activities focused on large intensive livestock operations created localized environmental conditions where the possibility of biological contamination of ground water has become a major concern. In particular, shallow groundwater supplies in sandy soils are at high risk of being contaminated. Poorly sealed and located wells also are responsible for a large percentage of contaminated aquifers.

Microbiological Contaminants

The pathogens of greatest concern in water supplies for farm animals include enteric bacteria such as *E. coli, Salmonella* and *Campylobacter jejuni.* Other bacterial diseases known to affect livestock that may be transmitted through water supplies include *Leptospira*, *Burkholderia (Pseudomonas) pseudomallei, and Clostridium botulinum.* Water supplies have been implicated in infections such as Newcastle Disease and Infectious Bursitis in poultry (CCREM 1987). Hence, a number of serious pathogenic conditions in farm animals caused by bacteria and viruses can be transmitted via contaminated water sources.

Notably, a very important (and probably most likely), cause of biological contamination of water sources is associated with the animal industry itself. For instance, in the situation of intensive livestock operation, the risk of water source contamination with animal waste may be very high. One way to assess water quality for microbial contamination with pathogens of animal origin is to measure numbers of bacteria that are likely associated with animal waste. For this purpose, indices such as water counts of coliform bacteria or *E.coli* are most commonly used, because these kinds of microorganisms are common in animal feces. Excessive presence of these bacteria in drinking water indicates poor hygiene.

Presence of *E.coli* in drinking water for human consumption usually triggers immediate administrative action. However, strict tolerance values for livestock have not been investigated. In most jurisdictions, it is generally recommended that drinking water for livestock should contain less than 100 coliforms/100 mL.

The following table summarizes the levels of coliform bacteria and *E.coli* found in the groundwater in Saskatchewan.

Table 2.2 Total Coliform Bacteria and *E.coli* Bacteria Counts in Saskatchewan Groundwater.

	Coliform Bacteria		***E.coli* Bacteria**	
Bacteria Counts (CFU* per 100 mL)	**No. of Samples**	**Percent of Total**	**No. of Samples**	**Percent of Total**
≤1	**2164**	**74.7**	**321**	**99.1**
1 to 10	**278**	**9.6**	**2**	**0.6**
10 to 100	**271**	**9.3**	**1**	**0.3**
>100	**185**	**6.4**	**0**	**0.0**

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base
*CFU - colony forming units

As evidenced by data presented above, the bacteria levels in groundwater appear to be generally low, but such data must be interpreted cautiously. A low count at the source level does not mean that there is no problem. Recent studies suggest that bacterial

contamination of drinking water at the point of watering may be a concern (Van Donkersgoed *et al.,* 2001; Sargeant *et al.,* 2004).

The amount of bacteria in surface water depends on the number of livestock and wildlife in the vicinity of the dugout and the source of the water. Dugouts in rural areas that are not contaminated usually have *E.coli* counts of 20 to 100 per 100 mL, with wildlife being the predominant source. With direct watering of cattle, these counts may increase to greater than 10,000 counts per 100 mL for extreme cases.

Of particular importance is the risk of contamination with a specific pathogen *E. coli O157*. These bacteria have been detected in cattle water sources, including ponds, free-flowing water such as streams, as well as water tanks (Faith *et al.,* 1996; Hancock *et al.,* 1998; Shere *et al.,* 1998; Van Donkersgoed *et al.,* 2001; Renter *et al.,* 2003).

2.2.1 Risk Associated with E. coli O157

The bacteria, *E. coli* O157:H7 and the *E. coli* O157:H-non-motile variants, generally referred to as *E. coli* O157, have become a significant public health concern throughout the world. From the perspective of livestock water quality issues, these bacteria should be recognized as a potential hazard because of its ability to survive and multiply in water (Armstrong *et al.,* 1996; Coia, 1998; Wang and Doyle, 1998).

Cattle are considered a primary source of these bacteria, and water contaminated with cattle feces, as well as direct or indirect contact with live cattle, are considered major routes of human infection. Cattle that carry *E. coli O157* are asymptomatic, but in humans this pathogen creates severe disease, and in many cases is the cause of death. The risk to the general population from contaminated water sources is very high (remember Walkerton, ON).

It is noteworthy that pathogenic *E. coli* O157 can easily be disseminated among cattle through contaminated water sources (Shere *et al.,* 1998), and drinking water can be a long-term reservoir and a persistent source of cattle exposure (Lejeune *et al.,* 2001).

Although cattle that carry E. coli O157 are not affected, these bacteria are important human pathogens. The mere presence of these bacteria in water sources may increase the risk of product (milk, meat) cross-contamination, which may have far reaching consequences on consumer confidence. Thus, water quality programs should be among the key control points in farm pathogen reduction strategies.

Microbiological Contaminants

At the herd level, *E. coli O157* is ubiquitous in both dairy and beef cattle operations (Faith *et al.,* 1996; Hancock *et al.,* 1998; Shere *et al.,* 1998; Van Donkersgoed *et al.,* 2001; Renter *et al.,* 2003). In situations more specific to the feedlot environment, contamination of drinking water with *E. coli* O157 appears to be wide spread problem.

VanDonkersgoed *et al.,* (2001) reported the presence of these bacteria in 12% of water tanks from pens containing pre-slaughter cattle. A more recent study (Sargeant *et al.,* 2004) showed at least one water tank was positive for *E. coli* O157 on 60% of the feedlots.

The health hazards associated with pathogens in both humans and livestock are well documented. A contaminated water supply may introduce high numbers of organisms into a group of animals, and this scenario may create a significant 'multiplier' effect through the food chain. The potential impact of pathogens such as *E. coli* O157 must be taken seriously in the context of water quality issues. In modern agriculture, strict management of water supplies for livestock must take into consideration contamination with water-borne microbial pathogens. The effort to address these problems should be focused on protection of water sources from contamination.

3. WATER REQUIREMENTS FOR HORSES

3.1 Water Supply

An adequate supply of good-quality, palatable water is essential for horses, but the exact water requirements in the horse are difficult to define because numerous dietary and environmental factors affect water absorption and excretion. Under proper management, the horse should have free access to fresh, clean water at all times.

In the horse, water is absorbed from most sections of the digestive tract. After a meal, water is needed in the gut to dilute the digesta and maintain the uniform consistency of the digesta throughout the gut. If water is consumed without any food being eaten, the water is absorbed more rapidly and completely. Dietary factors that may affect absorption include complex polysaccharides. These compounds tend to form gels in the gut and reduce water absorption.

The regulation of drinking is a highly complex physiological process, induced as a result of dehydration of body tissues. Most animals drink during or soon after eating and frequency of drinking and the water consumed increase in hot weather. When an animal is thirsty salivary flow is usually reduced, and dryness of the mouth may stimulate drinking.

Physiological variables such as age, growth rate, or lactation are major factors influencing water requirements for horses. Adult horses conserve body water more efficiently than foals, so foals dehydrate more quickly than adults. Adult horses at maintenance require a minimum of 2 litres of water per kg of dry food, whereas young growing horses may require 3 litres per kg of dry food. An adult horse needs about 5 litres of water per 100 kg of bodyweight for maintenance. Foals have a greater requirement for water than an adult horse in proportion to their size (Table 3.1).

Table 3.2 Changes in daily water intake of growing foals.

Age (days)	Water intake (kg)
11-18	Nil
30-44	3.9
60-74	5.5

Adopted from (Martin *et al.,* 1992).

The horse's water requirements may vary substantially depending on ambient temperature and humidity, water loss (e.g. sweating, urine condensation), and water content of feed. As in other animals, water requirements increase as environmental temperature increases. For instance, a rise from 15°C to 20°C in temperature will increase water loss by 20 per cent and therefore will increase an adult horse's water requirement by about 5 litres. However, from a water physiology stand point, higher water needs are mainly associated with the rate of water loss.

Horses

Feed composition has also a major impact on water intake. The amount of water provided by green forage can be very substantial. In fact, the resting horse grazing grass with moisture content over 70% may not need to drink any water. On the other hand, diets that are dry or high in salt will increase the horse's thirst.

3.2 Water Deficiency

Inadequate water intake is detrimental to the horse's health, and deficiency of water may result in death. The signs of inadequate water intake include decreased dry feed intake, followed by decreased physical activity. Inadequate water intake may increase the risk of intestinal impactions and colic.

Water deprivation for 24, 48, and 72 hours decreased the normal resting horse's body weight 4%, 6.8%, and 9%, respectively, when the ambient temperature was 63-81°F (17- 27°C). At an ambient daytime maximum temperature of 104°F (40°C), body weight decreased 11 to 13% after 60 hours, and 14 to 16% after 72 hours of water deprivation. Signs of dehydration, such dry mouth and sunken eyes are evident when 6% or more loss of body weight has occurred.

Water quality may have a tremendous impact on water palatability, and water intake may decrease substantially when water palatability is poor.

3.3 Water Quality

The single most reliable indication of water quality for horses is the amount of total dissolved solids (TDS) in the water. A TDS of 6,500 ppm constituting common mineral contaminants is generally considered the safe limit in water for horses. However, if the bulk of TDS is comprised mainly of minerals that may cause adverse effects, this parameter must be interpreted cautiously.

Horses can tolerate fluoride intakes two to three times greater than cattle. According to Lewis (1995), water fluoride at a concentration of 4 ppm is considered to be marginally safe for horses, but water containing more than 8 ppm should be avoided.

Chronic selenium toxicity has been reported as a result of consumption of water containing 0.0005 to 0.002 ppm selenium, but short term intake of water with Se concentrations below 0.01 ppm are not generally considered harmful.

Horses may develop some degree of adaptation to some water contaminants. For instance, water sulphate concentrations exceeding 1000 ppm may initially cause diarrhoea, but horses following adaptation can tolerate two to three times this concentration.

It is generally assumed that minerals such as sodium, potassium, calcium, magnesium, iron, chloride, and sulphate at levels commonly found in water are not toxic to horses

under most practical circumstances. However, at very high concentrations, these contaminants may affect water palatability, and of course, this may lead to decreased water intake and dehydration.

On the other hand, many potentially toxic compounds present in water do not reduce water palatability and water intake, and therefore they are potentially more harmful than those that affect palatability. A number of compounds that may be present in water can pose a toxicological hazard.

Toxic water contaminants include pesticides, herbicides, heavy metals, nitrites/nitrates, industrial pollutant, and microorganisms. It is noteworthy that, in comparison to other classes of livestock, horses appear to have higher tolerance to some contaminants, but may be more susceptible to adverse effects of others. Table 3.2 presents the recommended upper limits for some compounds in drinking water for horses with a potential to become harmful.

Although horses may appear to be more tolerant to some water contaminants, it has to be stressed that water quality for horses may not present so much of an overt health problem, but rather an aesthetic issue. Some horses may be particularly choosy and outright reject contaminated water.

In order to be unreservedly accepted by horses, water must be free from pollution by sewage, farm chemicals, or industrial contaminants.

Nitrate toxicity is rare in horses, and if it occurs, is most often associated with high nitrate levels in forage. Nevertheless, water may contribute significantly to the overall burden of dietary nitrites/nitrates. Water containing high nitrate levels resulting from surface contamination from manure and barnyard runoff is usually also high in microorganisms.

In many situations, bacteria in water pose a greater threat than the other water contaminants. Most infectious diseases can be transmitted via contaminated water. The sanitary quality of water is expressed by counting numbers of coliform bacteria. Not all coliform bacteria are harmful, but their mere presence is a very sensitive indicator of poor sanitary status. Commonly, when coliforms are present, there is a high risk that other infectious bacteria and viruses may be present in the water. Potentially dangerous microbiological contamination can occur in drinking water. For instance, water polluted by urinary excretion of leptospira by rodents can cause abortion in mares and death of foals.

Horses are sensitive to algae and toxins produced by cyanobacteria (blue-green algae). It is recommended that water contaminated with algae should be avoided. Some

species of cyanobacteria, which grow on pond and lake water, may result in poisoning. Cyanobacteria poisoning in domestic livestock may cause photosensitization, sudden death, weakness, bloody diarrhoea, tremors, and convulsions. Clumps of algae may be found in the gastrointestinal contents of animals that die suddenly. See Section 2.1 for more information on Cyanobacteria.

Table 3.2 Recommended Upper Safe Levels of Water Contaminants for Horses. Column with values recommended for other classes of livestock is included for comparison.

Water Contaminant	Horses (mg/L)*	Livestock (mg/L)**
Arsenic	0.2	0.025
Cadmium	0.05	0.08
Calcium	500	1000
Chloride	3000	NA
Chromium	1	0.05
Cobalt	1	1
Copper	0.5	0.5 to 5.0
Cyanide	0.01	None
Fluoride	2	1 to 2
Hardness	200	NA
Hydrogen Sulphide	0.1	NA
Iron	0.3***	NA
Lead	0.1	0.1
Magnesium	125	NA
Manganese	0.05***	NA
Mercury	0.01	0.03
Nickel	1	1
Nitrate	400	100
Nitrate nitrogen	100	23
Nitrite nitrogen	10	3
Potassium	1400	NA
Selenium	0.01	0.05
Silver	0.05	NA
Sodium	2500	NA
Sulphate	2500	1000
TDS	6500	3000
Vanadium	0.1	100
Zinc	25	50

* Adopted from Lewis, 1995;
** CCME Guidelines for Livestock (2005), NA-recommendation not available
*** Most likely for distribution purposes

4. WATER REQUIREMENTS FOR POULTRY

4.1 Water Supply

As with other animals, water for poultry must be regarded as an essential nutrient, and adequate supply of clean, good quality water is essential in order to fully utilise the potential of modern poultry genotypes selected for superior performance characteristics.

The requirement of poultry for water depends on numerous environmental variables such as temperature and relative humidity, the composition of the diet, and production parameters (growth rate, egg production). Examples of water consumption for various classes of poultry are presented in Table 4.2.

Table 4.1 Water Consumption (ml of water per week per bird) in various classes of poultry.

Age (weeks)	Broiler Chickens	White Leghorn Hens	Brown Egg Laying Hens	White Turkeys (Males)	White Turkeys (Females)
1	225	200	200	385	385
2	480	300	400	750	690
3	725	-	-	1135	930
4	1000	500	700	1650	1274
5	1250	-	-	2240	1750
6	1500	700	800	2870	2150
7	1750	-	-	3460	2640
8	2000	800	900	4020	3180
9	-	-	-	4670	3900
10	-	900	1000	5345	4400
11	-	-	-	5850	4620
12	-	1000	1100	6220	4660
13	-	-	-	6480	4680
14	-	1100	1100	6680	4700
15	-	-	-	6800	4720
16	-	1200	1200	6920	4740
17	-	-	-	6960	4760
18	-	1300	1300	7000	-
19	-	-	-	7020	-
20	-	1600	1500	7040	-

Based on data compiled from National Research Council, 1994.

Although there is large individual variability, it is generally assumed that water consumption in birds is approximately double the amount of feed consumed. Water intake can be influenced by diet form and composition. For instance, in comparison to mash diets, poultry offered pelleted or crumbled diets will increase both feed intake and

water intake. Increasing crude protein in the diet will increase water intake. Also, dietary salt content will influence water intake.

4.2 Defining Water Quality Parameters for Poultry

Drinking water used for poultry may contain considerable amounts of contaminants including various metals, sulphates, and nitrates. These compounds are usually readily absorbed from the gastro-intestinal tract, but in most practical situations, it does not appear that common water contaminants present any serious risk to poultry health. However, it should be noted that, although overt health effects are not likely to occur, water quality may have significant impact on production parameters in poultry highly selected for performance.

A high concentration of minerals (usually those associated with water hardness) may result in precipitation of salts in watering equipment, and this may restrict water flow, or in some situations, water lines may be completely plugged up. This may lead to inadequate water supply, and consequently water deprivation may occur. Water deprivation may have adverse effects on the growth rate in meat type poultry and egg production in laying hens. Water deprivation may result in increased morbidity and mortality.

It is important to stress that, if access to water is interrupted for a prolonged period of time, the restoration of watering must be managed carefully in order to avoid the situation where “water intoxication” may lead to mortality. Young turkeys are especially susceptible to this condition.

The commonly used parameters of water quality such as pH, hardness, or electrical conductivity are not very useful in predicting the effects of water contaminant on poultry performance. However, pH of water is likely the most important factor to consider while assessing the suitability of water as a medium for delivery of medication.

4.3 Potential Problems Associated With Water Contaminants in Poultry

With the exception of some very specific localized situations, under practical conditions, most water mineral contaminants, including heavy metals, would not present serious health problems in poultry. However, the potential impact of water contaminants on product quality should not be ignored, as some compounds may be deposited in eggs, meat, or liver. Also, several studies suggested that water quality issues in poultry are of significance for optimal performance.

Research regarding water quality issues for poultry is fragmented and, for the most part, outdated. In the older literature several reports indicated drastic increases in the incidence of damaged eggshells associated with drinking water. Balnave and Scott (1986), who investigated an eggshell quality problem on a commercial farm, identified well water as a possible cause. The water was reported to contain 293 ppm Na, 38 ppm

Ca, 155 ppm Cl, 46 ppm SO4, and 49 ppm nitrate N. In subsequent experiments they found that adding low levels of NaCl, KCl, CaCl, MgSO4, CuSO4, or NaNO3 to municipal drinking water over a 6-wk period substantially increased the incidence of cracked, broken, and soft shells, especially in those groups receiving the Cl ion.

However, most of the efforts in the past were devoted to investigation of salt (sodium chloride). The effects of salt on eggshell quality reported in the literature are highly variable. For instance, up to 6% dietary NaCl over a 21-d feeding period was not found to significantly reduce egg specific gravity by Damron and Kelly (1987). Adding up to 2,000 ppm NaCl resulted in more than half of the eggs from 80- to 95-wk-old hens showing defective shells (Yoselewitz *et al.,* 1988). The production of defective shells occurred more rapidly when saline water was given to 40-wk-old hens than to hens during the first few weeks of lay. But interestingly, saline drinking water in pullets before sexual maturity appears to have no detrimental effects on subsequent eggshell quality (Yoselewitz and Balnave, 1989). A more recent report by Pourreza *et al.,* (1994) showed mixed results. Eggshell thickness was reduced by 2,000 ppm NaCl in drinking water, but not by 1,000 ppm. In contrast to other literature reports, visually determined shell defects and egg specific gravity were not adversely affected by NaCl supplementation of layer drinking water (Damron, 1998, Chen and Balnave, 2001).

The effects of saline water on reproductive performance were studied by Zhang *et al.,* (1991). The incidence of eggs with defective shells doubled in hens receiving the saline drinking water at a level of 2 g NaCl/L. There was a significantly (twofold) higher incidence of embryonic deaths and a significantly lower (13%) hatchability of fertile eggs. For every 100 eggs laid, the numbers of settable eggs and chicks hatched were significantly reduced in hens receiving the saline drinking water. The saline water reduced the numbers of hatched chicks by 20%. The water treatment given to the cockerels had little effect on reproductive performance (Zhang *et al.,* 1991).

Studies on other contaminants of water are limited. In one study, Merkley and Sexton (1982) reported that fluoride at the level of 100 ppm in the drinking water did not affect reproductive performance of either pullets or cockerels, and no effects of fluoride on progeny growth were noted.

Interactions between drinking water contaminants and suboptimal nutritional status for performance and immune function in male broiler chickens were studied by Vodela *et al.,* (1997a,b). The latter authors investigated the effects of experimental drinking water containing a mixture of arsenic, benzene, cadmium, lead, and trichloroethylene (TCE) at low concentrations (0.80, 1.3, 5.0, 6.7, and 0.65 ppm respectively) and high concentrations (8.6, 13, 50, 67, and 6.5 ppm respectively). According to the authors, this set of chemicals was selected because they are among the most common contaminants found in ground water near hazardous waste sites. Both low and high concentrations of the chemical mixture, in comparison to chickens drinking normal water, affected feed consumption, body weight, and immune function. Interestingly,

even at low concentration, the chemical mixture significantly decreased egg production and egg weight, and increased the percentage of embryonic mortality.

Recommendations with regard to maximum, tolerable, or threshold values for poultry water supplies vary substantially. For instance, reported tolerances for iron may range from 0 to 50 ppm, for nitrates from 20 to 200 ppm, for sulphates from 200 to 1000 ppm, and for sodium from 50 to 1000 ppm.

Without a doubt, the major source of this variation stems from the fact that past research investigated adverse effects of each element individually, and without accounting for total dietary burden, whereas there are many dietary and environmental interactions that influence tolerance to water contaminants.

Moreover, older information may not be applicable to modern poultry strains, which have been highly selected for superior performance. Definitely, there is a lack of research data that would consider recent knowledge on water physiology, nutrition, and toxicology.

4.4 Water Use to Combat Heat Stress

Considerable research efforts with water have been centered around heat stress problems. Adding sodium chloride, potassium chloride, potassium sulphate or carbon dioxide to broiler drinking water has been shown to increase gain slightly and lower body temperature (Teeter, 1988). Most of this effect is probably attributable to the resulting increased water intake.

Cooling water to combat heat stress may be beneficial in some situations. Studies in broilers showed a benefit in daily gain from providing cool drinking water. However, work at the University of Florida showed that cooling hens' drinking water during hot daylight hours did not improve performance other than the shell and interior quality of eggs.

5. WATER REQUIREMENTS FOR RUMINANTS

5.1 Water Supply

All ruminant livestock require considerable amounts of water to produce at a high level. The water requirements of ruminant livestock are provided essentially from three sources: 1) drinking water, 2) water present in feed, and 3) metabolic water, which is formed by the oxidation of nutrients and body tissues.

It is important to remember that in order to perform at the maximum of their potential, highly producing animals need large amounts of good quality, clean, fresh water.

5.1.1 Effect of Feed on Water Intake

Dry matter content of the diet is one of the major factors affecting water intake. Diets high in salt, sodium bicarbonate, or protein appear to stimulate water intake (Holter and Urban, 1992; Murphy, 1992). Also, high-forage diets may increase water requirements (Dahlborn *et al.,* 1998). Holter and Urban (1992) reported that water intake decreased by 33 kg/d when diet DM decreased from 50 to 30%. Also, research by Stockdale and King (1983) demonstrated that cattle grazing pasture consumed only 38% of their daily water requirement.

Generally, as the feed moisture content decreases, the water intake increases in an almost linear fashion as demonstrated in Figure. 5.1

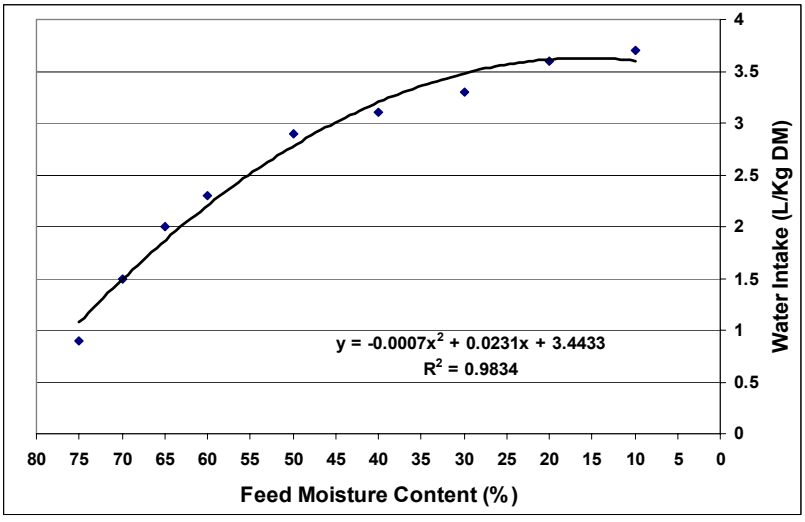

Figure 5.1. Correlation between level of moisture in the feed and water intake.

Graph was generated by the author based on information published by Hyder, *et al.,* 1968. J. Range Mgmt. 21:392.

5.1.2 Effect of Environmental Temperature on Water Intake

In addition to feed moisture level, another variable that will have a major impact on water intake is environmental temperature. Water metabolism is essential to the maintenance of body temperature. Ruminants such as sheep, goats and cattle dissipate internal and absorbed heat by evaporation of body water. Animals exposed to heat will require more water because a relatively large proportion of the body water pool may be lost via respiration from the lungs and as sweat.

At an environmental temperature that causes no heat stress, water intake tends to be about 3-5 units per unit of dry matter in adults. Environmental temperatures determine water requirements, and in general, the water intake is correlated with the environmental temperature over a wide range of values. Figure 5.2 illustrates the correlation between ambient temperature and water intake.

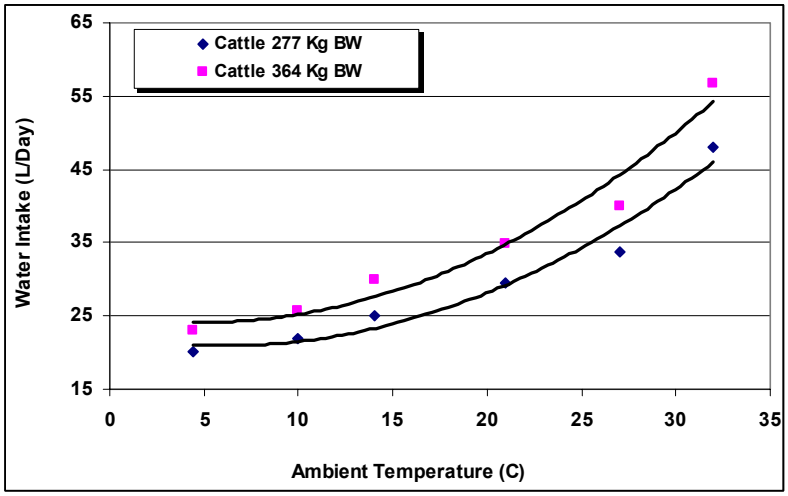

Figure 5.2 Examples of water intake changes as ambient temperature increases.

It is noteworthy that water requirements for animals with different body weights vary in magnitude, but generally the temperature dependent increments are remarkably similar.

Graph was generated by the author based on information published by NRC, 1994.

For practical purposes, the data compiled in Table 5.1 is frequently cited in the literature and can be used as a guide to estimate water intake in various classes of beef cattle at different environmental temperature. Dry matter intake has a major impact on water intake. Therefore, during winter, because heavier animals are assumed to be in better body condition, they may consume less dry matter and thus require less water. However, this table does not take into account the level of moisture in the ration.

Table 5.1 Water consumption rate in various classes of beef cattle with reference to environmental temperature.

	Water Consumption (Litres per Day at Different Temperature)					
Weight (kg)	4.4° C	10° C	14.4° C	21.1° C	26.6° C	32.2° C
Growing Cattle						
182	15.1	16.3	18.9	22.0	25.4	36.0
277	20.1	22.0	25.0	29.5	33.7	48.1
364	23.0	25.7	29.9	34.8	40.1	56.8
Finishing Cattle						
273	22.7	24.6	28.0	32.9	37.9	54.1
364	27.6	29.9	34.4	40.5	46.6	65.9
454	32.9	35.6	40.9	47.7	54.9	78.0
Wintering Pregnant Cows						
409	25.4	27.3	31.4	36.7		
500	28.7	24.6	28.0	32.9		
Lactating Cows						
409	43.1	47.7	54.9	64.0	67.8	81
Mature Bulls						
636	30.3	32.6	37.5	44.3	50.7	71.9
727	32.9	35.6	40.9	47.7	54.9	78.0

(Data Adopted from National Research Council,1974).

Environmental temperature also has an impact on water consumption in lactating cattle. The examples in Table 5.2 illustrate differences in water intake of dairy cattle at different milk production levels.

Table 5.2 Differences in water intake in dairy cows of similar weight, but differing in milk production.

Lactating Cows (600 kg) Milk Yield (kg/day)	Water Intake at Temp 10°C	Water Intake at Temp 32°C
15	59	89
30	92	146
45	124	203

As demonstrated above environmental temperature may substantially affect water intake, and this factor must be carefully considered and included in the overall evaluation of potential impact of water quality.

Ruminants

At lower temperature, when water intake is decreased, a total amount of ingested contaminants will be lower in comparison to higher temperature, when water intake is higher. Therefore, relatively higher concentration of water contaminants may be tolerated by animals at lower temperature than at higher temperature.

While considering the evaluation of potential adverse effects associated with water contaminants, it is very important to remember that the environmental temperature has a tremendous impact on water intake, and thus on intake of all contaminants present in this water.

5.1.3 Difference in Water Intake in Various Types of Ruminant Livestock

There is a shortage of published information on the water consumption for different classes of livestock under a variety of management and climatic conditions. It is important to note that water intake may vary drastically with the source of feed (feedlot vs pasture). Breeds of livestock, and sometimes strains within a given breed show significant differences in their water requirements. Young animals require more water than mature stock, whereas the requirements of pregnant or lactating animals are even greater. Table 5.3 provides an overview of approximate water requirements for a wide range of ruminant animals and production modes.

Table 5.3 Examples of water intake by various classes of ruminant livestock.

	Approximate Water Consumption Levels (Litres per Day)
Beef	26-66
Feeder calves	18-27
Steers	36-45
Dairy	28-110
Dairy (maintenance)	55-68
Dairy (lactating)	68-114
Calves (4-8 weeks)	4.5-6.8
Calves (12-20 weeks)	9.1-20
Calves (26 weeks)	17-27
Heifers (pregnant)	32-45
Lambs (weaned)	3.5-4.0
Ewes (dry)	4.0-5.0
Ewes (lactating)	4.0-12.0
Goats	3.0-15

There is no recently published data on specific water requirements of modern livestock. The issue is complicated further by the fact that many values cited are based on data from outdated research.

Attempts have been made to fit the water requirements into a mathematical model. A water equation for feedlot steers recommended by NRC based on work by Hicks *et al.,* (1988) is as follows:

Water intake (L/day) can be calculated using the following formula:

Water intake = -18.67 + (0.3937 x MT) + (2.432 x DMI) - (3.870 x PP) - (4.437 x DS)

Where: MT = maximum temperature (F);
DMI = dry matter intake (kg/d);
PP = precipitation (cm/day);
DS = dietary salt (%).

The estimation of water requirements for dairy cattle is more complex, because many more factors that affect the amount of water intake of dairy cows have been identified. Several equations considering different variables have been proposed to estimate water intake. The equation developed by Murphy *et al.,* (1983) takes into account, among other variables that have been shown to affect water intake, two very important variables, i.e. the water content of milk at a level that is biologically realistic and temperature.

Water intake = 15.99 + 1.58 x DMI (kg/d) + 0.90 x milk (kg/d) + 0.05 x Na intake (g/d) + 1.20 x min temp (^{o}C)

As discussed above, water intake may be affected by many factors, and the problem that water specialists frequently have is how to account for all specific requirements with accuracy under a variety of field situations. From a practical point of view, it is important to remember that, as the above-discussed physiological, dietary, and environmental variables will influence water intake, they also will have a major impact on the intake of water contaminants. All these variables must be considered and evaluated very carefully while assessing the impact of water contaminants on livestock.

5.2 Water Quality

The importance of water quality issues in ruminant livestock should be recognized in the context of specific metabolic features of ruminants. Because of differences in metabolic characteristics, some water contaminants may cause severe health and performance problems in ruminants, while the same contaminants may have only marginal (if any) effects on animals such as horses, pigs or poultry. For this reason, many aspects of

water quality for ruminants deserve special consideration. Specific issues arising from water contaminants in ruminants will be discussed in detail in the relevant sections.

6. WATER REQUIREMENTS FOR SWINE

6.1 Water Supply

The body water status of swine is under tight physiological control, and at a given body weight and fat content, the water content of a pig's body is remarkably constant. Therefore, the pig must have constant access to a water source in order to meet its daily requirements, as the amount of water excreted from the body must be essentially matched by water consumption. When water loss is not matched by water intake, body tissues may become depleted of water, and this may lead to dehydration.

The ingredients most commonly used in swine diets contain about 10 to 12% water (NRC, 1998), and so the amount of water supplied from this source is very limited. Thus, drinking water is by far the most important source of water for swine.

Determination of physiological water requirements in swine is a very challenging task. The estimates of water requirement based on measurements of water usage by pigs may give values that are usually grossly overestimated because wastage is generally not taken into account. Therefore, in determining water requirements, special attention must be exercised to differentiate between water consumption and water disappearance. Table 6.1 provides a summary of requirement estimates for the various classes of swine.

Table 6.1 Estimates of Water Requirements for Various Classes of Swine

Category	Estimated Water Requirements (litres per day)
Suckling pigs	**0.27 to 2**
Weanling pigs	**1 to 5**
Growing pigs	**5 to 10**
Finishing pigs	**5 to 12**
Gestating sows	**5 to 20**
Lactating sows	**15 to 35**
Boars	**8 to 17**

Values derived as cited by Thacker, 2001.

Major Factors Influencing Water Requirement in Swine: There are numerous physiological, nutritional, and environmental factors that may influence water requirement in swine (Patience *et al.,* 2005, Mroz *et al.,* 1995, Suzuki *et al.,*1998; Pfeiffer *et al.,* 1995). It is therefore difficult to provide universal estimates of requirements.

Swine

Water loss is one the most important variables that may alter water requirements. Water excretion is increased when pigs are fed diets that contain large amounts of minerals and protein.

A high level of protein in the diet may increase water loss, and thus increase the water requirement (Wahistrom *et al.,* 1970). Water loss also increases with an increased level of fiber intake (Cooper and Tyler, 1959). Increased intake of salt usually increases water intake, and a concomitant increase in urinary excretion.

Feedstuffs that have laxative properties also increase water intake. Water excretion via the feces is increased during diarrhoea (Thulin and Brumm, 1991).

Sweating and insensible water losses from the skin (e.g. through evaporation) are not major routes of water loss in swine, but water is continually lost via the respiratory tract during the normal process of breathing. Increased ambient temperature may lead to increased respiration and panting, and thus increased water loss.

Under limited feeding conditions, pigs tend to consume excessive and highly variable quantities of water (Yang *et al.,* 1981). Animals deprived of feed may show grossly excessive water intake, which is often referred to as hunger-induced polydypsia.

Factors influencing water intake must be taken into consideration while assessing the risk associated with water contaminants.

6.2 Water Quality for Swine

Various classes of water contaminants can occur in water at levels that can be potentially harmful to pigs. A survey of pig farms in SK (McLeese *et al.,* 1991) showed that concentrations of sulphate and total dissolved solids were above levels recommended in Canada for livestock in 25.0% and 7.4%, respectively, of the wells. Sodium and chloride were also high in many wells. According to the latter authors the incidence of minor to moderate scouring in weanlings, as reported by producers, was directly related to TDS, magnesium, calcium and sulphate.

Patience et al, (2004) concluded that weanling pigs can tolerate drinking water containing high concentrations of sulphates. Maenz *et al.,* (1994) who studied water containing 4,390 mg of TDS, 2,650 mg of SO_4, 947 mg of Na, 288 mg of Ca, 88 mg of Mg, 70 mg of Cl and 15 mg of K per litter on performance of weanling pigs found no evidence of impaired performance of weanling pigs offered high-sulphate drinking water, but the authors noted increased scouring associated with high-sulphate drinking water. It is of interest to note that TDS and SO_4 were well in excess of maximum levels recommended for livestock in the 2005 CCME Canadian Water Quality Guidelines.

Overall, the risk of health effects associated with common water contaminants appears to be very low. However water mineral contaminants may affect the physiological status of acid - base balance, and this may influence nutrient metabolism in pigs.

Of interest here is the possibility that water containing high levels of ionic components may alter the balance of dietary undetermined anion ((Patience and Wolynetz, 1990). This dietary undetermined anion is calculated as (Na + K + Ca +Mg) - (Cl + P + S inorganic). Notably, the ions comprising this equation are all major mineral contaminants commonly present in drinking water, and therefore may change the net acid or alkaline load contributed by the diet.

Water mineral contaminants may influence water pH, i.e. acidity or alkalinity, and pH can have a major impact on chemical reactions involved in the treatment of water, and depending water treatment system, high or low pH may significantly impair the efficiency of water treatment.

Water quality must be carefully assessed prior to administration of medication, as chemical incompatibility of water may cause precipitation or inactivation of medication delivered via the water system.

Water may contain a variety of microorganisms, including bacteria and viruses. Among bacterial contaminants, *Salmonella, Leptospira,* and *Escherichia coli* are the most commonly encountered (Fraser *et al.,* 1993). Bacterial contamination is usually more common in surface waters than in underground supplies such as deep wells and artesian water. Water can also carry pathogenic protozoa as well as eggs or cysts of various intestinal parasites.

CCME recommends only high quality water for ILO's. However, there are no clear guidelines for presence of microbes in livestock drinking water sources. At present, the suggested values are: for total bacteria <10,000/1000 mL and for total coliform <1/1000 mL. Some reports suggest that total coliforms need only be <5,000/1000 mL.

The best scenario would be that drinking water for swine is free of pathogens. Therefore, if there is a risk of microbial contamination, water disinfection is highly recommended. According to information from *Saskatchewan Pork,* presently most of the swine producers using surface water for animals disinfect water with chlorine, and some of those using groundwater also chlorinate.

Swine

7. WATER TREATMENT TECHNOLOGY

Water contaminants can be decreased considerably or even completely eliminated by a variety of treatment methods. Some methods are more effective than others, but for treating water for livestock consumption, economics are an important issue. The following sections critically review the most common methods used for water treatment.

Activated Carbon Filters: This method is based on passing water through a filter containing activated carbon granules. Contaminants attach to the granules and are removed. Chlorine, some organic compounds associated with coloration, odour and off-taste of water, mercury, some pesticides and volatile organic compounds can be removed by this method. The filters must be inspected and replaced frequently. Poor filter maintenance will decrease effectiveness, and may result in bacterial growth on the filter, causing potential contamination of the water with pathogens.

Air Stripping: This method of water treatment involves passing water down a tube while air is forced up through the tube. Contaminants are transferred from water to air and vented off. This method may be effective in removing hydrogen sulphide, some odours and tastes, and some volatile organic chemicals. Bacterial growth can be a potential problem.

Biological Filters: This method is effective at removing iron, arsenic, and organics. Manganese can be removed with a pre-treatment of a strong oxidant. A microbiological layer is used to filter and consume contaminants. Biological filters usually require infrequent backwashing, however, some are sensitive to variable flow rates and perform better with a constant flow rate.

Chlorination: This is one of the most common methods in water treatment for pathogen reduction in drinking water for livestock. Chlorination is much more effective if it follows a filtration system to remove large particles that can house bacteria. In particular, this is an effective and widely used method to kill many kinds of microorganisms in water. It also aids in removal of unwanted color, odour, or taste from water and will also remove hydrogen sulphide and dissolved iron and manganese, if followed by mechanical filtration. However, if the system is not properly operated, it can be potentially hazardous. In typical systems the chlorine content of the treated water should be closely monitored so it is not harmful to animals. High concentrations of chlorine released to the dairy water system may affect water intake and performance of cows. Chlorination of water containing high levels of organic contaminants may result in the formation of potentially toxic compounds.

Coagulation: This is being used in livestock operations to remove fine particles, iron, arsenic, manganese and organics. The removal of particles prior to chlorination makes disinfection much more effective and this is a standard treatment of surface water prior to chlorination. The coagulation chemicals such as aluminum sulphate (alum)

neutralize the charge on the particles and cause particles to coalesce into floc that can be removed by filtration or settling.

Ion (Cation or Anion) Exchange: This purification system is based on removal of ions by replacing one or more chemical ions with another. The most commonly used systems contain resin beads to trap ions Cation exchange is based on the principle that positively charged sodium (Na^+) ions attached to the resin are replaced (exchanged) with other positively charged ions such as Ca^{2+} Mg^{2+}, Mn^{2+}. Heavy metals will also be removed if they are present in an ionized state. Anion exchange systems remove negatively charged ions such as Cl, I, F, as well sulphates and nitrites/nitrates.

The most common application for cation exchange is in the water softening process where metals, that are the main contributors to water hardness (Ca^{2+} Mg^{2+}), are removed from water during treatment. However, the treated water will have elevated Na^+ concentrations. This may be a consideration in overall sodium status of animals.

Mechanical filters: This method is used to remove insoluble contaminants including some forms of oxidized iron and manganese, as well as sand and silt. Mechanical filters such as multi-media filters only remove particles greater than 10 microns therefore are ineffective on fine particles and micro-biological particles, unless preceded by coagulation chemicals.

Nano- Filtration: This technology uses membranes similar to reverse osmosis membranes, but because the pore size in the NF membrane is much larger, it takes less pressure to force the water through the membrane. Nano-filtration takes out about 90% of the dissolved solids and 95% of the hardness, therefore it is often referred to as the softening membrane. Water wasted is usually between 15% and 30% and is not as much of a concern as RO membranes. The added benefit is that the water is not nearly as corrosive as from RO membranes therefore chemicals rarely need to be added following treatment. Pre-treatment devices are usually needed.

Oxidizing filters: This method may help to remove some contaminants by chemical (oxidizing) reactions and then filtering by mechanical filtering. Contaminants typically removed include hydrogen sulphide, iron, and manganese. The common oxidants used are aeration, chlorine, potassium permanganate and ozone. Strength and type of oxidant varies based on the targeted dissolved ion to be removed.

Ozonation*:* This method of water treatment is based on application of ozone gas. Ozone is a very potent oxidizing agent, and destroys pathogenic microorganisms. The equipment typically is quite expensive. This method can also be used to remove color, off-taste, odours, hydrogen sulphide, soluble iron and manganese, but the water must be subsequently passed through a mechanical filtration system.

Reverse osmosis (RO): This technology is more and more applied in the treatment of water for livestock and horses, pigs, and poultry. Basically, water impurities are filtered

out through a system of membranes which have small pores that allow passage of water but not the contaminants. Depending on the system, more then 99% of contaminants can be removed by reverse osmosis, and the product of this process is highly purified water. Reverse osmosis has high initial costs, high membrane replacement cost, and needs consistent maintenance. Depending on the size of the system, the pressure, and the water quality, reverse osmosis systems waste between 50% and 90% of the water. The filtrate containing high concentration of contaminants must be disposed of in some manner.

Slow Sand Filters: This method is a type of biological filter that is simple and relatively inexpensive. It will remove fine particles and iron. It will also remove arsenic if iron is present and manganese with some pre-treatment. As with most biological filters, it is sensitive to variable flow rates. It can be used on both surface and groundwater but tends to perform better with groundwater.

Ultra-Filtration: This technology uses membranes with pores larger than nano-filtration therefore requires even lower pressure and wastes less than 10% of the water. Pressures common to municipal systems are often used. Particles less than 0.1 microns such as bacteria, viruses, oocysts, large organic particles, and colloidal substances such as fine soil particles. It does not reduce dissolved solids and therefore does not remove hardness. Ultra-filtration has been used to purify water for washing milk equipment and containers.

Ultraviolet radiation: This method uses a special light source that generates ultraviolet radiation. It is a very effective method of killing micro-organisms in water, including pathogens, but it may not work if the water is too cloudy, or if water is passing by the light source too fast. It may be difficult to assess the efficiency of UV or if it is working at all unless it is equipped with an intensity monitor. Water should be monitored for bacteria.

Water Softening: The high concentration of minerals associated with water hardness may result in malfunctioning of watering equipment, which may lead to water deprivation. Consequently, some producers attempt to remedy the problem by using water treatments known as “softening”. The process of water softening is based on exchange of hardness-causing ions such as calcium or magnesium, with sodium ions. This process may add a considerable amount of sodium ion to the water and therefore, for extremely hard water, there may be a risk of adverse effects associated with sodium overload.

7.2 Approximate Costs of Water Treatment

Table 7.1 summarizes the approximate costs of water treatment for a 100 and 500 cattle herd. Costs will vary according to the concentration of the contaminants, economic conditions and the level of controls and monitoring. The concentration of

contaminants is based on the Saskatchewan average for water that would require treatment.

Assumptions for the cost table are as follows:

- Heated building, electrical supply and water supply is existing
- Pressure system or variable frequency drive (VFD) pump and one-day storage system is existing (approximate costs for 100 cattle is $500 for pressure system and $1000 for a 1000 USgal tank; costs for 500 cattle is approximately $700 for the pressure system and $5,000 for a 5000 USgal tank)
- Basic controls with manual operation except for automated shutdowns for low water or treatment failure
- Consumption of 40 L/d per cow
- Daily treated water requirement supplied in 20 hours
- Amortized loan at 8% interest for capital expenditure
- Replacement of water filter media and membranes are included
- Water treatment chemical costs are included (coagulation, oxidation, disinfection)
- Wasted water disposal costs are not included (for backwashing filters, membrane concentrate disposal, etc)
- Labour for scheduled maintenance is included at $20/hr
- Labour for daily operational checking is not included

Table 7.1: Approximate Annual Treatment Costs (2008) for a 100 and 500 Cattle Operation

Treatment System	Contaminant Removed	Cost/animal/year (100 cattle)	Cost/animal/year (500 cattle)
Air Stripping	Hydrogen Sulphide, Methane	$2	$0.5
Chlorination	Bacteria, Oxidize metals	$2	$1.5
Multi-Media Filter	Large particles, Oxidize metals	$2	$1.5
Ultraviolet Radiation	Bacteria	$4	$2
Ion Exchange (softening)	Hardness, Iron < 2 mg/L	$6	$5
Slow Sand Filters	Iron, Arsenic	$7	$4
Oxidizing Filter	Iron, Arsenic, Manganese*	$10	$4
Activated Carbon Filters	Taste, Odour, Chlorine	$10	$6
Ozonation	Bacteria, Oxidize metals	$12	$6
Biological Filters	Iron, Arsenic, Organics, Manganese*	$19	$10
Coagulation	Particles, Iron, Arsenic, Manganese	$20	$20
Ultra-Filtration	Bacteria, Viruses, Soil Particles	$40	$18
Nano-Filters	TDS, Hardness, Arsenic, Sulphates, Manganese, Iron*	$45	$20
Reverse Osmosis (RO)	TDS, Sulphates, Hardness, Arsenic, Manganese, Iron*	$50	$20

***** Removal will require additional equipment and cost

8. WATER TREATMENT: POTENTIAL ADVERSE EFFECTS ON WATER CONSUMPTION, AND ANIMAL PERFORMANCE OR HEALTH

8.1 Water Softening

As mentioned earlier, a high concentration of minerals associated with water hardness may result in malfunctioning of watering equipment, which may lead to water deprivation. Consequently, some producers attempt to remedy the problem using water treatments known as "softening". Since the process of water softening adds the sodium ion to water, there is a risk of adverse effects associated with sodium overload. Roush and Mylet (1986) who studied the influence of softening on hens over a 308-day period recommended that the sodium of softened water should be monitored.

Dairy farmers in some parts of Canada believe that softening improves the palatability of water for cattle. Blosser and Soni (1957) compared the influence of hard (116.4 mg/L as $CaCO_3$) and soft (8.4 mg/L as $CaCO_3$) water on milk yield of dairy cattle. No significant difference was found between the two types of water. Graf and Holdaway (1952) also found no effects of hard water (290 mg/L as $CaCO_3$) on milk yield, change of body weight, water intake or ratio of water intake to milk yield as compared with soft water (0 mg/L as $CaCO_3$). Softening of hard water adds about 0.63 mg of sodium per mg of hardness (as $CaCO_3$) so 290 mg/L as $CaCO_3$ translates into 182 mg/L of sodium. MAFRI (2004) suggests that water that contains over 800 mg of Na/L can potentially result in diarrhoea and decreased milk production in dairy cows, and an excess amount of sodium may also require ration adjustments. This level of sodium in water appears to be very conservative as most literature does not mention sodium as an issue. Research on impact of TDS on dairy production also indicates that TDS concentrations less than 2000 mg/L likely have little impact, yet TDS is usually comprised of a high percentage of sodium (Bahman et al 1993).

More recently, Looper and Waldner (2002) suggested that the degree of hardness does not appear to affect animal health or productivity. A limit of 300 to 400 mg/L of magnesium is recommended for dairy cows (MAFRI 2004).

8.2 Water Chlorination

Disinfection of water for livestock is highly recommended if microbial contamination is a concern, and sodium hypochlorite is probably the most common product used for water sanitation. Based on personal experience (Olkowski, unpublished observations) sodium hypochlorite has a relatively high margin of tolerance. Even considerable overdosing can be well tolerated by poultry over a short period, with minimal or no effects on production. Accidental application of 50 ppm (i.e.10 fold recommended dose) resulted in slight transient decline in water consumption. However, long term exposure to high levels of sodium hypochlorite in water should be avoided.

The possibility of adverse effects of chlorinated water on medication administered via water must be considered. Potential problems that may arise from water disinfection must be carefully assessed while planning delivery of medication via water (for review see Vermeulen *et al.,* 2002).

Administration of medication in water treated with a disinfecting agent may alter drug solubility or even result in precipitation. In some cases, water disinfectants may affect pharmacological potency of the medication, or even complete inactivation of drugs may occur.

Excessive water chlorination many be required under some practical situations, but it is important to remember that excess chlorine may have different impacts depending on class of animals. For instance, high levels of chlorine in water may affect the efficiency of the rumen microbial population, therefore in ruminant livestock metabolic impairment of rumen function may occur. On the other hand, monogastric livestock will likely be less affected by direct effects of chlorine, and most affected by pathogens in drinking water, so risk-benefit analysis would suggest that more aggressive water disinfection may be beneficial in this class of farm animals in situations where risk of bacterial contamination is high. However, more research is needed to determine appropriate levels of chlorine for different types of livestock.

Although direct adverse effects associated with disinfection chemicals based on sodium hypochlorite are very unlikely, application of these products in water containing organic matter may lead to synthesis of disinfection by-products, which can be toxic.

8.2.1 Potential Problems Associated with Water Chlorination: Emerging Issues

Undoubtedly, of the disinfection procedures used in Canada, the most common method of water treatment for livestock is chlorination. In this context, the emerging issues of potential adverse effects associated with the production of chlorinated contaminants generated as a result of disinfecting drinking water need to be addressed as a water quality issues.

Several compounds, known as disinfection by-products (DBPs), are formed through the interaction of chlorine molecules with naturally occurring residual organic compounds, such as humic and fulvic acids, that are ubiquitous in most water sources. Residual organic matter is present in many livestock water sources, and, in particular, in surface waters. Following chlorination, the generated DBPs may be a source of contaminants that pose risks to both human and animal health.

The health hazard associated with DBP in humans has been recognized for some time (Health Canada. 1995, WHO, 1996), yet these issues have not been adequately addressed in the context of water quality for livestock.

There are three main classes of DBPs in drinking water that represent potential risks to livestock: (1) chlorophenols, (2) trihalomethanes (THMs), and (3) haloacetic acids (HAAs). Chlorophenols occur in drinking water as a result of the chlorination of phenols.

Wide range of adverse effects has been associated with generation of DBPs. Several phenolic DBPs produced during chlorination have been shown to cause lymphomas, leukemia, and hepatic tumors in rats. THMs have been closely linked to an increased incidence of bladder cancer and possible increases in rectal and colon cancer in humans (Mills *et al.,* 1999). Carcinogens are usually not an issue for livestock as their productive life is short, therefore cancer is infrequent.

Although the carcinogenic characteristics of DBP could potentially present a health hazard in livestock used for breeding and milk production (longer life span) more so than animals used for meat (short life span), the practical aspect of such problems would be rather negligible. On the other hand, chronic adverse effects that may be of significance from an animal production standpoint stem from adverse effects of DBPs on reproductive parameters. It has been shown that dichloroacetic acid causes alterations in spermiation, sperm morphology, and sperm motility (Linder *et al.,* 1997). According to Veeramachaneni (2000), DBPs can be associated with deteriorating trends observed in male reproduction.

There is a possibility that some reproductive problems in farm animals may be associated with adverse effects of disinfection by-products.

The potential impact of DBPs on reproductive performance of farm animals should not be underestimated. In many situations, water commonly used for livestock from surface sources such as dugouts, sloughs, lakes, and streams usually has a high content of organic matter, and also water from such sources is frequently contaminated with bacteria. It is a common practice that disinfection procedures are applied more aggressively to kill bacteria in surface water sources, but undoubtedly, at the same time there is high risk of DBPs formation.

Given the fact that DPBs have the potential to affect reproduction in laboratory animals, they can also have an adverse effect on reproductive performance of farm animals. It is not uncommon, in many practical situations, that the producers face a decline in fertility that is difficult to explain. The possibility that DPBs may be associated with poor fertility deserves thorough attention.

9. FACTORS AND CONTAMINANTS ESSENTIAL TO WATER QUALITY ISSUES AND CONSIDERATIONS FOR MANAGING THEIR DETRIMENTAL EFFECTS

9.1 Alkalinity, pH and Hardness

Alkalinity is a term frequently used to describe water quality. Total alkalinity is the sum of the concentrations of alkali metals, which are primarily sodium and potassium, but may also include lithium, rubidium, cesium, and francium. Sodium and potassium are most common in Canadian water sources.

These metals, upon reaction with water, form hydroxides that are alkaline, and as such they tend to increase the pH of water. In order to offset the alkaline pH, acidic ions are required. The total alkalinity of water is always less than its TDS, or salinity, since TDS and salinity include the sum of the concentrations of all substances dissolved in water, and total alkalinity includes only the sum of the concentrations of alkali metals.

Table 9.1.1 Alkalinity Levels in Saskatchewan Groundwater

Alkalinity Content (mg/L)	Number of Samples Analysed	Percent of Total
<200	95	3.3
200 to 500	2169	75.0
500 to 1000	610	21.1
>1000	19	0.7

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Water pH is a measure of concentration of hydrogen ions. The values are expressed in pH units ranging from 1 to 14. A pH of 7 is neutral, values less than 7 indicate acidic pH, whereas values above 7 indicate alkaline pH.

Little is known about the specific pH's effect on water intake, animal health and production, or the microbial environment in the rumen. The preferred pH of drinking water for dairy animals is 6.0 to 8.0. Waters with a pH outside of the preferred range may cause nonspecific effects related to digestive upset, diarrhea, poor feed conversion and reduced water and feed intake.

The pH of water may impact animal health in some animals more than in others. For instance, in ruminants, consumption of water with a pH below 5.5 may contribute to metabolic acidosis, whereas alkaline water with pH greater than 8.5 may result in higher risk of metabolic alkalosis. In dairy cattle, these conditions have been associated with reduced milk yield and milk fat, low daily gains, increased susceptibility to infectious, metabolic disorders, and reduced fertility.

Water hardness is another term frequently found on water analysis results. It indicates the tendency of water to precipitate soap or to form a scale on heated surfaces. Hardness is generally expressed as the sum of calcium and magnesium reported in equivalent amounts of calcium carbonate. Other substances, such as strontium, iron, zinc, and manganese, also contribute to hardness. See Section 8.1 on Softening for more information on effects of hardness and softened water on livestock.

Alkalinity, Salinity and TDS should not be confused with hardness. Highly saline waters may contain low levels of the minerals responsible for hardness. Although there are no guidelines, water with hardness greater than 500 mg/L (as calcium carbonate) is considered very poor quality for water distribution systems and will be prone to scaling. In Saskatchewan, more than 50 percent of the water has a hardness level greater than 500 mg/L (as calcium carbonate). For applications where water is heated and/or used for cleaning milk tanks, hardness should be less than 200 mg/L (as calcium carbonate).

Table 9.1.2 Hardness Levels in Saskatchewan Groundwater

Hardness Content (mg/L as $CaCO_3$ equivalents)	Number of Samples Analysed	Percent of Total
<100	239	8.3
100 to 200	126	4.4
200 to 500	1003	34.7
500 to 1000	953	32.9
1000 to 1500	343	11.9
1500 to 2000	137	4.7
>2000	92	3.2

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Treatment technology for hardness and pH adjustment is relatively inexpensive. Hardness is removed by a softener (ion exchange) and pH is adjusted by adding either acid or caustic soda to decrease or increase pH respectively. See Section on water treatment for further discussion on specific treatment systems.

9.2 Arsenic

Arsenic is widely distributed in the biosphere and earth's crust and can be a major source of contamination for livestock drinking ground water. Most arsenic-based products are discontinued, therefore, biosphere poisoning is often the result of discarded containers or industrial pollution. The principal sources of arsenic in ambient air are the burning of fossil fuels (especially coal), smelting, and waste incineration. Arsenic is introduced into water through the erosion and weathering of soil, minerals, and ores, from industrial effluents, and via atmospheric deposition (Hindmarsh and McCurdy, 1986; Hutton and Symon, 1986).

The potential sources of arsenic for farm animals are food, drinking water, soil, and air. According to the estimates of Environment Canada and Health Canada, in a typical situation, the significance of exposure source, in terms of contributing to arsenic intake, can be ranked in the following order of importance: food, drinking water, soil, and air.

The initial, 1987 CCME guideline for arsenic in water was set at a relatively high level of 500 µg/L, but this recommendation was with a provision that arsenic content in feed was low. The 1987 CCME guideline was changed to 71 µg/L in 1993, and more recently an interim guideline of 25 µg/L was adopted. It should be noted that the reasoning for this guideline for arsenic is largely based on an outdated research using beagle dog (Byron et al., 1967), which is a rather unrealistic model for derivation water quality standards for livestock. The value of 25 µg/L was established by applying a safety factor of 10, and to account for arsenic contribution from diet, an apportionment factor of 0.2 was also applied (CCME 1999).

While assessing the risk associated with arsenic in drinking water for farm animals, total intake of arsenic from dietary sources should be taken into consideration (Table 9.2.1).

9.2.1 Evaluation of Risk

Chemical forms of arsenic include arsenite (trivalent) and arsenate (pentavalent) with arsenite salts being 5 to 10 times more toxic than arsenic. The concerns related to arsenic are the carcinogenic properties to humans, at low level exposure.

The carcinogenic properties are generally not a major issue for livestock used for meat, as their lifespan is short. However, bioaccumulation of arsenic in livestock used for meat may be a concern from the perspective of meat quality. The bioaccumulation occurs mainly in the internal organs of animals consuming a diet high in arsenic

According to the most recent Health Canada guidelines, the concentration for arsenic in drinking water for humans is set at10 µg/L, which is more in line with the World Health Organization recommendation. The Health Canada guidelines are set for human consumption, where the overall risk associated with the ingestion of arsenic in drinking water is calculated based on lifetime exposure to arsenic, which results in more than one cancer endpoint in different individuals. In comparison to the livestock guideline, the Health Canada guideline for humans provides a substantial factor of safety. Such

safety assessment is not likely to be practical or applicable to farm animals under common farm practices.

Table 9.2.1 Examples of dietary intake of arsenic associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Arsenic‡**		
Water As content (µg/L)	**Estimated Water Contribution to Total Dietary Arsenic Intake (mg/day)**	**Estimated Contribution of Arsenic Allowed From Normal Feed (mg/day)**	**Estimated Dietary Arsenic Levels Generally Regarded as Safe and Dietary Levels Consideration for Risk of Adverse or Toxic Effect (mg/day)**	
25† (500)[A]	**0.8 to 1.0 (16 to 20)[A]**	**48.4 - 61.6***	**Acceptable Levels** *(generally regarded as safe)*	**<61.6***
			Excessive Levels *(possible risk of adverse metabolic effects)*	**330-420****
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>420**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day.
‡ Feed Intake estimates taken from the CowBytes® ration balancing program. Values for feed are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.
† Guidelines for water are based on CCME 2005 recommendation.
[A] 1987 CCME Guideline may be appropriate for livestock if levels of arsenic in feed are low
* Calculation based on values used by CFIA as "Metal Reporting Limits" for arsenic 4.4 ppm (information provided by Feed Specialist Inspector, CFIA, author's personal communication).
** Calculation based on tolerance level values from NRC - Mineral Tolerance of Animals 2005, 2nd Revised Ed, Committee on Minerals and Toxic Substances in diets and Water for Animals, The National Academies Press, Washington, DC..

Arsenic in some forms has a high inherent potential to cause toxicity, but because it is present in water at very low levels, the risk of adverse health effects in farm animals is generally very low. If one excludes accidental poisoning and industrial pollution, the risk of health hazard to livestock associated with arsenic in drinking water *per se* can be considered as extremely low.

Although the bulk of arsenic burden in livestock comes from feed, water contribution should not be ignored, and the exposure assessment should include total intake from both water and feed sources. In particular, in areas near a natural geological source or a source of anthropogenic contamination, drinking water has been calculated to be the most important contributor to overall exposure.

Health Effects: Symptoms of acute arsenic intoxication associated with the ingestion of well water containing arsenic at 1.2 and 21.0 mg/L have been reported (Feinglass, 1973; Wagner et al.,1979). Acute toxicity signs may include abdominal pain, depression, salivation, or diarrhoea. Long term, low level exposure may cause chronic toxicity, with characteristic signs including skin pigmentation and development of keratoses, peripheral neuropathy, skin cancer, peripheral vascular disease, hypertensive heart disease, and cancers of internal organs. Early signs include neurological disorders, such as in-coordination, swaying and ataxia ('drunken hog syndrome'), but affected animals remain alert and continue to eat and drink. The clinical manifestation of arsenic poisoning depends on the specific characteristics of arsenic exposure such as form, pattern, and source (for more details see Puls, 1994).

Production Effects: The risk of a direct effect of arsenic in water on production parameters in practical situations is low, if any. However, because arsenic interacts with selenium at a very specific molecular level which may lead to depletion of selenium, some subtle signs associated with arsenic overload may be essentially the same as those associated with selenium deficiency (more detail will be provided in ensuing section on metabolic interactions).

Of note, although the risk of a direct effect of arsenic in water on health or production parameters in practical situations is negligible, the issue of arsenic intake may be relevant to contamination of animal products.

Because arsenic is classified in Group I (carcinogenic to humans), the importance of arsenic as a water quality parameter may be an issue for meat quality, due to the potential for accumulation in some edible tissues. The data from CFIA (*the Report On Pesticides, Agricultural Chemicals, Veterinary Drugs, Environmental Pollutants and Other Impurities in Agri-Food Commodities of Animal Origin)* indicate that heavy metals have been detected in some samples of Canadian meat from all kinds of livestock, albeit (as CFIA stated) at levels that are not considered violations of the ACT. Notably, arsenic is the most likely metal to be detected in meat, followed by cadmium and lead, in that order.

There is insufficient recent scientific data on the issues of heavy metal in Canadian animal products, but studies from other countries have shown that farm animals can accumulate toxic metals at levels that may be of concern for the consumer (Lopez *et al.,* 2002, Wilkinson *et al.,* 2003).

Metabolic Interactions: Arsenic is considered to have antagonistic effects on I, Se, Cu, Hg and Pb. High dietary arsenic can exacerbate copper deficiency (Uthus, 2001),

but the most likely metabolic effects of practical significance associated with excessive intake of arsenic are those resulting from its interactions with selenium. Consumption of water containing elevated arsenic concentrations over a long time, may lead to adverse metabolic effects associated with specific interference of arsenic with selenium homeostasis. Arsenic-selenium interactions result in the formation of glutathione-arsenic-selenium complexes that are excreted via bile (Gailer *et al.,* 2002). Because of the possibility of continued depletion of body selenium, caused by biliary excretion of arsenic-selenium complexes, there is an increased risk of selenium deficiency in livestock that are chronically exposed to even low levels of arsenic. Such adverse effects of arsenic would be of particular concern when dietary levels of selenium are only marginally sufficient.

Close monitoring of selenium status should be considered in areas where low level, long term, exposure of livestock to arsenic is widespread. In the management of risk associated with water arsenic, the nutritional status of selenium should be routinely taken into consideration, particularly, since the effects of low level, long term, exposure on production parameters in livestock are not known.

Table 9.2.2 Summary of practical information relevant to arsenic exposure in livestock.

Guidelines	**Interactions**			**Adverse Effects and Signs of Toxicity**	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Acute Toxicity** (short term, high level exposure)	**Chronic Toxicity** (long term, low level exposure)
25 µg/L	Copper Iodine Selenium	Mercury Lead	Arsenic increases excretion of selenium which may lead to selenium deficiency. In highly producing animals, production parameters can be adversely affected without overt signs of toxicity.	abdominal pain, depression, salivation, diarrhea Note: In practical situations, acute toxicity in livestock associated with arsenic in drinking water is unlikely to occur.	Increased skin pigmentation, keratoses, skin cancer, peripheral neuropathy, peripheral vascular disease, hypertensive heart disease, cancers of internal organs can occur, but this is not a very likely scenario under practical situations. Subclinical signs of chronic exposure to arsenic may be manifested as subtle signs of selenium deficiency.

† CCME 2005. The threshold toxic dose in domestic ruminants appears to be between 1 – 2 mg/kg BW, but production parameters may be affected at lower levels of exposure.

9.2.2 Water Types or Conditions Where High Levels Occur

Arsenic levels in surface water are usually low unless there has been industrial contamination. In ground water, arsenic levels in water are determined primarily by the geological formations. There are seams of high arsenic levels in Saskatchewan. Arsenic levels ranged from 0.5 to 105.0 µg/L in municipal treated water supplies in 539 Saskatchewan communities between 1976 and 2002, with concentrations in 97% of samples being less than or equal to 10 µg/L, and the average 3.0 µg/L. According to the Saskatchewan Watershed Authority Rural Water Quality Data Base for 2966 samples, in Saskatchewan, arsenic levels were below 10 µg/L in 85% of the samples. The table below summarizes the frequency of other levels. The maximum level recorded in Saskatchewan was 210 µg/L.

Table 9.2.3 Arsenic Levels in Saskatchewan Groundwater

Arsenic Content (µg/L)	Number of Samples Analysed	Percent of Total
<10	2525	85.3
10 to 25	295	10.0
25 to 50	106	3.6
50 to 100	29	1.0
100 to 200	3	0.1
>200	1	0.03

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Canadian water sources outside of Saskatchewan also contain elevated levels of arsenic. In Nova Scotia, 9% of well water samples tested for arsenic at the Environmental Chemistry Laboratory in Halifax between 1991 and 1997 exceeded 25 µg/L. According to Méranger *et al.,* (1984), in some areas of Nova Scotia, arsenic levels exceeded 50 µg/L in 33–93% of wells sampled, with concentrations being higher than 500 µg/L in 10% of the wells sampled. In Newfoundland, arsenic levels ranged from 6 to 288 µg/L in public water supplies (54 wells) surveyed in 2002. In British Columbia, a maximum arsenic concentration of 580 µg/L was reported in groundwater samples taken on Bowen Island (information compiled from *Technical Document Prepared by the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment Health Canada, Ottawa, Ontario. May, 2006*).

9.2.3 Management Considerations

The natural antagonism between arsenic and selenium can be used in management strategies for problems associated with excess of both arsenic and selenium.

In the management of risk associated with arsenic, the nutritional status of selenium should be routinely taken into consideration, as secondary selenium inadequacy may have a significant impact on production parameters in all classes of livestock.

In the areas where water arsenic levels are moderately high, proper balancing of the dietary selenium to fulfill metabolic requirements may be sufficient to alleviate the adverse effects of arsenic (Biswas *et al.,* 1999).

9.2.4 Treatment Technology

Treatment technology includes:

- Coagulation (also removes iron)
- Manganese greensand (also removes iron and manganese)
- Slow sand filter (if iron is present)
- Biologically activated carbon with pre-oxidation (also removes iron and manganese)
- Oxidation/pH modification and filtration (also removes iron and manganese)
- Absorption on activated alumina (only arsenic)
- Nano-Filtration or RO membranes (if TDS is high)

Treatment used to remove only arsenic from water for livestock is rarely economical. Often iron or manganese exists in water with high arsenic content, and removal of both substances with one treatment system may provide economic benefit. See Section on water treatment for further discussion on specific treatment systems.

9.3 Calcium

Calcium is an essential nutrient, but if its intake grossly exceeds metabolic requirements, potential risk of adverse effects ought to be taken into consideration. Calcium is routinely supplemented in the diet at a level between 0.5 to 1%, depending on species and production objectives. In some situations water may be a major contributor to total dietary calcium.

The CCME guideline of 1,000 mg/L is commonly cited as safe. Indeed, at this level, calcium in the water for livestock is not likely to present a toxicological problem, but when calcium from water and dietary sources is considered, cumulative daily intake may be excessive, or in some situations, toxic.

In this context, without considering the total burden of dietary calcium, a general recommendation of "safe" calcium levels in water may be of limited practical value. Calcium in water is rarely, if at all, taken into consideration when dietary requirements are calculated. Yet as demonstrated in Table 9.3.1, in some situations calcium in water, even at recommended levels, may be a concern, when cumulative feed calcium levels are high.

Table 9.3.1 Examples of dietary intake of calcium associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Calcium‡**		
Water Ca content (mg/L)	**Estimated Water Contribution to Total Dietary Calcium Intake (g/day)**	**Estimated Contribution of Calcium From Normal Feed (g/day)**	**Estimated Dietary Calcium Levels Generally Regarded as Safe and Dietary Calcium Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
1000	**32 to 40**	**85 to 110**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**29 – 144**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**145 – 201**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>201**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of calcium in feed.
† Guidelines for water are based on CCME 2005 recommendation.
‡Values for dietary levels are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.

Calcium

Under the majority of practical situations, livestock should tolerate concentrations of calcium in water up to 1000 mg/L, if calcium is the dominant cation and dietary phosphorus levels are adequate. However, in the presence of high concentrations of magnesium and sodium, or if calcium is added to feed as a dietary supplement, the level of calcium tolerable in drinking water may be less.

Therefore, the potential adverse effects associated with high levels of Ca in the water must be considered together with the overall dietary Ca. Furthermore, even though the risk of calcium toxicity *per se* may be relatively low, adverse effects of high levels of calcium in the water must be considered in the context of its complex anti-nutritional effects.

9.3.1 Evaluation of Risk

Calcium in water for livestock is not likely to result in outright toxicity, but if dietary calcium levels are already high, contribution of water calcium may become significant. Notably, even a moderately excessive, cumulative intake of calcium from drinking water and diet, may lead to metabolic disturbances.

Health Effects: The most likely health effects may be associated with skeletal disorders. Prolonged intake of excessive levels of Ca may cause osteopetrosis, vertebral ankylosis and degenerative osteoarthritis. However, under some circumstances, calcium can be deposited in skeletal muscles as well as in the heart muscle. Cardiac function can be compromised, or in more extreme and advanced cases, heart failure can be a result.

Production Effects: From a nutritional stand point, high dietary Ca may reduce nutrient uptake, and in particular, may affect fat digestibility. Even at moderately high levels, water Ca must be considered in the context of homeostasis of several other essential metals. Excess dietary Ca can cause reduced absorption primarily of phosphorus and zinc, but it may also affect magnesium, iron, iodine, manganese, and copper. This can lead to secondary deficiency of these elements, particularly when the dietary level of these elements is already low or only marginally adequate. In the case of copper, the bio-availability of this element may be further compromised by other dietary factors such as sulphur and molybdenum (for details see sections on sulphur and molybdenum).

Under a practical field situation, performance of animals exposed to excess dietary calcium can be affected, not as much by direct effects of calcium on the host's metabolism, but rather through secondary metabolic interactions with other nutrients.

There is a general consensus that high dietary calcium can reduce feed intake and adversely affect digestibility of nutrients practically in all classes of farm animals, but there are major variations among species with regard to tolerance levels (Alfaro *et al.,* 1988; Ammerman *et al.,* 1963; Zimmerman *et al.,* 1963; Combs *et al.,* 1966; Clark *et al.,* 1989; Fungauf *et al.,* 1961).

In these terms, the generalized effects of excess dietary calcium, such as lowered feed intake and reduced digestibility, may affect production parameters in all classes of farm animals. However, highly producing animals may be at higher risk of exposure, solely associated with water calcium, simply because the water intake increases proportionally with increased production. Moreover, highly producing animals are more susceptible to metabolic disorders.

In the context of the CCME guideline of 1,000 mg/L, water calcium alone may readily increase the total burden of dietary intake to levels that may cause serious metabolic consequences, as can be illustrated using the following examples.

For instance, in highly producing dairy cows, excess calcium may be among the predisposing factors of milk fever. Excessive dietary Ca (>100 g/day) or P (>80 g P) inhibits production of parathyroid hormone and the 1,25 dihydroxy cholecalciferol activation necessary to liberate Ca stores from bones. As discussed in the section on water intake physiology, a dairy cow producing 30 kg milk per day will drink, depending on environmental temperature, between 92 and 146 L of water per day. If the water would contain 1,000 mg/L, water contribution to the Ca intake would be 92 to 146 g/day.

A similar problem can be extrapolated to beef cows. For instance, if the same generic animal used as an example in Table 9.3.1 for calculations of total intake of calcium was a lactating cow, her water intake (depending on environmental temperature) would be approximately 64 to 80 litres per day, and therefore calcium intake with water alone would amount to 64 to 80 g per day. If we would apply the same criteria as presented in Table 9.3.1 for estimated contribution of calcium from feed, considering risk of adverse or toxic effects, it is evident that, even under a well balanced ration of calcium, this animal could be categorized as being at high risk of adverse metabolic effects, and bordering on low risk of health problems associated with high levels of calcium in water.

In essence, the examples discussed above underline several important issues with regard to setting water quality guidelines for livestock: 1) water calcium alone can increase total dietary burden to levels that may cause metabolic disturbances even under a balanced calcium diet, 2) water guidelines must include provisions to accommodate feed calcium contribution, so the total dietary burden of calcium does not exceed tolerance levels, and 3) total dietary (water and feed) tolerance levels should be considered in the context of metabolic and nutritional interaction of calcium with other essential nutrients, and the levels of these nutrients should be adjusted accordingly to account for possible adverse interactions.

Table 9.3.2 Summary of practical information relevant to calcium exposure in livestock.

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Nutrients**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
1000 mg/L	magnesium, iron, iodine, manganese, copper, zinc Vit D	lead cadmium	Excess Ca reduces the absorption of F, Mg, Mn, P, Zn, Pb, Cd, Fe, Cu, I. Metabolic problems can occur if dietary levels of essential metals such as Cu, Zn, Mn, or Mg are marginally sufficient. High dietary Ca may reduce nutrient digestibility. Excess Vit D may increase uptake and release of Ca from bone, and thus amplify detrimental effects Ca. Excess dietary Ca (>100 g/day) or P (>80 g P) inhibits production of parathyroid hormone and the activation of 1,25 dihydroxy cholecalciferol necessary to liberate Ca stores from bones.	Calcium in the water for livestock is not likely to present a toxicological problem.	Prolonged intake of excessive levels of Ca may cause osteopetrosis, vertebral ankylosis and degenerative osteoarthritis. Excess dietary calcium may be among the predisposing factors of milk fever.

†The CCME guideline of 1,000 mg/L is commonly cited, but without considering total burden of dietary calcium, this recommendation is of limited value.

It is important to understand that under practical field conditions, metabolic problems not necessarily specific *per se* to calcium toxicity, may occur. For instance, if dietary levels of essential metals such as Cu, Zn, Mn, or Mg are deficient or marginally sufficient, calcium excess may induce signs that are more specific to deficiency of the particular element of which the metabolism is affected by an excess of calcium. On the other hand, the apparent detrimental effects of calcium may be substantially amplified if the diet contains excessive levels of vitamin D.

Metabolic Interactions: High levels of dietary Ca reduced the absorption of several essential nutrient including F, Mg, Mn, P, Zn, Fe, Cu, and I. Thus, excessive intake of Ca may precipitate secondary deficiency of these elements. In particular, in practical situations, metabolic problems can occur readily when dietary levels of essential metals such as Cu, Zn, Mn, or Mg are deficient or marginally sufficient.

Calcium homeostasis, even at moderately excessive levels, can be compromised by unbalanced dietary phosphorus, and by excessive supplementation of Vitamin D. Calcium deposition in skeletal and cardiac muscle has been observed in animals fed high Vitamin D diets. It should be noted that vitamin D in animal diets is frequently supplemented in doses several fold higher than NRC recommendations for a variety of perceived health or production reasons.

9.3.2 Water Types or Conditions Where High Levels Occur

Calcium is an abundant natural element and the calcium concentration in water is primarily determined by the geological formations. Saskatchewan does not have limestone deposits therefore the calcium in groundwater is generally not excessive. As calcium is one of the main contributor to hardness, water with high hardness has high levels of calcium. To convert calcium concentration to hardness (as $CaCO_3$), the calcium concentration must be multiplied by 2.5. Therefore, for a water with calcium levels of 1000 mg/L, the hardness must be at least 2500 mg/L.

Table 9.3.3 Calcium Levels in Saskatchewan Groundwater

Calcium Content (mg/L)	Number of Samples Analysed	Percent of Total
<250	2502	86.5
250 to 500	367	12.7
500 to 1000	25	0.9
>1000	0	0.0

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.3.3 Management Considerations

In the assessment of the potential risk of adverse effects associated with calcium in water one should take into consideration at least three dietary variables: 1) balance of

phosphorus levels, 2) factors that may increase bio-availability of calcium (e.g. Vit D), and 3) antagonistic effects of calcium towards other divalent essential metals.

Considering the wide array of metabolic interactions, dietary levels of essential metals and phosphorus must be balanced to prevent Ca induced deficiency.

9.3.4 Treatment Technology

Treatment technology includes:

- Water softening technology
  - May effectively remove calcium but will elevate levels of sodium, which may be detrimental if sodium is excessive
- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.4 Chloride

Chloride ion is the most common form of chlorine in water. Chlorine can be present in the water in various chemical forms either naturally, or by being added during water treatment. Naturally occurring chloride ions occurs most commonly in association with sodium, and the content of both chloride and sodium must be considered while evaluating water quality.

CCME sets an aesthetic objective of <250 mg/L for chloride in drinking water. According to Puls (1994), the maximum tolerated drinking water level of chloride is 1,000 mg/L.

Table 9.4.1 Examples of dietary intake of chloride associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Chloride**		
Water Cl content (mg/L)	**Estimated Water Contribution to Total Dietary Chloride Intake (g/day)**	**Estimated Contribution‡ of Chloride From Normal Feed (g/day)**	**Estimated Dietary Chloride Levels Generally Regarded as Safe and Dietary Chloride Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
1000	**32 to 40**	**33 to 110**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**NA**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**NA**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**NA**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
† Pulse (1994)
‡ Natural Toxicants in Feeds Forages & Poisonous Plants 2nd ED, 1998, P.R. Cheeke, Interstate Publishers Inc.
NA=data not available

9.4.1 Evaluation of Risk

It has to be stressed that the estimates of adverse effects associated with chloride in water *per se* are somewhat conjectural, because chloride in water under normal circumstances is always associated with positive ions, most likely sodium. Water chlorination is one of the most often used methods of water treatment for farm animals. Chlorine used for water disinfection can react with organic matter in water and form disinfection by-products which may be harmful (for more information see Section 7.2 Water Chlorination). In typical systems the chlorine content of the treated water should

be closely monitored, so it is not harmful to animals and that the chlorine level does not cause livestock to reduce water intake.

Health Effects: Most animals can tolerate relatively large amounts of chloride. Under normal physiological conditions, the body has very effective mechanisms to control chloride levels, and from a water quality perspective, under most practical situations, the toxicity of chloride is generally low or negligible. Since chloride ion in water is most likely associated with sodium ion, adverse effects must be considered from both chloride and sodium. Sodium chloride (NaCl) at a 10,000 ppm in drinking water can cause toxicity, whereas 7,000 ppm NaCl in water can affect herd health and performance. For more detail see chapter on Sodium.

Production Effects: At concentrations above 250 mg/L chloride may reduce water palatability, which may result in lowered water intake. Since the chloride ion is an important component of acid-base homeostasis, excessive intake of chloride for a prolonged period of time may disturb the normal acid-base balance. Although the risk associated with the chloride ion in water to animal health would be very low (if any), disturbance of the acid-base balance in highly producing animals may lead to metabolic consequences affecting performance.

Metabolic Interactions: The adverse effects of chloride in drinking water cannot be considered on a stand-alone basis. The chloride ion is one of the ionic components contributing to salinity (see chapter on salinity). Therefore, the most likely scenario to consider would be combined effects of ions such as sodium, chloride, and sulphate. For instance, the study of Sanchez *et al.,* (1994) indicated that high intakes of chloride and sulphate affect milk production during summer months. Another study compared water dissolved solids from sodium chloride at 196 mg/L and 2,500 mg/L. Lactating cows consuming water with a high salt content increased water intake by 7 percent and exhibited a tendency for less milk yield compared to cows consuming low-saline water (Jaster *et al.,* 1978). In the study of Salomon *et al.,* (1995) saline water where chloride was a major component (580 mg/L) negatively affected milk production, and improvement of water quality by desalination increased production of milk and milk constituents.

Table 9.4.2 Summary of practical information relevant to chloride exposure in livestock.

Guidelines	Interactions		Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Ionic components commonly present in water**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
At present, there are no established guidelines for maximum concentrations for chloride in livestock drinking water. CCME sets an aesthetic objective of <250 mg/L for chloride in drinking water.	Sodium Sulphate	Chloride ion in water is closely associated with sodium, and the adverse effects of chloride and Na are difficult to separate With regard to interaction of chloride with sulphate, imbalance of either sulphate or chloride, or as a synergistic effect of both may upset acid-base homeostasis.	Most animals can tolerate relatively large amounts of chloride.	The body has very effective mechanisms to control chloride ion levels, and from a water quality perspective, under most practical situations, the risk of chronic toxicity of chloride is generally negligible.

†Peterson, 2000.

9.4.2 Water Types or Conditions Where High Levels Occur

Chloride concentrations in groundwater is determined by the geological formation in the aquifer and recharge area. Some deep and old groundwater sources in Saskatchewan may contain significant chloride content but only about 1% of the groundwater sources exceed the Canadian guideline for livestock of 1000 mg/L (Saskatchewan Watershed Authority Rural Water Quality Data Base). The highest chloride level recorded in the Saskatchewan Watershed Authority database is 4090 mg/L. Chloride is generally present at low concentrations in natural surface waters in Canada except in coastal regions where there may be salt water influence.

High chloride levels will also result in high TDS and conductivity levels. Chloride is usually associated with sodium which also contributes to high TDS and conductivity. In most cases, the Canadian guideline for TDS (3000 mg/L) is exceeded before the chloride levels reach the guideline of 1000 mg/L.

Chloride

Table 9.4.3 Chloride Levels in Saskatchewan Groundwater

Chloride Content (mg/L)	Number of Samples Analysed	Percent of Total
<250	2737	94.5
250 to 500	100	3.5
500 to 1000	28	1.0
1000 to 2000	27	0.9
>2000	3	0.1

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.4.3 Management Considerations

In the assessment of the potential risk of adverse effects associated with chloride in water, one should take into consideration balancing dietary salt levels, as well content of sodium and sulphate ions.

9.4.4 Treatment Technology

Treatment technology includes:

- Nano- Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.5 Fluoride

Fluoride is the stable form of fluorine having combined with another element. It is abundant in the biosphere and earth's crust and can be a major source of contamination for livestock drinking ground water.

The major sources of fluorides in Canada are phosphate fertilizer production, chemical production, and aluminum smelting. These three sources collectively account for over 75% of the estimated 23,500 tons of inorganic fluorides released to the Canadian environment annually. More than 13,500 tons of fluoride-containing materials are released in effluents, hence the risk of water contamination in some areas may be high. The amount of fluoride in water can be influenced by pH and water hardness.

CCME guidelines for livestock are 1 to 2 mg F/L, but it has also been noted that, at a level of 2 mg/L, mottling of teeth may occur. It is important to stress that the tolerance levels in water may depend on total intake of fluorine from all dietary and environmental sources.

Table 9.5.1 Examples of dietary intake of fluoride associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Fluoride**		
Water F content (mg/L)	**Estimated Water Contribution to Total Dietary Fluoride Intake (mg/day)**	**Estimated Contribution of Fluoride From Normal Feed (mg/day)**	**Estimated Dietary Fluoride Levels Generally Regarded as Safe and Dietary Fluoride Levels Consideration for Risk of Adverse or Toxic Effect (mg/day)**	
2	**64 to 80**	**220 to 280***	**Safe Levels** *(generally regarded as nutritionally balanced)*	**NA**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**440– 560****
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>560****

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.

† Guidelines for water are based on CCME recommendation.

* Calculation based on values cited as upper limit found in natural forages being 20mg F/kgDM (NRC, 1974).

** Calculation based on tolerance level values from NRC - Mineral Tolerance of Animals 2005, 2nd Revised Ed, Committee on Minerals and Toxic Substances in diets and Water for Animals, The National Academies Press, Washington, DC..

NA=data not available

Table 9.5.2 NRC recommended maximum levels of fluorine in feed and water for various classes of ruminant livestock.

	Maximum recommended	
Class of Livestock	**Diet (ppm)**	**Drinking water (mg/L)**
Young dairy cattle	30	2.5-4.0
Slaughter cattle	100	12-15
Mature dairy cattle	40	3-6
Mature beef cattle	50	4-8
Ewes	60	5-8
Finishing Lambs	100-150	12-15

These figures, set by the National Research Council (NRC, 1980), are widely used in many publications. However, since the availability of fluorine largely depends on the form and source, these values may not be universally applicable to every situation. For instance, the limit of tolerance for dairy set by NRC is approximately 40 mg F/kg DM when ingested as NaF. Tolerance of dairy cows to the fluoride in CaF (and presumably to soil fluoride) may by twice as high (Shupe *et al.,* 1962). According to Lewis (1995), water fluoride at a concentration of 4 ppm is considered to be marginally safe for horses, but water containing more than 8 ppm should be avoided.

Downward revision of the safe fluoride allowances for breeding ewes was suggested by Wheeler *et al.,* (1985). However, these numbers may need to be further revised to account for possible differences in metabolic tolerance of modern, highly producing animals.

9.5.1 Evaluation of Risk

In industrial areas, emission of fluorine fumes or fluoride dusts may contaminate the plants and water consumed by the animals. Considering that the environmental output of fluorine in some areas may be very high, the possibility that water may become contaminated must be considered. Higher risk of exposure to toxic or potentially toxic amounts of fluorine by farm animals exists in areas where the drinking water is naturally high in fluoride (known as endemic fluorosis). Total intake of fluorine may be increased when the animal's diet contains an excess of fluoride-bearing minerals used as a source of extra calcium and phosphorus.

Fluorine levels in water may be highly variable, depending on area and industrial activity. While considering the risk of exposure to fluoride, several factors must be evaluated. The mere presence of fluoride may, or may not be, a factor mitigating the risk of adverse effects because the bioavailability of fluorine depends on the source and form. For instance, retention of fluorine from aluminum or calcium fluorides is low. On the other hand, soluble fluorides are rapidly and almost completely absorbed from the GI tract. Absorbed fluorine is distributed rapidly throughout the body as the fluoride ion, and readily crosses cell membranes. Furthermore, other components of water and diet

must be considered. For instance, calcium and magnesium salts, as well as sodium chloride may reduce absorption of fluorine from the GI tract. Inadequate dietary carbohydrate intake enhances F absorption.

Health Effects: Signs of acute F toxicity include: restlessness, sweating, anorexia, salivation, dyspnea, nausea, gastroenteritis, muscle weakness, clonic convulsions followed by depression, pulmonary congestion and respiratory and cardiac failure. However, acute toxicity is very unlikely to occur in association with water fluorine under normal circumstances.

Fluorine is a cumulative toxin, and for this reason animals that live longer (e.g. dairy or beef cows) are more likely to develop chronic fluorosis.

No single criterion can be used to define F toxicity. Dental defects are the most sensitive indicators of elevated fluorine intakes with signs such as:

- delayed eruption of permanent incisor teeth.
- changes in teeth shape, size, color, and orientation.

Bone lesions associated with fluorosis can occur in animals exposed at any age. Bones of animals with signs of fluorosis appear chalky, rough, and porous compared with normal bones. Associate signs may be manifested as lameness, stiffness, treading of the feet, curled and abnormal hoofs, dry, lustreless hair and non pliable skin. Reduced immune response has also been observed.

Production Effects: Usually, in cases of chronic, moderate levels of exposure, clinical signs of toxicity appear only after several weeks or even months, and, at a low level of exposure, clinical signs of toxicity may develop over several years. For instance, at 50 mg F per kg DM, signs of fluorosis may appear within 3-5 years (Suttie *et al.,* 1957). In the study of Shupe *et al.,* (1963) when exposure commenced with young calves and lasted for 7 years, the tolerance for soluble fluoride was 30 mg F kg DM.

However, fluorine deposition in the skeleton occurs even at low levels of exposure. Exposure of the pregnant and lactating animal to fluoride may increase levels of fluoride in the milk and blood of the neonate (Wheeler *et al.,* 1985). During the initial stages, milk production parameters may not be significantly affected (Suttie and Kolstad, 1977). Also, digestibility and utilization of energy and protein are not significantly depressed (Shupe *et al.,* 1962, 1963). Nevertheless, secondary effects of subclinical changes associated with fluoride should not be ignored. For instance, impaired mastication and increased sensitivity to cold drinking water may lead to impaired feed intake, protein absorption, and consequently stunted growth and reduced milk yield.

High dietary fluoride levels may affect milk production (Stoddard *et al.,* 1963). Also, adverse effects on reproduction have been reported (IPCS, 2002). Poor reproductive performance in association with water fluorine in cattle may occur, but the risk of these effects in a practical situation is very low, if at all realistic. The apparent threshold for reproductive effects associated with fluorine in drinking water has been set at 100 to

200 mg/L ((NRC, 1993). With some exceptions possible, such levels are not very realistic under normal situations.

Metabolic Interactions: Fluorine may interfere with Mg, Mn, Fe, Mo, Cu and Zn metabolism. Vitamin B12 synthesis and folic acid activity are compromised. Protein utilization decreases with increasing dietary F. Aluminum (as sulphate, chloride, lactate, or hydroxide) reduces F toxicity and accumulation in bone.

Table 9.5.3 Summary of practical information relevant to fluoride exposure in livestock.

Guidelines	Interactions		Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Metabolic Effects**	**Short Term, Moderate or High Level Exposure**	**Long Term, Low or Moderate to High level of Exposure**
1 to 2 mg F/L	magnesium, iron, manganese, copper, zinc, molybdenum	Fluoride may interfere with Mg, Mn, Fe, Mo, Cu and Zn metabolism. Vitamin B12 synthesis and folic acid activity are compromised. Protein utilization decreases with increasing dietary F. Calcium and Magnesium salts may reduce absorption of fluorine from the GI tract. Inadequate dietary carbohydrate intake enhances F absorption.	Acute toxicity is very unlikely in association with water fluorine. Signs of acute toxicity include: restlessness, sweating, anorexia, salivation, dyspnea, nausea, gastroenteritis, muscle weakness, clonic convulsions followed by depression, pulmonary congestion and respiratory and cardiac failure. In chronic, moderate levels of exposure, clinical signs of toxicity appear only after several weeks or even months.	At low level of exposure, clinical signs of toxicity may develop over several years Bone lesions associated with fluorosis can occur in animals exposed at any age. Bones of animals with signs of fluorosis appear chalky, rough, and porous compared with normal bones. The problem may be manifested as: lameness, stiffness, treading of the feet, curled and abnormal hoofs. At high levels, signs ay include: dry, lusterless hair and non pliable skin, reduced immune response. delayed oestrus and poor reproductive performance, stunted growth and reduced milk yield.

† CCME guidelines for livestock are 1 to 2 mg F/L, but it has also been noted that, at a level of 2 mg/L, mottling of teeth may occur. Tolerance levels in water may depend on many dietary variables, as well as on total intake of fluorine from all dietary and environmental sources.

9.5.2 Water Types or Conditions Where High Levels Occur

Fluoride occurs naturally in geological formations and concentrations vary depending on the source of the water. Fluoride is used in the manufacturing of aluminum, phosphate fertilizers and bricks so there are potential for surface water contamination. Rarely does the fluoride level in Saskatchewan groundwater exceed the Canadian guideline for livestock of 1 to 2 mg/L (Saskatchewan Watershed Authority Rural Water Quality Data Base).

Table 9.5.4 Fluoride Levels in Saskatchewan Groundwater

Fluoride Content (mg/L)	Number of Samples Analysed	Percent of Total
<1	934	97.0
1 to 1.5	21	2.2
1.5 to 2	4	0.4
2 to 4	2	0.2
>4	2	0.2

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.5.3 Management Considerations

In the assessment of the potential risk of adverse effects associated with fluorine in water one should take into consideration balancing fluorine levels in the diet. Also factors that may increase bio-availability of fluorine may be used to offset low to moderate levels.

9.5.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.6 Iron

Iron in earth's crust is the fourth most abundant element, and is widely distributed in the biosphere. Most ground water sources contain iron, but the content may be highly variable, depending on geographical and geological location. Deep well water sources tend to have higher content of iron than shallow wells, or sand point sources. Although iron is an essential element, its availability from water may be variable depending on its chemical form. In some water sources, iron may be most likely present in a form of insoluble iron oxides, and therefore its bioavailability is rather low.

Iron in the water for livestock is usually considered to be a nuisance problem (mainly with water lines), rather than a toxicological problem. CCME does not provide guidelines for water iron levels suitable for livestock. The aesthetic objective for iron in drinking water (for humans) is 0.3 mg/L.

Table 9.6.1 Examples of dietary intake of iron associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Iron‡**		
Water Fe content (mg/L)	**Estimated Water Contribution to Total Dietary Iron Intake (g/day)**	**Estimated Contribution of Iron From Normal Feed (g/day)**	**Estimated Dietary Iron Levels Generally Regarded as Safe and Dietary Iron Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
NA	**0.96 to 1.2 (based on 0.3 mg/L iron in water)**	**1.7 to 2.2**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**<5.31**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**5.32 – 7.97**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>7.97**

† CCME does not provide guidelines for water iron levels suitable for livestock.
* Arbitrary calculation based on content of iron 10mg/L, which is common in some parts of Saskatchewan
Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of iron in feed.
‡Values for feed are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.
NA=data not available

9.6.1 Evaluation of Risk

Health Effects: The risk of iron toxicity *per se* in livestock is considered to be very low. Direct toxic effects associated with iron overload *per se* in cattle have not been recorded. Fe overload increases the risk of infection and neoplasia. Secondary copper insufficiency may compromise first line of defence immune responses (Boyne and Arthur, 1986).

Production Effects: Characteristic signs of chronic iron overload are reduced feed intake, growth rate, and efficiency of feed conversion. At 1,600 ppm, iron caused significant reductions in daily gains and feed intake (Standish *et al.,* 1969). In calves, poorer performance may occur at dietary iron levels of 500 ppm or more (Koong *et al.,* 1970). Undesirable effects of iron on veal meat quality have been noted.

Although iron in the water for livestock is not likely to result in adverse effects or production parameters, contrary to common belief, the problem of iron in water should not be ignored. Iron in water, if present in an ionized form as a divalent cation, may interfere with the bioavailability of other divalent metals such as copper, zinc, magnesium, manganese, or calcium. Most of the adverse effects of dietary iron are indirectly associated with secondary deficiencies resulting from antagonistic interactions. Cu deficiency is the most likely outcome of excess dietary iron in cattle and sheep.

Interestingly, it has been suggested that elevated iron concentrations in the drinking water may be a significant risk factor promoting intestinal proliferation of *Clostridium botulinum* and subsequent botulism (Pecelunas *et al.,* 1999). Our recent research has shown that high iron water promotes proliferation of *Clostridium perfringens* in the chicken intestinal content, and thus may increase the risk of necrotic enteritis (Olkowski *et al.,* manuscript in preparation).

Although high levels of iron in drinking water may not be of toxicological significance per se, secondary metabolic effects should be considered for at least two reasons: 1) iron may affect water palatability, and thus reduce water intake, and 2) excessive intake of iron may have detrimental effects on metabolism of several essential micronutrients.

Metabolic Interactions: Excess iron may affect many metabolic processes via a wide range of metabolic interactions. Among the physiologically significant effects are interactions with essential nutrients such as Co, Cu, Mn, Se, and Zn, where deficiency of these elements can be induced by high dietary iron. Antagonisms between copper and iron may have metabolic consequences (Suttle *et al.,* 1984, Suttle and Peter, 1985).

Iron

Copper status in cattle has been lowered by as little as 250 mg Fe/kg DM (Bremner *et al.,* 1987). The Fe antagonism towards copper does not appear to be manifested in the pre-ruminant calf (Bremner *et al.,* 1987). At a level of 1,000 mg of supplemental iron per kilogram diet, the deleterious effect on copper status of cattle could not be alleviated by either copper sulphate or copper proteinate at the supplemental concentrations (5 or 10 mg/kg diet). Simmental steers consistently had lower copper status than Angus cattle, suggesting that Simmental have a higher copper requirement (Mullis *et al.,* 2003).

The accelerated depletion of liver copper reserves in weaned, iron-supplemented calves (Humphries *et al.,* 1983) probably reflects inhibition of copper absorption, and the interactions in both sheep (Suttle *et al.,* 1984) and cattle (Bremner *et al.,* 1987) are in part dependent on sulphur.

Ruminants consuming forage-based diets are often exposed to high levels of Fe through water, forage, and/ or soil ingestion. High dietary Fe has been shown to greatly reduce Cu status in cattle (Standish *et al.,* 1971; Campbell *et al.,* 1974; Humphries *et al.,* 1983) and sheep (Prabowo *et al.,* 1988). Steers supplemented with 1000 mg Fe/kgDM also had reduced liver Zn concentrations (Standish *et al.,* 1971), suggesting that bioavailability of Zn is also reduced by high dietary Fe.

Ascorbic acid (vitamin C) is known as an enhancer of iron absorption. Interactions of ferrous salts with vitamin C have been shown to have detrimental effects on animals (Fisher and Naugton, 2004).

At the10 ppm level, water iron may contribute significantly to the overall dietary iron intake. For example, a cow producing 30 kg milk per day will drink, depending on environmental temperature, between 92 and 146 L of water per day. If the water contained 10 mg/L of Fe, water contribution to the Fe intake would be 920 to 1460 mg/day.

Table 9.6.2 Summary of practical information relevant to iron exposure in livestock.

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
NA	**Essential Nutrients**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
CCME does not provide water iron levels for livestock. The aesthetic objective for iron in drinking water is 0.3 mg/L.	selenium, cobalt, manganese, copper, zinc calcium Vit C and E	NA	Water palatability may be affected by high levels of iron in water. Co, Cu, Mn, Se, and Zn deficiency can be induced by high Fe. Copper status in cattle has been lowered by as little as 250 mg Fe/kg DM. Depletion of liver copper reserves in weaned, iron-supplemented calves may be associated with impaired copper absorption, and the interactions in both sheep and cattle are in part dependent on sulphur. Ascorbic acid (vit C) may enhance iron absorption, whereas vit E can prevent adverse effects.	Direct toxic effects associated with iron overload *per se* in cattle have not been recorded.	Iron in water, if present in an ionized form as a divalent cation, may interfere with the bioavailability of other divalent metals such as copper, zinc, magnesium, manganese, or calcium. Most of the adverse effects of dietary iron are indirectly associated with secondary deficiencies resulting from antagonistic interactions. Cu deficiency is the most likely outcome of excess dietary iron in cattle and sheep. Characteristic signs of chronic iron overload are reduced feed intake, growth rate, and efficiency of feed conversion. At 1,600 ppm, iron caused significant reductions in daily gains and feed intake. In calves, poorer performance may occur at dietary iron levels of 500 ppm or more.

NA=data not available

9.6.2 Water Types or Conditions Where High Levels Occur

Both surface and groundwater sources contain iron, although groundwater sources tend to have higher concentrations. In surface water sources the oxidative environment often causes precipitation and settling of the iron. Anaerobic conditions can dissolve the settled iron and bring it back into water body. In groundwater, the reductive environment dissolves iron and maintains it in a dissolved state.

Table 9.6.3 Iron Levels in Saskatchewan Groundwater

Iron Content (mg/L)	Number of Samples Analysed	Percent of Total
<0.1	1405	47.3
0.1 to 0.3	328	11.1
0.3 to 1	416	14.0
1 to 2	258	8.7
2 to 5	351	11.8
5 to 10	161	5.4
10 to 20	39	1.3
>20	11	0.4

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.6.3 Management Considerations

Iron in the water for livestock is more likely to be considered a nuisance problem (mainly with water lines), rather than a toxicological problem. Dietary balancing of nutrients affected by excessive intake of iron should be effective to alleviate adverse effects of iron associated with metabolic interactions. Iron removal is probably the most practical approach to effectively deal with high iron content in water.

9.6.4 Treatment Technology

Treatment technology includes:

- Coagulation
- Manganese greensand filters may be effective in reducing iron in water
- Slow sand filter
- Biologically activated carbon with pre-oxidation
- Oxidation/pH modification and filtration
- Nano-Filtration or RO membranes
- Oxidation and settling

Treatments used to remove only iron from water for livestock can be economically feasible. Often iron or manganese exists in water with high arsenic content, and removal of both substances with one treatment system may provide economic benefit. See Section on water treatment for further discussion on specific treatment systems.

9.7 Lead

Lead occurs naturally in the earth's crust at a concentration of about 13 mg/kg, but there are some areas with much higher concentrations, including the lead ore deposits scattered throughout the world. The concentration of lead in surface water is highly variable depending upon sources of pollution; lead content of sediments; and the pH, salinity, and organic matter content of the water. Dissolved lead concentrations in unpolluted freshwaters are generally very low, <0.01 mg/L (Fergusson 1990, Galvin 1996). Most lead (over 90%) transported by unpolluted streams is associated with suspended particulate matter (Salomons & Förstner 1984). A major source of lead for waterfowl and other wildlife is spent lead shot, bullets, cartridges, and the lead sinkers used in sport fishing (Burger and Gochfeld, 2000; D Francisco *et al.,* 2003).

According to the Canadian guidelines (CCREM 1987), drinking water lead concentration should be below 0.1 mg/L. In some classes of highly producing livestock, a lead level of 0.1 mg per litre water may contribute to the overall intake of several milligrams of lead daily (Table 9.7.1).

Table 9.7.1 Examples of dietary intake of lead associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Lead‡**	
Water Pb content (mg/L)	**Estimated Water Contribution to Total Dietary Lead Intake (mg/day)**	**Estimated Contribution of Lead From Normal Feed (maximum limit)***	**Maximum Tolerable Dietary Level (mg/kg DM)**
0.1	3.2 to 4.0	NA‡ (55 to 70 mg/day)*	NA‡ (30 mg/ kg)¥

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of lead in feed.
† Guidelines for water are based on CCME 2005 recommendation.
‡Values for dietary levels are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.
NA=data not available.
Note 3: According to NRC (2005) in ruminants, 250 mg/kg lead in the diet can be tolerated for several months without significant effects on performance; however, levels of lead in kidneys are hone become of concern if consumed by humans.
Note 4: *Values used by CFIA as "Metal Reporting Limits" for lead are set at 5 ppm (information provided by Feed Specialist Inspector, CFIA, author's personal communication).
¥Recommendation according to Puls, 1994,

9.7.1 Evaluation of Risk

The risk of lead toxicity in livestock depends largely on the type of animal, physiological and nutritional status, and age. Although the risk of adverse effects associated with lead in drinking water is generally very low, water may contribute to the overall burden of dietary lead.

Feed can contain considerably larger quantities of lead than water, but it has to be stressed that lead in water is more efficiently absorbed than lead in food (Goyer 1997). Hence, animals can tolerate considerably higher daily exposure levels of lead when it is consumed in the diet than in the water. Lead ingested in water, without simultaneous food consumption, is considerably more toxic than when water is ingested with a meal.

Young animals absorb lead more efficiently than older animals and show lower tolerance to lead. Cattle, especially young calves, are extremely susceptible to lead toxicity (Neathery and Miller, 1975).

Among dietary factors, calcium status is one of the most important factors modulating lead toxicity. High levels of dietary calcium and phosphorus decrease intestinal absorption of lead and thus decrease its toxicity. Low dietary iron enhances gastrointestinal lead absorption, and thus increases the susceptibility of animals to lead toxicity. Lactose promotes lead absorption in calves (Zmudzki *et al.,* 1986). Selenium and monensin increases lead accumulation is chickens (Khan *et al.,* 1993,1994).

With low to moderate body burden, most lead is retained in the skeleton. However, beyond a certain point, the kidney and liver may accumulate lead in large quantities. Lead passes the placenta more readily then other heavy metals.

Health Effects: Lead can be a lethal toxin if ingested by livestock in large amounts. For instance, it has been reported that calves died after accidental exposure to an estimated dose of 5–8 mg Pb/kg BW/d for 30 days (Osweiler & Ruhr 1978). Sheep death was reported following dietary exposure to 5.7 mg Pb/kg BW/day (James *et al.,* 1966).

Lead affects several organ systems, including the nervous, hematopoietic, renal, endocrine, and skeletal. Initially, lead is accumulated in the skeleton, but when the threshold is exceeded, lead levels in circulation may increase drastically until signs of poisoning occur. Signs of lead toxicity are mostly not specific and may include: anaemia, anorexia, fatigue, depression, constipation or diarrhoea, abdominal pain, nephropathy, blindness, head pressing, bawling, trembling, convulsions, and salivation. Chronic exposure may result in loss of weight.

Chronic effects such as anorexia and respiratory distress are associated with low level poisoning. In chronically exposed animals, blood Pb increases at the end of pregnancy and beginning of lactation as bone minerals are mobilized. Abortions have been

observed. Pb begins to transfer to milk when blood Pb exceeds 0.30 ppm. Difficulty swallowing or suckling in calves has been observed. Lead is known to decrease immune response. Reduced resistance to diseases has been reported following low-level intake of lead (Hemphill *et al.,* 1971).

Diagnosis of lead toxicity can easily be confirmed post mortem. In acute cases, high lead concentrations may be found in digesta and feces, as well as in kidneys.

Production Effects: Low dietary intake of lead does not result in any appreciable rise of lead in products such as milk or meat, but liver and kidney accumulate lead. At high dosage rates lead can accumulate in soft tissues of animals to a degree that might exceed acceptable levels for human consumption, if livestock are raised in areas contaminated with lead (NRC 1980). Lead may adversely affect both female and male reproductive functions (IPCS, 1995; Sallmen, 2001).

In addition to the direct effect of lead on health or production parameters, the exposure to lead ought to be also considered in the context relevant to contamination of animal products.

It is noteworthy that even at low levels of exposure, potentially consumable organs such kidney or liver may accumulate lead. Although there is no appreciable rise of lead in milk at low level of lead intake, lead exposure studies showed a dose-related increase in milk (Sharma *et al.,* 1982). Since lead in milk is highly available (Hallen and Oskarsson, 1995), suitability of milk from cows exposed to dietary lead for human consumption may become an issue.

Metabolic Interactions: Lead may interfere with the metabolism of several essential metals. Dietary lead increases liver zinc, but decreases liver copper and kidney manganese. Increased levels of calcium, cobalt, zinc, copper, iron, and selenium may reduce lead toxicity. Increased cadmium may enhance lead toxicity. Lead toxicity also impairs vitamin D metabolism, and may increase the apparent need for dietary calcium. Ascorbic acid, thiamine, and nicotinic acid may reduce lead toxicity.

Table 9.7.2 Summary of practical information relevant to lead exposure in livestock.

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
0.1 mg/L.	calcium, selenium, iron, manganese, copper, zinc, Vit. D	cadmium	Dietary lead increases liver zinc, but decreases liver copper and kidney manganese. Increased levels of calcium, cobalt, zinc, copper, iron, and selenium may reduce lead toxicity. Increased cadmium may enhance lead toxicity. Ascorbic acid, thiamine, and nicotinic acid may reduce lead toxicity.	Signs of acute toxicity may be manifested as: anorexia, fatigue, depression, constipation or diarrhea, abdominal pain, nephropathy, blindness, head pressing, bawling, trembling, convulsions, loss of weight, abortion or salivation. Difficulty swallowing or suckling in calves has been observed.	Chronic effects such as anorexia and respiratory distress are associated with low level poisoning. Lead affects both male and female reproductive functions. Lead may decrease immune responses. Reduced resistance to diseases has been reported following low-level intake of lead.

†CCME2005

9.7.2 Water Types or Conditions Where High Levels Occur

Lead is the most common heavy metal and is widely used for production of batteries, gasoline additive and other chemicals. Saskatchewan does not have high concentrations of lead ore deposits therefore unless the water is contaminated, lead levels are low. More than 99% of all water is less than the Canadian guideline of 0.01 mg/L for humans, and only 1 in 3000 samples is greater than the 0.1 mg/L established for livestock (Saskatchewan Watershed Authority Rural Water Quality Data Base).

Table 9.7.3 Lead Levels in Saskatchewan Groundwater

Lead Content (mg/L)	Number of Samples Analysed	Percent of Total
<0.01	2943	99.3
0.01 to 0.1	21	0.7
>0.1	1	0.03

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.7.3 Management Considerations

Since in practical situation feed can contain considerably larger quantities of lead than water, major effort in risk management should be focused on feed. However, it has to be remembered that lead in water is more efficiently absorbed than lead in food, so if lead contend in water is significant, water treatment would be highly recommended. In view of the fact that low dietary iron enhances gastrointestinal lead absorption, and thus increases the susceptibility of animals to lead toxicity, dietary iron status should be monitored in areas where water lead exposure is prominent.

9.7.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.8 Magnesium

Drinking water from natural sources usually contains magnesium, but levels may vary greatly with location and often with season.

Magnesium is an essential nutrient required for numerous biochemical and physiological functions. Magnesium is present in variable amounts in common animal feed (NRC, 1979), but there is a large degree of variability among different feedstuffs, in particular in forages (Reid *et al.,* 1970). Legumes are generally higher in magnesium than grasses. There are a number of sources of supplemental magnesium commonly used in the feed industry. Bioavailability of magnesium may differ substantially, depending on source.

At present, there is no guideline for magnesium for livestock drinking water. A concentration of 6000 mg/L reduced growth and bone mineralization in immature chickens. An upper limit of 300 to 400 mg/L has been suggested for dairy cows (Peterson, 2000)

Table 9.8.1 Examples of dietary intake of magnesium associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Magnesium‡**		
Water Mg content (mg/L)	**Estimated Water Contribution to Total Dietary Magnesium Intake (g/day)**	**Estimated Contribution of Magnesium From Normal Feed (g/day)**	**Estimated Dietary Magnesium Levels Generally Regarded as Safe and Dietary Magnesium Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
400¥	**12.8 to 16**	**24 to 31**	**Adequate Levels** *(generally regarded as nutritionally balanced)*	**27.5 – 48.0**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**110 – 560**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of magnesium in feed.
† No CCME Guideline. ¥Value based on suggested upper limit for dairy cows (Peterson, 2000)
‡Values for dietary levels are from R. Puls , 1994.

9.8.1 Evaluation of Risk

The risk of toxicity associated with magnesium present in Canadian water sources appears to be extremely low. Furthermore, if one excludes accidental nutritional errors, under normal practical conditions adverse effects associated with magnesium due to ingestion of natural feedstuffs and water are unlikely to occur.

Nevertheless, magnesium can be toxic when administered at high levels, and while assessing the tolerance criteria for magnesium in drinking water, total dietary magnesium, as well as magnesium bioavailability should be taken into consideration.

Generally, cattle and sheep should be able to tolerate 0.5% magnesium, whereas the maximum tolerable level for poultry and swine appears to be 0.3%. The risk of outright magnesium toxicity in practical situations is negligible, but it has to be stressed that much lower levels of dietary magnesium have been found to affect performance.

Health Effects: The signs of acute toxicity include disturbance in locomotion, lethargy, coma and death. Scouring is a common problem with high dietary magnesium levels. Very high levels of magnesium in drinking water may present serious problems in farm animals. In one report, magnesium levels in water of about 1% was reported to cause a weakening effect on humans and farm animals in parts of Minnesota, the Dakotas, and Montana (Allison, 1930). Cattle and hogs raised in these areas could not be fattened for market while drinking this water. Calves were stunted and many never matured. Cattle developed a “run-down-ragged appearance,” and many died prematurely. A degeneration of the bones occurred. Peirce (1959) reported that drinking water containing 0.2-0.3% magnesium chloride was harmful to sheep.

Production Effects: Younger animals may be more sensitive to excessive intake of magnesium. For instance, increasing the level of dietary magnesium from 0.16 to 0.22% has resulted in lower rate and efficiency of weight gain in swine during earlier stages of growth (20 to 45 kg), but had no effect thereafter (Krider *et al.,* 1975). Studies of O’Kelley and Fontenot, (1969, 1973) have shown that mature cows, regardless whether during gestation or lactation, were not affected by dietary magnesium levels as high as 0.29%.

Excess dietary intake of magnesium has been found to cause depressed growth rate in chicks (Nugara and Edwards, 1963; Chicco *et al.,* 1977), and sheep (Kerk, 1973). The decrease in performance appears to be caused partly by decreased feed intake.

In monogastric animals, the most likely adverse effect of magnesium in drinking water is the laxative effect, particularly with magnesium sulphate. However, in ruminant livestock, the detrimental effects of sulphate would be of more patho-physiological importance than the adverse effects of magnesium (for details see chapter on sulphur).

Magnesium

Metabolic Interactions: Excess intake of magnesium can affect bioavailability and metabolism of several divalent essential elements such as Cu, Fe, Mn, Ca, and Zn. However, in comparison to other minerals, magnesium interaction with Ca and P appears to be of more specific patho-physiological significance.

When 0.6 percent magnesium was supplemented, growth and bone mineralization were adversely affected regardless of the calcium and phosphorus levels, but lower levels of 0.2 or 0.4% magnesium tended to alleviate the adverse effects of deficiencies of both calcium and phosphorus in chicks (Chicco *et al.,* 1967).

High levels of calcium and phosphorus have been shown to depress magnesium absorption in sheep (Chicco *et al.,* 1973; Pless et al.,1973). Calcification in hearts and kidneys of rats administered high levels of vitamin D was aggravated by high dietary levels of magnesium (Whittier and Freemen, 1971).

High dietary potassium depresses magnesium absorption in ruminants (Newton *et al.,* 1972).

9.8.2 Water Types or Conditions Where High Levels Occur

According to studies conducted by Environment Canada, magnesium concentrations as high as 168 mg/L have been found in Canadian water sources, but in most cases, magnesium content was below 25 mg/L. Two national surveys of drinking water supplies, encompassing 115 municipalities across Canada, were conducted in 1976 and 1977 (Méranger *et al.,* 1979, 1981). Magnesium concentrations in distributed water ranged from 0.2 to 2230 mg/L, with the highest median concentrations being in Alberta (17 mg/L), Saskatchewan (28 mg/L, and Manitoba (23 mg/L). In Saskatchewan, magnesium levels over 400 mg/L is rare, therefore magnesium is rarely a concern in water supplies (Saskatchewan Watershed Authority Rural Water Quality Data Base).

9.8.3 Management Considerations

An excess of dietary magnesium can be managed through the following measures: 1) modification of the diet to balance total Mg intake, and 2) dietary intervention aimed at balancing nutrients that can be affected by metabolic interactions with magnesium.

9.8.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

Table 9.8.2 Summary of practical information relevant to magnesium exposure in livestock

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
400mg/L	calcium iron, manganese, copper, zinc, potassium	cadmium	Excess intake of magnesium can affect metabolism of Cu, Fe, Mn, Ca, and Zn. In comparison to other minerals, magnesium interaction with Ca and P appears to be of more specific patho-physiological significance. High dietary potassium depresses magnesium absorption in ruminants.	Magnesium is toxic when administered at high levels. The signs of acute toxicity include disturbance in locomotion, lethargy, coma and death. Scouring is a common problem with high dietary magnesium levels.	In monogastric animals, the most likely adverse effect of magnesium in drinking water is the laxative effect, particularly with magnesium sulphate. In ruminant livestock, the detrimental effects of sulphate would be of more patho-physiological importance than the adverse effects of magnesium (for details see chapter on sulphur).

† Not a guideline. Value based on suggested upper limit for dairy cows (Peterson, 2000)

Table 9.8.3 Magnesium Levels in Saskatchewan Groundwater

Magnesium Content (mg/L)	Number of Samples Analysed	Percent of Total
<40	1033	35.7
40 to 100	1127	39.0
100 to 200	570	19.7
200 to 400	136	4.7
>400	27	0.9

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.9 Manganese

Manganese can be present in natural surface waters as dissolved or suspended matter, but water is a minor source of the total manganese intake. Presently there is no Canadian guideline for livestock for manganese. There is a Canadian aesthetic guideline of 0.05 mg/L for distribution systems which is not based on toxicity but rather potential problems in restricted flow devices in water lines. Research indicated that 50 to 125 mg/L reduced haemoglobin in baby pigs and 45 mg/L caused anaemia in lambs. Generally, the contribution of water manganese to the total dietary manganese appears to be negligible (Table 9.9.1).

Table 9.9.1 Examples of dietary intake of manganese associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Manganese‡**		
Water Mn content (mg/L)	**Estimated Water Contribution to Total Dietary Manganese Intake (g/day)**	**Estimated Contribution of Manganese From Normal Feed (g/day)**	**Estimated Dietary Manganese Levels Generally Regarded as Safe and Dietary Manganese Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
NA† **5.0¥**	**0.16 to 0.20**	**0.46 to 0.59**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**0.43 – 1.27**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**1.28 – 2.55**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>2.55**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of manganese in feed.
† Canadian guidelines are not available. ¥Value of 5 mg/L is based on observation of Peterson (2000).
‡Values for dietary levels are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.

9.9.1 Evaluation of Risk

Overall, manganese is considered as a metal of very low toxic potential. In most cases, the risk of adverse health effects associated with manganese in drinking water is, if any, very low. At a concentration greater than 0.05 ppm manganese may affect water palatability.

The most likely source of excessive manganese is the dietary component. Levels of Mn in excess of 30 mg/kg can be found in some grains, rice and nuts. Although the risk of toxicity associated with manganese is negligible, if dietary content of manganese is already high, water manganese may increase the risk of subtle metabolic disturbance associated with manganese interaction with other essential metals.

Manganese may cause problems in plumbing and watering equipment. There are known cases where water pipelines were totally blocked by manganese precipitate. In Saskatchewan, the greatest danger to producers is not from toxic effects but rather from having line blockage and thereby restricting water availability to livestock.

Health Effects: Notably, levels of manganese toxicity cited in the past research are extremely variable. Adverse health effects have not been observed in most species with dietary concentrations of 1,000 ppm manganese or less, but there is a general consensus that at 2,000 ppm and above, growth retardation, anaemia, gastrointestinal lesions can be observed in most species. According to Puls (1994) tolerance limits for manganese in mature cattle is approximately 1000-2000 ppm, and for calves 500 ppm. Swine appear to be more sensitive to manganese than cattle, sheep, or poultry.

At low level, long term exposure, the brain appears to be especially vulnerable to manganese toxicity. In humans, manganese is most commonly associated with occupational exposure to aerosols or dusts that contain extremely high levels of manganese, and consumption of contaminated well water.

Production Effects: Although relatively high levels of manganese may be required to cause overt toxicity, it is important to note that subtle patho-physiological changes associated with metabolic interaction of manganese with other elements may occur at relatively low levels of manganese excess.

A number of experimental studies have shown that exposure to manganese can cause deleterious effects on the male reproductive system. A delayed growth and maturation of the testes was reported in young mice dosed orally with 140 mg of Mn oxide per kilogram per day for 90 days (Gray and Laskey, 1980). Manganese chloride ingested in drinking water may affect fertility and reproduction (Elbetieha *et al.,* 2001). Exposure to manganese was found to be associated with a reduction in sperm motility and concentration (Ponnapakkam et a., 2003, Wirth *et al.,* 2007).

Metabolic Interactions: Manganese may adversely affect metabolism and homeostasis of several divalent metals including Ca, Cd, Co, Fe, P and Zn. Iron deficiency may enhance absorption of manganese (Thomson *et al.,* 1971, 1972; Flanagan *et al.,* 1980).

It is noteworthy that metabolic interaction may be induced at relatively low levels of manganese excess. For instance, decreased copper absorption has been observed in a calf supplemented 50 ppm manganese above 12 ppm in the basal diet (Ivan and

Grieve, 1976). Negative calcium balance during early lactation was observed in cows fed 70 ppm manganese (Reid *et al.,* 1947).

Table 9.9.2 Summary of practical information relevant to manganese exposure in livestock

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
NA† 5.0 mg/L¥	calcium cobalt, iron, copper, zinc phosphorus	cadmium	Manganese may adversely affect homeostasis of several essential metals including Ca, Co, Fe, Cu, P and Zn. Metabolic effect associated with interactions with other essential elements may be induced at relatively low levels exposure.	Acute toxicity is very unlikely Manganese is considered as a metal of very low toxic potential.	At low levels, long term exposure, the brain tissue appears to be especially vulnerable to manganese toxicity. Manganese can have detrimental effects on the male reproductive system.

† Canadian Guideline for manganese not available. ¥Value of 5 mg/L is based on observation of Peterson (2000).

9.9.2 Water Types or Conditions Where High Levels Occur

The analysis conducted for Water-Quality Assessment Program of the US Geological Survey (USGS, 2005), suggests that approximately 6% of domestic wells contain high levels of Mn in drinking water in the range of 0.3 mg/L. A survey of Canadian surface waters undertaken in 1980–1981 showed that the usual range of manganese in freely flowing river water was 0.01–0.40 mg/L. The highest concentrations recorded were in the Carrot River in Saskatchewan; dissolved manganese reached 1.7 mg/L, whereas extractable manganese peaked at 4.0 mg/L.

Manganese is more prevalent in groundwater supplies than in surface water supplies owing to the reducing conditions that exist underground. High concentrations of manganese are also found in some lakes and reservoirs as a result of acidic pollution.

In Saskatchewan most groundwater sources have manganese exceeding 0.05 mg/L (Saskatchewan Watershed Authority Rural Water Quality Data Base). This is a concern for producers with long distribution pipelines. Producers should be knowledgeable regarding the manganese level in their water and expect to have deposits develop on the inside of their pipelines.

Table 9.9.3 Manganese Levels in Saskatchewan Groundwater

Manganese Content (mg/L)	Number of Samples Analysed	Percent of Total
<0.05	958	32.3
0.05 to 0.1	271	9.1
0.1 to 0.2	353	11.9
0.2 to 0.4	469	15.8
0.4 to 1.0	597	20.1
1 to 2	234	7.9
2 to 4	73	2.5
4 to 8	12	0.4
>8	2	0.1

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.9.3 Management Considerations

An excess of dietary manganese can be managed through the following measures: 1) modification of the diet to balance total manganese intake, and 2) dietary intervention aimed at balancing nutrients that can be affected by metabolic interactions with manganese.

9.9.4 Treatment Technology

Treatment technology includes:

- Manganese greensand (also removes iron and arsenic)
- Biologically activated carbon with pre-oxidation (also removes iron and arsenic)
- Oxidation/pH modification and filtration
- Nano-Filtration or RO membranes

Often producers will not treat water for manganese and replace pipelines as required. Measures to mitigate the problem of build-up in pipelines include sequestering agents and flushing or pigging pipelines. Scaling potential can also be reduced by ensuring that the water is not exposed to air or chlorine which will oxidize the manganese and

cause precipitation. See Section on water treatment for further discussion on specific treatment systems.

9.10 Molybdenum

Water may contain variable levels of molybdenum, but in general, drinking water is a minor source of dietary molybdenum in livestock. Concentrations of molybdenum in normal herbage often range from 0.1 to 3 ppm (Underwood, 1977), whereas plants growing on soils containing naturally high levels of molybdenum or industrially contaminated with molybdenum have been reported to contain up to 231 ppm molybdenum (Gardner and Hall-Patch, 1962).

The soils in some geographic areas have relatively high molybdenum levels, and this is correlated with a regional incidence of molybdenosis in livestock. Levels of molybdenum in naturally growing herbage usually reflect the molybdenum content of the soil. Elevated levels of molybdenum in excess of 1 ppm in milk have been associated with high molybdenum pastures. While estimating safe levels of molybdenum in drinking water for livestock, a total dietary intake of molybdenum must be taken into consideration (Table 9.10.1), however risk assessment must include several nutritional, physiological, and metabolic variables.

Table 9.10.1 Examples of dietary intake of molybdenum associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Molybdenum**		
Water Mo content (mg/L)	**Estimated Water Contribution to Total Dietary Molybdenum Intake (mg/day)**	**Estimated Contribution of Molybdenum From Normal Feed (mg/day)**	**Estimated Dietary Molybdenum Levels Generally Regarded as Safe and Dietary Molybdenum Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
0.5	**16 to 20**	**NA 1.4 to 42***	**Safe Levels** *(generally regarded as nutritionally balanced)*	**NA‡**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**NA‡**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**NA‡**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.

† Guidelines for water in livestock (CCME, 2005).

NA=data not available

*Concentrations of molybdenum in normal herbage often range from 0.1 to 3 ppm (Underwood, 1977

‡Safe level will depend on the content of dietary sulphur and copper.

9.10.1 Evaluation of Risk

Species differences: Estimates of the maximum tolerable levels for molybdenum cited in the literature are highly variable depending on species. Tolerance limits ranging from 6.2 ppm in growing cattle to approximately 1,000 ppm in adult mule deer have been reported, but red deer may be more sensitive Grace *et al.,* 2005).

Horses appear more resistant to molybdenosis than cattle, as they can graze the pastures that are known to cause diarrhoea in cattle without apparent problems. However, clinical cases of rickets in foals and yearlings have been thought to be due to molybdenosis from pasture or dam's milk (Walsh and O'Moore, 1953). Levels of 5 and 10 ppm have been weakly associated with impaired bone development in young horses and cattle respectively. Walsh and O'Moore, suggested that excess of molybdenum in herbage may be a contributory factor in equine osteodystrophia.

In comparison to cattle or horses, pigs appear to be more resistant to Mo. Gipp *et al.,* (1967) and Kline *et al.,* (1973) have reported little to no effect of 26 to 50 ppm molybdenum upon swine growth in the presence of supplemental copper and sulphate, while Davis (1950) reported no apparent effect of 1,000 ppm molybdenum in growing swine. It is important to note that substantially higher levels of molybdenum would be tolerated in the presence of adequate copper and inorganic sulphate.

Avian species appear less susceptible to molybdenum. Only a slight growth inhibition in young chickens fed 200 ppm molybdenum, and a 25 percent growth inhibition in poults fed 300 ppm molybdenum were noted (Kratzer, 1952). Feeding molybdenum to young chicks at levels ranging from 500 to 8,000 ppm resulted in growth depression and anaemia at the lower levels, and 61% mortality at the highest level (Davies *et al.,* 1960).

Risks associated with molybdenum in drinking water: Undoubtedly, the risk of overt health effects associated with water molybdenum alone would be very low, but molybdenum in drinking water should not be ignored. However, without a complete evaluation of all relevant dietary factors influencing molybdenum toxicity, it may be difficult to predict the potential of adverse effects of molybdenum in water.

While assessing the tolerance criteria for molybdenum in drinking water, total dietary intake of molybdenum, as well as its metabolic interactions should be taken into consideration.

Ratios, lower than 10:1, of dietary copper to molybdenum may produce molybdenosis in cattle, especially if sulphur intake is excessive. High sulphates in the water and/or high molybdenum concentrations in the feed decrease dietary copper availability (Smart *et al.,* 1992). In many parts of Canada, forages and grains are marginal or deficient in copper, but in particular, a combination of dietary copper insufficiency, excess molybdenum, and high intake of sulphur are prevalent in some parts of Manitoba, Saskatchewan, and Alberta.

Some studies suggest that dietary Mo concentrations greater than 10 ppm are hazardous to cattle regardless of Cu concentration, but other reports indicate that this may not be the case.

For instance, Kincaid (1980) using dietary levels of 13 ppm copper and 0.29% sulphur, demonstrated that with these dietary levels of copper the minimum toxic concentration of molybdenum in drinking water for calves is between 10 and 50 ppm, and the critical copper-to-molybdenum ratio is less than 0.5. Also Raisbeck *et al.,* (2006) observed that 17 ppm of copper supplement to pregnant cows grazing pasture contaminated with 13 ppm molybdenum prevented molybdenosis. The authors concluded that even moderate supplementation of copper permitted cows to graze a site heavily contaminated with Mo with no adverse effects on general health or reproduction.

At present, recommended maximum concentrations for molybdenum in livestock drinking water is set at 500 µg/L (CCME, 2005). However, based on the facts discussed above, it would be more practical to consider the guidelines for water molybdenum content in the context of at least 2 important dietary variables i.e. copper and sulphur. Moreover, as evidenced by the studies of Kincaid (1980) and Raisbeck *et al.,* (2006), the problem of molybdenum in practical situations can readily be offset by dietary management of copper and sulphur.

Problems with molybdenum are more likely to occur in ruminant livestock. Sheep appear slightly more resistant to molybdenosis than cattle. In sheep, the manifestations of molybdenum-induced, secondary hypocuprosis include reduced crimp and pigmentation of wool, anaemia, alopecia, and reduced weight gains. Neonates born to hypocupremic dams exhibit enzootic ataxia (swayback), a debilitating disease that may also be accompanied by blindness.

Natural feedstuffs containing up to 6.2 ppm molybdenum were found by Smith *et al.,* (1975) to be associated with bone malformations in calves. Cunningham *et al.,* (1953) have reported that natural forages containing 25.6 ppm molybdenum were responsible for diarrhoea, emaciation, anemia, loss of hear pigmentation (achromotrichia), and even death in cattle of various age groups.

Molybdenum toxicity has been observed in young lactating cattle consuming as little as 40 ppm molybdenum when the diets contained 0.3 percent sulphate (Vanderveen and Keener, 1964). It appears that 100-200 ppm dietary molybdenum is required to significantly increase the molybdenum content of milk (Cunningham *et al.,* 1953).

Health Effects: Signs such as growth retardation signify more advanced molybdenosis. Manifestations of molybdenum toxicity in cattle include diarrhoea, anorexia, loss of pigmentation in the hair (achromotrichia), nervous system disturbances, and posterior weakness. This condition is essentially an effect of secondary copper deficiency induced by molybdenum, and it is probable that the main

signs, such as general growth retardation and anorexia, associated with molybdenosis are related to deficiencies of copper- dependent enzymes.

Production Effects: In the herd situation, it is more likely that adverse effects associated with excessive intake of molybdenum can fall in the category of subtle metabolic disturbances, which may cause economic losses without clear, specific clinical manifestation. In many cases adverse effects of molybdenum are due to secondary effects caused by metabolic interactions of molybdenum with other essential nutrients. Among the most important and best understood effects are those associated with molybdenum induced copper deficiency.

Also of practical importance to the livestock industry are the potential effects on reproductive performance. Thomas and Moss (1951) have observed decreased libido and testicular degeneration in young bulls fed 1-2 g sodium molybdate dihydrate daily for a period of 120 days. Several studies attributed reproductive effects such as early deaths of offspring, dead litters, maternal deaths, failure to breed with molybdenum (for review see Vyskocil and Viau, 1999). Various functions of the immune system can be affected (Boyne and Arthur, 1986; Gengelbach and Spears, 1998).

Metabolic Interactions: A wide variation in the apparent susceptibility of various livestock species to molybdenum toxicity is due to interactions with dietary levels of copper and sulphur. The apparent effects of molybdenum are also influenced by manganese, zinc, iron, lead, tungstate, ascorbic acid, methionine, cysteine, protein, and alkalinity of soils. The basis for many of these interactions is yet unexplained.

Of practical interest here are three way interactions between molybdenum, sulphur, and copper in ruminant animals (for review see Gooneratne *et al.,* 1989). Goodrich and Tillman (1966) investigated the effect of 2 and 8 ppm molybdenum on lambs receiving either 10 or 40 ppm copper and either 0.1 or 0.4 percent sulphate. At a level of 8 ppm, molybdenum eliminated the detrimental effects of the high sulphate on rate of gain and feed efficiency, and also reduced liver copper levels. The latter effect was reversed by the addition of 40 ppm copper.

9.10.2 Water Types or Conditions Where High Levels Occur

Molybdenum is not viewed as a contaminant in water that is sufficiently high to cause problems. Even water for human consumption is rarely tested for molybdenum. No data on the prevalence of molybdenum in Saskatchewan water were found.

9.10.3 Management Considerations

Mild to moderate excess dietary molybdenum can be managed reasonably well by dietary intervention. Preventative measures to be considered should include balancing the nutrients likely affected by molybdenum. In particular, attention should be focused on the dietary Sulphur and copper levels.

Table 9.10.2 Summary of practical information relevant to molybdenum exposure in livestock

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock[†]	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
500 µg/L	copper, sulphur, manganese, zinc, iron,	lead, tungstate	A wide variation in the apparent susceptibility of various livestock species to molybdenum toxicity is due to interactions with dietary levels of copper and sulphur. Of practical interest here are three way interactions between molybdenum, sulphur, and copper in ruminant animals. Metabolic effects are associated secondary copper deficiency induced by molybdenum. Main signs, such as general growth retardation and anorexia, associated with molybdenosis may be related to deficiencies copper-dependent enzymes. The apparent effects of molybdenum are influenced by manganese, zinc, iron, lead, tungstate, ascorbic acid, methionine, cysteine, protein, and alkalinity of soils.	Acute toxicity is not very likely under practical circumstances.	Manifestations of molybdenum toxicity in cattle include diarrhoea, anorexia, loss of pigmentation in the hair (achromotrichia), weakness nervous system disturbances. Signs such as growth retardation signify more advanced molybdenosis. Of practical importance to the livestock industry are the potential effects on reproductive performance.

[†] CCME (2005)

9.10.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems

9.11 Mercury

Mercury is one of most toxic metals that may be present in the farm animal environment. Anthropogenic activities such as mercury manufacture and disposal, fossil fuel combustion, and intensive agricultural practices contribute most of the mercury in the farm animal environment.

Mercury occurs in various sources in several chemical configurations, both organic and inorganic. Drinking water is one of the many possible exposure sources of mercury in farm animals. The concentration of mercury found in unpolluted streams and ground-waters is generally well below 0.001 mg/L. However, it is important to understand that mercury has a great potential for bio-accumulation in the food chain, and therefore intake of mercury from water and feed must be monitored, particularly in areas where the risk of potential contamination is high. Inorganic mercury is converted to organic compounds, which are stable, and may persist in the environment. Methyl-mercury is the form widely found in the water environment, and it bio-accumulates in the food chain (for recent review see Gochfeld, 2003).

At present, recommended maximum concentrations for mercury in livestock drinking water is set at 3 µg/L (CCME, 2005). However, feed contribution to the overall intake of mercury needs to be defined (Table 9.11.1).

Table 9.11.1 Examples of dietary intake of mercury associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Mercury‡**		
Water Hg content (mg/L)	**Estimated Water Contribution to Total Dietary Mercury Intake (mg/day)**	**Estimated Contribution of Mercury From Normal Feed (mg/day)**	**Estimated Dietary Mercury Levels Generally Regarded as Safe and Dietary Mercury Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
0.003	**0.096 to 0.12**	**NA**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**NA**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**NA**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**NA**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25°C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
† Guidelines for water in livestock (CCME, 2005).
NA=data not available

9.11.1 Evaluation of Risk

The various forms of mercury differ greatly in toxicological potency. Elemental mercury is poorly absorbed through the skin or gastrointestinal (GI) tract, but can volatilize readily, and mercury vapour can be efficiently absorbed in the lungs. Inorganic mercurial salts vary in solubility and absorptive properties. Most organic mercurial compounds are readily absorbed through the lungs and GI tract, and some are readily absorbed through the skin.

All mercury compounds are toxic to humans and animals, but the organic forms, particularly methyl-mercury and dimethyl-mercury, have the highest toxicity. Methyl-mercury is the form found most widely in nature, and this form is of a major toxicological concern because it bio-accumulated readily in the food chain.

Methyl-mercury is the form to which the risk of exposure is greatest under practical circumstances. However, it is important to understand that farm animals can be exposed to mercury not only from drinking water, but also from air, soil, and feedstuffs. Fish concentrate mercury by direct uptake from the water, and by ingestion of contaminated food. In some species (particularly predatory fish), muscle mercury levels may be as high as thousands of times greater than the level of the water from which they were taken. If food-producing animals are exposed to mercury for a prolonged time, considerable amounts of mercury may accumulate in hair or feathers (Nelson *et al.,* 1971; Herigstad *et al.,* 1972). Undoubtedly, mercury in fish, hair and feathers could be a source of mercury in livestock.

Among the most important sources of mercury under practical feeding conditions would be associated with dietary supplements such as fishmeal, feathers, and hair. Therefore, in establishing guidelines for mercury in drinking water for livestock, thorough consideration must be given to total environmental exposure and dietary content of mercury, as well as the high potential of possible accumulation in the animal.

Health Effects: A high dietary intake of mercury from consumption of fish has been hypothesized to increase the risk of coronary heart disease in humans (Salonen *et al.,* 1995, Guallar *et al.,* 2002; Yoshizawa *et al.,* 2002). The Minamata catastrophe in Japan in the 1950s was caused by methyl mercury poisoning from fish contaminated by mercury discharges by a factory to the surrounding sea. Residents of the area were plagued with tremors, sensory loss, ataxia and visual field constriction.

This scenario is relevant to the potential risk in some farm animals' situation because fishmeal and fish oil are frequently used as dietary supplements. Acute poisoning in farm animals is possible under some specific exposure circumstances, but the risk under most practical situations is extremely low. Acute toxic signs include nausea, vomiting, severe gastrointestinal irritation and pain, shock, and cardiac arrhythmias. Death may occur, and is usually associated with uraemia, caused by damage to renal tissue.

Chronic, clinical or sub-clinical toxicity scenarios in farm animals are possible in areas where environmental exposure to mercury is high. However, the onset of chronic mercury toxicity is variable and slow. Although signs of chronic toxicity may be manifested in some animals, the risk of significant health effects is generally very low.

Differences in tolerance to organic mercury among sex and strain of chicks, swine, and rats have been reported (Miller *et al.,* 1970; Piper *et al.,* 1971; Parizek *et al.,* 1974). Studies with one broiler strain and three White Leghorn strains indicate genetic differences in the degree of tissue concentration of mercury from dietary fishmeals (March *et al.,* 1974). Signs of mercury poisoning were observed at 2 mg/kg in turkey, 8 mg/kg in cattle and 10 mg/kg in sheep (Palmer *et al.,* 1973).

Of note, the issue of mercury in livestock is not as much a problem from the perspective of animal health effects, but rather the perceived problem regarding exposure must receive considerable attention because of the potential risk of toxicity associated with consumption of animal products in the human population.

Production Effects: If total dietary mercury is already high, even relatively low levels of mercury in drinking water for livestock may increase mercury content in edible animal products to a level that may pose a human health risk.

Notably, chickens, turkeys, ducks, and pheasants tolerated 3.3 ppm supplemental dietary mercury without evidence of adverse effects, although increased tissue mercury has been shown at levels lower than this. Laying hens given 10 ppm mercury for 70 days accumulated 55 percent of the mercury in the eggs (Sell *et al.,* 1974). Cattle receiving only 0.48 mg/kg of methyl-mercury compound per day accumulated 100 mg/kg in the kidney within 27 days, whereas sheep accumulated 120 to 210 mg/kg under the same conditions (Palmer *et al.,* 1973).

The mercury content of cows' milk can range from 3 to 10 ppb (Mullen *et al.,* 1975; Roh *et al.,* 1975). At 24 days following an 8-day exposure, goat's milk had 1.22 and 0.22 percent of total oral dosages, respectively, of organic and inorganic mercury (Sell and Davidson, 1975).

Exposure to mercury of livestock can have a detrimental effect on reproductive success. Male reproductive effects associated with mercury include impaired spermatogenesis and sperm motility. In females, mercury increases fetus resorption and induces abortion. Oral administration of methyl-mercury during gestation or lactation may cause developmental problems (Nielsen and Andersen, 1995).

Metabolic Interactions: Excess dietary selenium and zinc may provide some protection against toxicity of mercury (Potter and Matrone, 1974, Chapman and Chan, 2000; Zalups and Lash, 1994). Some studies suggested that simultaneous equimolar ratios of selenium and mercury are necessary to prevent toxicity of either one (Ganther and Sunde, 1974; Moffitt and Clary, 1974). Mercury toxicity is enhanced in zinc deficient animals.

Vitamin E has been shown to protect against the toxic effects of methylmercury in Japanese quail (Kling *et al.,* 1985; Welsh and Soares, 1975) and rats (Welsh, 1979).

Table 9.11.2 Summary of practical information relevant to mercury exposure in livestock

Guidelines	Interactions		Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Nutrients Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
3 µg/L	Dietary selenium, zinc, and Vit. E may have protective effect against toxicity of methyl mercury and mercuric mercury.	Inorganic mercury is converted to organic compounds, such as methyl mercury, which is very stable and accumulates in the food chain. Methyl mercury is the form found most widely in nature, and this form is of a major toxicological concern because it bio-accumulated readily in the food chain. Methyl mercury is the form to which the risk of exposure is greatest under practical circumstances.	Acute toxic signs include nausea, vomiting, severe gastrointestinal irritation and pain, shock, and cardiac arrhythmias. Death may occur, and is usually associated with uraemia, caused by damage to renal tissue.	Chronic, clinical or sub-clinical toxicity may occur in farm animals in areas where environmental exposure to mercury is high. The onset of chronic mercury toxicosis is slow. The risk of health effects in livestock is generally very low.

†(CCME, 2005). Farm animals can be exposed to mercury not only from drinking water, but also from air, soil, and feedstuffs.

9.11.2 Water Types or Conditions Where High Levels Occur

Mercury is a natural element that can be found in small concentration in many rocks. Its unique properties makes it attractive for consumer products and only recently has been banned from items such as mercury switches. As it has been used for centuries for various purposes, it can be found in the air, soil and water.

Background levels in water are generally low unless there has been contamination. In Saskatchewan, mercury levels are almost always below detection limits in the water and therefore are often not analyzed. The Saskatchewan Watershed Authority Rural Water Quality Data Base tested 50 sites and found all had mercury levels below the detection limit of 0.05 µg/L.

9.11.3 Management Considerations

Drinking water is one of the many possible exposure sources of mercury in farm animals. However, it is important to understand that generalized water contamination through industrial emissions, accidental spills, and intensive agricultural practices can increase mercury levels in drinking water sources rapidly. Therefore, regular monitoring of mercury levels in drinking water for farm animals is highly recommended in areas where the risk of potential contamination is high.

9.11.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.12 Nitrate and Nitrite

Nitrate and nitrite are oxidized forms of nitrogen. These compounds occur naturally in waters, although nitrate generally predominates. Nitrate is usually present in unpolluted streams at very low, usually less then 1 mg/L, levels (Meybeck 1982).

The recommended levels of nitrates and nitrites in water for livestock, according to present Canadian guidelines for livestock drinking water, are 100 mg/L nitrate (22 mg/L as nitrate-N); 10 mg/L nitrite (3.0 mg/L as nitrite-N) (CCME, 2005).

Confusion can arise concerning guideline values for nitrate and nitrite, because concentrations are sometimes reported on the basis of their respective nitrogen (N) content, that is, as nitrate Nitrogen (NO_3 Nitrogen) and nitrite Nitrogen (NO_2 Nitrogen). Generally one can assume that nitrates and nitrites are not referring to the nitrogen content unless it is specifically stated.

The levels of nitrate expressed as NO_3 and expressed as NO_3 nitrogen (NO_3-N) and corresponding guidelines recommended by NRC are listed in Table 9.12.1.

Table 9.12.1 Effects of various levels of nitrates on cattle

Nitrate Ion (NO_3 mg/L)	Nitrate Nitrogen (NO_3-N mg/L)	Guidelines
<44	<10	Safe for consumption by ruminants
45-132	10-20	Generally safe in balanced diets with low nitrate feeds
133-220	20-40	Could he harmful if consumed over long periods
221-660	40-100	Cattle at risk; and possible death
661	>100	Unsafe-possible death; should not be used as a source of water

SOURCE: National Research Council (1974).

Much of the values commonly accepted in the guidelines were derived from older, and, fragmented, studies. The recommended values are extrapolated from a range of findings.

Winks (1963) reported death of calves and cattle drinking water containing 2200 mg/L nitrate. He suggested a toxic nitrate concentration for cattle as somewhere between 300 mg/L and 2200 mg/L. In dairy cows, nitrate concentrations up to 180 mg/L in drinking water did not increase the concentration of nitrate in milk (Kammerer *et al.,* 1992).

It is generally assumed that nitrate concentrations less than 400 mg/L in livestock drinking water should not be harmful to animal health. Livestock may tolerate higher nitrate concentrations in drinking water provided nitrate concentrations in feed are not high. Depending on the nitrate content of feed, the type of livestock and other factors such as animal age and condition, concentrations up to 1500 mg/L nitrate may be

tolerated, at least for short-term exposure. Concentrations of nitrite exceeding 30 mg/L may be hazardous to animal health.

Comments: A safe level for nitrate ion (NO_3) is less than 44 mg/L and, for nitrate-nitrogen (NO_3 N) in water, is less than 10 mg/L. However, it is notable that there is a wide range of levels cited in the literature that have been shown to be associated with potential harmful effects.

There are several reasons why there is a wide range of levels in the guidelines. Much of the data that is included in the guidelines is derived from research papers, and the variability of results is among the key reasons for this wide range of derived values. One of the main reasons why scientific papers provide such very variable data is that there has been a lack of uniformity in experimental approach among various publications. In most cases, the outcome of experiments may have been influenced by factors associated with animals (species, breed or strain, production level, physiological status, etc), nutritional factors (feed and water), climatic, agricultural and industrial factors.

All the above listed factors can have tremendous impact on the risk of adverse effects. The same levels of nitrates in the water may produce toxic effects in some situations, but have no impact on health in other situations. For instance, in ruminants, nitrates have a high inherent toxic potential, but the compounds that are actually outright toxic are nitrites.

The rate of nitrate reduction in the rumen can be dependent on numerous nutritional and physiological factors. In essence, it is the systemic nitrite reducing activity that will primarily predetermine whether an animal will tolerate a certain level of nitrate or will show signs of toxicity. Therefore, it is not necessarily the level of nitrate in water or feed, but rather the rate of nitrite synthesis in the rumen, that will have a major influence on the outcome. Also, an important issue is that the true background levels of nitrites are rarely known in both feed and water upon routine analysis.

At best, the current water quality recommendations are based on very fragmented and, more importantly, outdated research. The major problem is that the current guidelines do not take into consideration several very important variables such as physiological status of the animal, developmental stage, age, nutritional status, and species differences.

9.12.1 Evaluation of Risk

Groundwater may contain elevated nitrate concentrations due to natural processes, but more typically, high nitrate concentrations in groundwater sources are associated with contamination. High concentrations of nitrates and nitrites in both ground and surface water are often associated with excessive use of nitrogen fertilizers, excessive application of manure, run-off from livestock holding areas, or leakage from septic systems and municipal waste.

Nitrate and Nitrite

Frequently, water sources in the vicinity of intensive livestock operations may have elevated levels of nitrates and nitrites. Elevated nitrite concentrations typically are found only under conditions where the source is polluted by organic wastes and oxygen levels are very low.

Table 9.12.2 Examples of dietary intake of nitrate associated with water and feed in a generic animal representing cattle.

Guideline for Water†		Guidelines for Dietary Nitrate		
Water Nitrate content (mg/L)	**Estimated Water Contribution to Total Dietary Nitrate Intake (mg/day**	**Estimated Contribution of Nitrate From Normal Feed (mg/day)**	***Dietary Nitrate Levels Consideration For Risk of Adverse or Toxic Effect % of diet DM or (mg/kg of diet DM)**	
100	**NA**	**NA**	**Potentially Toxic Levels** *(high risk of metabolic disturbances and/ or overt health problems)*	**0.5 % (> 5,000)**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
† Guidelines for water in livestock (CCME 2005).
NA=data not available
* Mineral Tolerance of Animals, 2005. National Research Council.

Excessive fertilization of plants with nitrogen fertilizers, or animal manure rich in nitrogen may lead to excessive nitrate accumulation in plants. Nitrates can accumulate in some grasses and barnyard weeds (pigweed, lambs quarters, kochia) at very high levels. Plants under stress (e.g. from frost, heat stress, drought, lack of adequate nutrition or sunlight, etc.) may also accumulate nitrate. Nitrate/nitrite toxicity in cattle and sheep has been associated with plants (McKenzie *et al.,* 2004).

Since some plants may contain high levels of nitrates, the dietary load may be increased. Animals are likely to be at higher risk of nitrate/nitrite poisoning through consumption of pastures, forages and feeds containing high levels of nitrate than from drinking water.

Nitrate in the water can change abruptly, and depends on numerous climatic, environmental, and agricultural factors. Therefore, analysis of water should be performed on a regular basis. However, it is important to note that if nitrate levels in the water supply are high, this may indicate that nitrate levels in locally grown feed may also be elevated. In the situation of suspected nitrate toxicity in livestock, a thorough assessment of total dietary nitrate/nitrite burden from both feed and water sources must be taken into consideration.

Both nitrate and nitrite can cause toxicity. However, nitrite is considerably more toxic than nitrate (Case 1963). To cause toxicity, nitrate must first be reduced to nitrite. Nitrate can be reduced to nitrite in the rumen by bacteria. For this reason, ruminant livestock is more susceptible to nitrate poisoning than mono-gastric animals. Non-ruminants (pigs and chickens) are less susceptible because they rapidly eliminate nitrate in the urine.

Ruminant animals previously fed high nitrate diets show an increased rate of nitrate/nitrite reduction. Nitrate toxicity is also dependent on the rate of consumption, with a slow intake and a balanced ration reducing toxicity (Crowley 1985). Ruminants fed high carbohydrate diets are more tolerant of forages with high nitrate levels. Because the nitrate reducing environment in the rumen may change, nitrate (relatively less toxic) in some instances can be rapidly reduced to nitrite (highly toxic).

As ingestion of nitrite leads to a more rapid onset of toxic effects than nitrate, the guideline values for nitrite must be correspondingly lower than that for nitrate. The total dietary intake of nitrate by livestock needs to be considered when interpreting the acceptable safety limits for water nitrate.

Nitrite is absorbed into the blood where it converts haemoglobin to methaemoglobin, and, because of this interaction with haemoglobin, blood has reduced oxygen carrying capacity. Lack of oxygen in blood will inevitably lead to tissue deprivation of oxygen. Prolonged insufficiency of oxygen for normal biochemical reactions may lead to serious metabolic derangements, and, in more severe cases, death.

Health Effects: The clinical signs of acute nitrate toxicity vary according to specific metabolic characteristics of the species. In general, ruminant animals most likely would develop methemoglobinemia, while monogastric animals would exhibit severe gastritis.

The key symptoms of acute nitrate or nitrite poisoning are gasping for air, laboured breathing, rapid pulse, frothing at the mouth, convulsions, blue muzzle and bluish tint around the eyes, and chocolate-brown blood. Mild to moderate levels of nitrate exposure have been incriminated in poor growth, infertility problems, abortions, vitamin A deficiencies, but research has not always substantiated these claims (Crowley *et al.,* 1974; Stuart and Oehme, 1982).

Nitrate ingestion has also been linked to impairment of thyroid function, decreased feed consumption, and interference with vitamin A and E metabolism. Hematologic changes seen with chronic high nitrate exposure include both compensatory increases in red blood cells and anemia, along with increased neutrophils and eosinophils.

Nitrite affects the metabolism of sulfonamide drugs in animals such as the pig, guinea pig, and rat. The N-nitroso compound dimethylnitrosamine may cause toxic hepatosis in cattle and sheep. Nitrosamines have been reported in cows' milk and have been found

to pass into the milk of goats under experimental conditions (Bruning-Fann and Kaneene, 1993).

An association between exposure to nitrates in drinking water and spontaneous abortions, intrauterine growth restriction, and various birth defects has been suggested. However, nitrates may be just one of the contaminants in drinking water contributing to adverse outcomes.

A recent review of the literature indicates that there is no epidemiological evidence of a direct cause-effect relationship between drinking water nitrate level and adverse reproductive effect (Ward *et al.,* 2005, Manassaram *et al.,* 2006).

There is no evidence that nitrate or nitrite ingestion may be a cause of teratogenic effects. Adverse reproductive effects reported occurred at doses that were about one thousand times and higher than the estimated human intake. There is no data available relative to livestock reproductive effects of nitrate or nitrite ingestion. Neither nitrate nor nitrite in experimental animals concentrated in the mammary gland or milk.

It has to be remembered that exposure to nitrates/nitrites can be lethal. Unfortunately, acute nitrate toxicity may be not recognized generally until some deaths have occurred. Therefore, in any suspected nitrate poisoning, veterinary assistance should be requested immediately. Administration of a solution of methylene blue may prevent death of the affected animal if the poisoning is not too far advanced. Since the absorption of nitrates/nitrates from the rumen may continue for some time, the status of the animal must be monitored, and treatment may need to be repeated as required. Mineral oil can be administered orally and may help to reduce the absorption of nitrates, as well as protect mucous membranes form irritation.

Production Effects: It is common that water quality guidelines provide levels that are safe for consumption. However, based on the literature it is difficult to define exactly how “safe level” should be understood. In most cases, the common understanding of “safe” means how much of the contaminant an animal can tolerate without overt signs of toxicity. In this context, there is a lingering question as to whether the water quality guidelines based on tolerance levels are appropriate for the modern livestock industry.

Most certainly, the success of the modern livestock industry is dependent on performance, therefore setting standards based on what levels the animal may tolerate without showing signs of toxicity may not be adequate to ensure that there is no effect on production parameters. In the contemporary livestock industry even subtle effects on performance may significantly affect the bottom line. Therefore, it would be more practical if the guidelines for nitrate levels in the water set for livestock were based on protection from methemoglobinemia under various loads of total dietary nitrate.

There is no systematic study that would clearly define the dose-effect relationship in livestock. Consequently, the levels of nitrates causing subtle adverse effects

associated with metabolic disturbance and possibly affecting production are not clearly defined for livestock.

A recent study by Zaki *et al.,* (2004) showed that in experimental animals after a 5-month treatment, nitrate at levels 150 and 500 mg/L induced a significant decrease in the serum level of thyroid hormones. Also, nitrate induced a dose-dependent increase in the weight of the thyroid gland and histological changes of the thyroid gland. This suggests that nitrate in drinking water may affect function of thyroid hormones, which in turn, may negatively affect the growth rate.

Epidemiologic data have suggested an association between developmental effects in offspring and the maternal ingestion of nitrate from drinking water, but a definite conclusion on the cause and effect relationship cannot be drawn. Experimental data have shown reproductive toxicity associated with high exposure levels to nitrate or nitrite, which are not likely to be encountered in drinking water.

Since highly producing animals have higher requirements for water, the potential of adverse effects that may occur at lower levels of contaminant concentration, but at higher levels of water consumption, cannot be excluded.

Metabolic Interactions: Excess intake of nitrates only affects the animal's capacity to absorb oxygen. There are no known substances that aggravate or mitigate the effect of excess nitrate consumption.

9.12.2 Water Types or Conditions Where High Levels Occur

Generally high concentrations of nitrates are normally associated with contamination. Improperly sealed wells combined with intensive livestock operation are likely the most common cause of contaminated groundwater. Permeable soils with a shallow groundwater table in either intensively farmed land, intensive livestock operations or septic tank infiltration fields are other scenarios that can result in contaminated groundwater.

Contamination of surface water by a fertilizer spill, or sewage or manure contamination can occur but the high levels of nitrates are generally short-lived as the nitrate is rapidly utilized by microorganisms that consume the oxygen in the water causing it to become anaerobic. This process effectively changes nitrate into nitrogen gas which then gases off into the atmosphere.

Table 9.12.3 Nitrate Levels in Saskatchewan Groundwater

Nitrate NO_3 (mg/L)	Nitrate N (mg/L)	Number of Samples Analysed	Percent of Total
<10	<2.3	2114	73.1
10 to 30	2.3 to 6.8	314	10.8
30 to 100	6.8 to 23	285	9.8
100 to 300	23 to 68	130	4.5
>300	>68	51	1.8

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.12.3 Treatment Technology

Nitrate removal options include:

- Nano-Filtration or RO membranes
- Ion exchange resins using nitrate selective resins
- Biological process

See Section on water treatment for further discussion on specific treatment systems.

9.13 Salinity, Total Dissolved Solids (TDS) or Total Soluble Salts (TSS)

Salinity, TDS, and TSS are all measures of water-soluble constituents commonly used in North America. Components associated with salinity are bicarbonate, sulphate, calcium, magnesium and silica, and, a secondary group (lower concentrations) of constituents including iron, nitrate, strontium, potassium, carbonate, phosphorus, boron and fluoride (Looper and Walder, 2002).

Total dissolved solids provide a measure of the total inorganic salts dissolved in water and is frequently used as a guide to water quality (Table 9.13.1).

Table 9.13.1 Guidelines of total dissolved solids (salinity) in drinking water (mg/L) for various classes of farm animals.

Animal	[1]Recommended	[2]Maximum	[3]Tolerance Limits
Sheep	5,000	5,000–10,000	10,000–13,000
Beef cattle	4,000	4,000–5,000	5,000–10,000
Dairy cattle	2,500	2,500–4,000	4,000–7,000
Horses	4,000	4,000–6,000	6,000–7,000
Pigs	4,000	4,000–6,000	6,000–8,000
Poultry	2,000	2,000–3,000	3,000–4,000

Adapted from Australian and New Zealand Guidelines for Fresh and Marine Water Quality 2000.
[1]some minerals may be beneficial; [2] no overt problems under normal feeding practices,
[3]concentration that may be safe for limited periods.

Undoubtedly, essential elements in water such as iron, copper, magnesium, manganese, sodium, selenium, may be desirable even if present at a relatively high concentration, because they can be utilized as nutrients. However, in practice, water as a source of essential minerals is rarely (if at all) considered by nutritionists. Therefore, it is important to understand that the classification of levels as desirable, maximum, or tolerable will grossly depend on water intake, the type of feed, and ultimately the total dietary burden of minerals from feed and water. Any particular mineral that constitutes the overall salinity value in water may cause adverse effects if the levels in the diet are already high.

Notably, from the table above it can be surmised that tolerance to TDS varies widely depending on classes of farm animals. It is also noteworthy that among ruminant animals, dairy cattle are least tolerant to TDS. Sheep and goats have a greater tolerance of dissolved salts than cattle. Poultry appears to be the least tolerant. Research findings comparing the effects of high-saline waters on performance of dairy cows have been variable.

These differences in sensitivity to salinity are most likely reflective of specific metabolic demands of animals. For instance, because water metabolism and intake is directly

linked to milk production, dairy cattle are more sensitive to intake of ions present in water. The main ionic components contributing to "salinity" of natural sources are most likely the high content of ions such as sodium, chloride, and sulphate. These ions in water may have a major impact on a highly producing animal's acid-base homeostasis. The study of Sanchez *et al.,* (1994) indicated that high intakes of chloride and sulphate affect milk production during summer months. Another study compared water dissolved solids from sodium chloride at 196 mg/L and 2,500 mg/L. Lactating cows consuming water with a high salt content increased water intake by 7 percent and exhibited a tendency for lower milk yield and DMI compared to the cows consuming low-saline water (Jaster *et al.,* 1978).

Reduction of TDS in water from about 4,400 to 440 mg/L resulted in a 20 percent increase in milk production, water intake, and feed intake (Challis *et al.,* 1987). A study using Holstein cows, producing milk at over 30 kg/day, showed that cows consuming desalinated water consumed 11 kg more water per day and produced 2.2 kg more milk per day than cows consuming salty water (Salomon *et al.,* 1995). However, according to Bahman *et al.,* (1993) there were no differences in milk production in cows drinking natural saline water (TDS at 3,574 mg/L) and desalinated water (TDS at 449 mg/L).

With regard to highly producing dairy cattle, the guidelines for salinity ought to be considered according to the production status.

Table 9.13.2 Guidelines for use of saline waters for dairy cattle

TDS Level (mg/L)	Recommendation
<1,000	Safe and should pose no health problems. Presents no serious burden to livestock.
1,000-2,999	Generally safe but may cause a mild temporary diarrhea in animals not accustomed to the water.
3,000-4,999	Water may be refused when first offered to animals or cause temporary diarrhea. Animal performance may be adversely affected. .
5,000-6,999	These waters should be avoided for pregnant or lactating animals. May be offered with reasonable safety to animals where maximum performance is not required.
>7,000	These waters should not be fed to cattle. Health problems and/or poor production will result.

SOURCE: National Research Council, 1974; Looper and Waldner 2002, based on National Research Council 2001.

The recommendations listed in Table 9.13.2 above should be interpreted critically, because most of the information on which these recommendations are based was derived from older research. Looking at the issue from a long term perspective, it appears that the tolerance of livestock to TDS has been declining. Of interest here are some examples of historical data from the 1930's and 1940's where it was found that dairy cows were able to adapt to survive on water containing 15,000 ppm (Heller, 1933), or 7000 to 10,000 ppm TDS has been used without any effect on milk production (Frens 1946). It is possible that in the past animals were more tolerant to TDS simply because their production was also lower.

There is evidence that the tolerance of modern, highly producing, animals is much lower, and may depend not as much on total salinity, but rather on individual components. For example, TDS values of 1,000 - 2,999 listed in the table above as generally safe can cause a wide range metabolic effects, affecting both health and performance if the major constituent of the total salinity is sulphate. This issue will be discussed at length later in a chapter devoted to sulphur.

In view of current knowledge, water quality parameters such as Salinity, Total Dissolved Solids or Total Soluble Salts provide very little, if any, information that would be of patho-physiological or toxicological relevance.

TDS may or may not have an impact on organoleptic properties of water and reduce water intake. However, the recommendations regarding suitability of water quality for use in any class of livestock should not be based on the values of TDS alone, even if the water appears to be palatable.

9.13.1 Water Types or Conditions Where High Levels Occur

Aquifers in Saskatchewan vary in their content of water soluble salts. Some large aquifers can vary significantly with location and age of the water. In general, surface water is much lower in TDS than groundwater, but the occasional lake or dugout may be recharged by groundwater and have a high TDS level. During drought periods, the water in the dugout may drop to a level below the groundwater table and high TDS water may seep in. When this happens, the water quality can change drastically over a matter of weeks from a source of good quality water to water that is unfit for livestock consumption. The highest TDS level recorded in the Saskatchewan Watershed Authority Rural Water Quality Data Base is 11,300 mg/L.

Often soluble salts are measured by a conductivity meter reading mS/cm. Measuring conductivity is a simple and inexpensive method of estimating the TDS. The conversion factor from conductivity to TDS usually varies from 0.54 to 0.96 depending on the chemical composition. A value of 0.67 is often used as an approximation if the actual factor is not known (TDS in mg/L ≈ 0.67 x Conductivity in µS/cm).

Table 9.13.3 TDS Concentration in Saskatchewan Groundwater

TDS Content (mg/L)	Number of Samples Analysed	Percent of Total
<500	215	7.4
500 to 1000	844	29.2
1000 to 2000	1088	37.6
2000 to 3000	511	17.7
3000 to 4000	159	5.5
4000 to 5000	41	1.4
>5000	35	1.2

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Table 9.13.4 Specific Conductivity Levels in Saskatchewan Groundwater

Specific Conductivity (µS/cm)	Number of Samples Analysed	Percent of Total
<1500	1414	48.9
1500 to 4000	1346	46.5
4000 to 7000	123	4.2
>7000	10	0.4

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Table 9.13.5 TDS Levels in Saskatchewan Surface Water

TDS Content (mg/L)	Number of Samples Analysed	Percent of Total
<500	170	54.5
500 to 1000	80	25.6
1000 to 2000	35	11.2
2000 to 3000	12	3.8
3000 to 4000	7	2.2
4000 to 5000	0	0.0
>5000	8	2.6

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.13.2 Treatment Technology

TDS removal is best accomplished by nano-filtration or RO membranes. See Section on water treatment for further discussion on specific treatment systems.

9.14 Selenium

Much of the toxicity research related to selenium has been based on the effects of plant species that are classified as "selenium accumulators". These plants may contain very high selenium levels, and, when consumed by livestock, may cause acute toxicity and a syndrome described as the blind staggers.

The CCME water quality recommendation for selenium in livestock is 50 µg/L, but at this level water contribution to the total selenium intake can be substantial and total dietary selenium intake should be monitored (Table 9.14.1).

Table 9.14.1 Examples of dietary intake of selenium associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Selenium‡**		
Water Se Content (mg/L)	**Estimated Water Contribution to Total Dietary Selenium Intake (mg/day)**	**Estimated Contribution of Selenium From Normal Feed (mg/day)**	**Estimated Dietary Selenium Levels Generally Regarded as Safe and Dietary Selenium Levels Consideration for Risk of Adverse or Toxic Effect (mg/day)**	
0.05	**1.6 to 2**	**2.0 to 2.55**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**2 – 4**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**4.1 – 6**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>6**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
† Guidelines for water are based on CCME 2005 recommendation.
‡Values for feed are adopted from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.
Note 2: Salt or Mineral Supplements are not included in estimates of selenium in feed.

9.14.1 Evaluation of Risk

Selenium is routinely supplemented in the diet, most often without prior knowledge of basal levels of selenium in the diet. In calculations of selenium requirements in the diet, water selenium content is rarely, if at all, taken into consideration. In this context, the contribution of water containing 50 µg Se/L (CCME water quality recommendation) to the total dietary burden of Se may be grossly underestimated. As demonstrated in

Table 9.14.1, at this level, water selenium intake can increase the total burden of dietary selenium to levels considered as excessive.

The maximum tolerance of Se commonly cited in literature for all livestock is 2 ppm (NRC, 1980), but in view of recent research this assumption must be evaluated critically (NRC, 2005). For instance, in ruminants, the condition “blind staggers” was historically thought to be caused by Se toxicity, but the research of O'Toole and Raisbeck (1995) questioned this. These authors found that dietary exposure for 4 months to 0.15, 0.28, and 0.8 mg Se/kg body weight in the form of selenomethionine and to 0.8 mg Se/kg in the form of sodium selenite did not produce neurological, renal, or hepatic lesions, supporting the contention that blind staggers is caused by factors other than excessive dietary selenium. It is noteworthy that exposure levels of 0.8 mg Se/kg body weight would be equivalent to dietary Se concentrations exceeding 25 mg Se/kg DM (25 ppm), which is considerably higher than the tolerance level of 2 ppm. This raises a question whether the previously established tolerance data was valid.

Furthermore, it has commonly been assumed that Se has a uniquely narrow margin between nutritionally required levels and those that are toxic, but the validity of this has also been questioned. Recent data from the University of Florida (Cristaldi *et al.,* 2005; Davis *et al.,* 2006) have shown that sheep tolerated over 10 ppm Se for relatively long periods of time.

Health Effects: The risk of acute toxicity *per se* associated with water selenium under normal management, is very low, if any. Susceptibility to selenium toxicity may vary substantially depending on species, age, nutritional status, and physiological status. Young animals are generally less tolerant in comparison to adults.

Poultry and fish appear to be more sensitive to teratogenic effects of selenium than other animals. A chronic syndrome commonly associated with Se toxicity has been described in cattle and sheep as alkali disease, with symptoms such as loss of vitality, emaciation, deformity and shedding of hoofs, loss of long hair, and erosion of joints of long bones. Interestingly, O'Toole and Raisbeck (1995) reproduced these symptoms, but only when using levels of 0.28 and 0.8 mg Se/kg of body weight, which represent rather high levels of Se exposure (equivalent to dietary concentrations of approximately 10 to 25 mg Se/kg DM).

The effects of long term-low level exposure are not known, particularly in livestock selected for high performance traits. In particular, the effects of long-term exposure on fertility and production parameters in livestock are poorly characterized.

Production Effects: Excess selenium has produced loss of fertility and congenital defects, thus in the practical field situation the contribution of excess selenium to the overall reproductive failure of livestock should not be underestimated.

The selenium concentrations in milk are particularly sensitive to high selenium intakes by cows. Values ranging between 0.16 and 1.27 mg/L have been reported for cow’s

milk from seleniferous rural areas in the USA (Rosenfeld and Beath, 1964). This may be an issue for the human consumer.

High levels of selenium in drinking water may be a factor limiting water palatability due to garlicky odour and astringent taste.

Metabolic Interactions: Mechanisms of toxicity and metabolic interactions remain unclear. Elements such as Ag, As, Cd, Ca, Cu, Hg, Pb, Zn, and S have been mentioned in the literature to interact with selenium. These compounds may reduce toxicity or induce deficiency of Se. Noteworthy is the natural antagonism between arsenic and selenium. Selenium shows some similarities with sulphur, and this may lead to substitution of S with Se in biologically active molecules, and this may lead to disruption of metabolic activities of these molecules.

Vitamin E deficiency may increase susceptibility of animals to selenium toxicity, whereas increased intake of vitamin E may increase tolerance to selenium. Monensin appears to enhance Se uptake, hence use of this compound should be monitored in the situation of Se overload.

Table 9.14.2 Summary of practical information relevant to Selenium exposure in livestock

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Short Term, Moderate or High Level Exposure**	**Long Term, Moderate or Low Level Exposure**
50 µg/L	Calcium, Copper, Manganese, Zink, Sulphur	Arsenic, Lead, Cadmium, Mercury, Silver	Compounds that interact with Se may reduce its toxicity. Substitution of Sulphur with Se in biologically active molecules may lead to disruption of metabolic activities.	The risk of acute toxicity associated with Se is generally very low.	Signs such as loss of vitality, emaciation, deformity and shedding of hoofs, loss of long hair, and erosion of joints of long bones. Selenium may produce loss of fertility and congenital defects. Milk selenium levels are particularly sensitive to selenium intake by cows.

† CCME 2005 guidelines recommendation for selenium in livestock is 50 µg/L, but at this level water may likely contribute to the overall body burden of selenium, if feed selenium levels are already marginally high. The maximum tolerance of Se for all livestock was set at 2 ppm in 1980 (NRC, 1980).

9.14.2 Water Types or Conditions Where High Levels Occur

Selenium is found in low concentrations in soil and rocks. Soils do have a higher concentration of selenium than rocks and often higher selenium concentrations are found in shallow aquifers. Shallow wells generally have a higher concentration of selenium than deeper wells, so it is speculated that the source of the selenium may be primarily soils. In Saskatchewan there also appears to be a higher concentration of selenium in the groundwater in the Southwest part of the province.

In Saskatchewan, only 3 percent of the groundwater samples exceeded the Canadian Water Quality Guideline for livestock of 50 µg/L (Saskatchewan Watershed Authority Rural Water Quality Data Base).

Table 9.14.3 Selenium Levels in Saskatchewan Groundwater

Selenium Content (µg/L)	Number of Samples Analysed	Percent of Total
<10	2652	89.7
10 to 20	105	3.6
20 to 50	112	3.8
50 to 100	47	1.6
100 to 200	22	0.7
200 to 500	19	0.6
>500	1	0.03

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.14.3 Management Considerations

Selenium overload can be managed through the following measures: 1) modification of the diet to balance total Se intake, 2) dietary intervention aimed at limiting selenium absorption and increasing excretion, and 3) treatment of the soil to reduce selenium uptake by plants. Also, the natural antagonism between arsenic and selenium can be used in management strategies for problems associated with an excess of selenium.

9.14.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.15 Sodium

Sodium is widely distributed in the water environment, but its content varies considerably depending on regional and local hydrological and geological conditions, the time of year, and industrial salt utilization patterns (e.g. for snow removal or de-icing, food/feed processing, etc.). Large amounts of salt used for road maintenance during winter will inevitably end up in the environment.

Sodium in drinking water sources occurs most commonly in association with sulphate or chloride ions, and the content of these ions should not be ignored. In particular, the sulphate ion may be a more important factor determining water quality than sodium itself.

Table 9.15.1 Examples of dietary intake of sodium associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Sodium‡**		
Water Na content (mg/L)	**Estimated Water Contribution to Total Dietary Sodium Intake (g/day)**	**Estimated Contribution of Sodium From Normal Feed (g/day)**	**Estimated Dietary Levels Generally Regarded as Safe and Dietary Sodium Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
1000	**32 to 40**	**11**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**9 – 26**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**27 – 85**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>85**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of sodium in feed.
† At present, there are no established guidelines for maximum concentrations for sodium in livestock drinking water. CCME sets an aesthetic objective of <200 mg/L for sodium in drinking water for humans. A value of 1000 mg/L was based on 98 percentile of groundwater in Saskatchewan being below this level.
‡Values for feed are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.
NA=data not available

9.15.1 Evaluation of Risk

Under normal physiological conditions, the body has very effective methods to control sodium levels, and therefore sodium generally is not considered to be a toxic element. In humans, the aesthetic threshold for sodium in drinking water is approximately 200 mg/L. The taste of drinking water is generally considered offensive at sodium concentrations above the aesthetic objective.

Health Effects: High levels of intake for prolonged periods of time may disturb normal homeostasis, potentially can lead to some forms of hypertension, congestive cardiac failure, renal disease, cirrhosis, toxaemia of pregnancy. Salt poisoning has been described under various circumstances in adult cattle. Signs of NaCl poisoning include gastrointestinal irritation with vomiting, diarrhoea, mucoid feces, abdominal pain, anorexia, thirst, salivation and polyuria. Nervous system signs include knuckling, blindness, muscular spasms, paresis and convulsions.

Adverse effects associated with sodium sulphate (Na_2SO_4) in drinking water depend on type of animals, total dietary intake of sulphur, and amount of water consumed. In ruminants, a disorder of the central nervous system, known as polioencephalomalacia, has been associated with high levels of sodium sulphate in drinking water. However, in cases where sodium in water is present as sulphate salt, the adverse effects are more likely associated with sulphate rather than sodium (for details see chapter on Sulphur).

Production Effects: At concentrations above 200 mg/L, sodium may reduce water palatability, which may result in lowered water intake. Sodium ion is an important component of acid-base homeostasis, and disturbance of the acid-base balance in highly producing animals may lead to metabolic consequences affecting performance. Lactating cows consuming water with a high salt content increased water intake by 7 percent and exhibited a tendency for less milk yield compared to cows consuming low-saline water (Jaster *et al.,* 1978).

Metabolic Interactions: The adverse effects of sodium in drinking water cannot be considered on a stand-alone basis. The sodium ion is one of the ionic components contributing to salinity (see chapter on salinity). Therefore, the most likely scenario to consider would be combined effects of ions such as sodium, chloride, and sulphate.

Table 9.15.2 Summary of practical information relevant to sodium exposure in livestock.

Guidelines	Interactions	Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
At present, there are no established guidelines for maximum concentrations for sodium in livestock drinking water CCME sets an aesthetic objective of <200 mg/L for sodium in drinking water for humans.	The effects of Na are difficult to separate from other ions such as chloride or sulphate since sodium in water does not exist in its pure state in water. With regard to sodium sulphate, sulphate is probably more important as a toxicant. On the other hand, while considering NaCl it is the Na+ ion that appears to be responsible for most of the recognized effects of "salt" poisoning. Metabolic effects are related to cellular dehydration, or "tissue shrinking", and edema.	Most animals can tolerate relatively large amounts of sodium, and responses are variable. Water containing 6726 - 6826 mg Na+/L resulted in a loss of condition, scouring and death in 15/220 cattle. Sodium chloride at a 10,000 ppm in drinking water can cause toxicity, and at 5,000 to 7,000 ppm NaCl in water can affect herd health and performance.	If abundant good quality drinking water is available, animals can tolerate large doses of Na. Cattle ingesting water containing 2500 mg NaCl/L (975 mg Na+/L) for 28 days showed increased water intake, decreased milk production and diarrhea.

†(Health Canada 2008).

9.15.2 Water Types or Conditions Where High Levels Occur

In ground waters, sodium concentrations normally range between 6 and 130 mg/L. Sodium concentrations in Canadian surface waters range from less than 1 mg/L to more than 2000 mg/L. In Saskatchewan the highest sodium concentration recorded in the Saskatchewan Watershed Authority Rural Water Quality Data Base for in groundwater and surface water is 2710 mg/L and 3840 mg/L respectively. The following table shows the frequency of various ranges of sodium in groundwater.

Table 9.15.3 Sodium Levels in Saskatchewan Groundwater

Sodium Content (mg/L)	Number of Samples Analysed	Percent of Total
<200	1997	69.0
200 to 500	593	20.5
500 to 1000	261	9.0
1000 to 2000	40	1.4
>2000	2	0.1

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Table 9.15.4 Sodium Levels in Saskatchewan Surface Water

Sodium Content (mg/L)	Number of Samples Analysed	Percent of Total
<200	292	93.6
200 to 500	9	2.9
500 to 1000	5	1.6
1000 to 2000	5	1.6
>2000	1	0.3

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.15.3 Management Considerations

Sodium-containing chemicals are used in various water-softening treatment systems, and this process can be an important source of sodium in drinking water. The lime-soda ash purification process may contribute significant quantities of sodium, if a large concentration of non-carbonate hardness must be removed. In domestic water softening systems using ion-exchange resins, for every 100 mg of calcium removed per litre of water, sodium concentration in the treated water will rise by 115 mg/L.

9.15.4 Treatment Technology

Treatment technology includes:

- Nano-Filtration or RO membranes

See Section on water treatment for further discussion on specific treatment systems.

9.16 Sulphate

Sulphur in water may be present in several different chemical forms. Sulphate is the most commonly occurring form of sulphur in drinking water for livestock, but in some water sources, due to highly reducing environment, sulphates may be reduced to sulphides. Among more common forms of reduced sulphur in some water sources is hydrogen sulphide, which gives drinking water this very characteristic scent associated with "rotten eggs". The sulphate ion is probably the most common contaminant of water sources for livestock in Canada, and especially in the Prairie provinces. The problems associated with excessive intake of sulphur have been intensively studied, but it appears that the importance of sulphur as a water quality issue is still not completely recognized at the field level.

High levels of sulphur in water can be detrimental in any class of farm animals, but ruminants are most susceptible. Higher levels of sulphur in drinking water can be tolerated by animals such as pigs or poultry, whereas relatively low levels can be detrimental to health and performance in cattle or sheep. For this reason the ensuing discussion will be focused predominantly on ruminant livestock.

The CCME guideline of sulphate at 1,000 mg/L is commonly cited as safe. Sulphur accounts for approximately 33.3 % of sulphate ion, hence at a level of 1000 mg/L of sulphate, every litre of water consumed will contribute approximately 333 mg of dietary sulphur. Indeed, at this level, sulphur in the water for most farm animals is not likely to present a toxicological problem, but in ruminant livestock this level may cause serious health problems, in particular when sulphur from water and dietary sources is considered, cumulative daily intake may be excessive, or in some situations toxic (for details see later). Table 9.16.1 demonstrates examples when cumulative intake of sulphur from water and feed may easily reach toxic levels even under apparently normal nutritional conditions.

9.16.1 Evaluation of Risk

Importance of Sulphate in Water in the Overall Dietary S Intake: From the perspective of water quality for farm animals, sulphur is probably the most significant water contaminant in ruminant livestock, having considerable impact on both health and performance. In many areas sulphur present in drinking water may be a major contributor to the overall intake of sulphur.

Drinking water is probably the most common source of excessive intake of S in livestock on many Canadian farms. A comprehensive study assessing the distribution of S content in feeds or in water in Canada has not been done. However, case study reports indicate that the problem is widely spread. Episodic information from various publications in Canada (Harries 1987, Boila 1988, McLeese *et al.,* 1991, Beke and Hironaka 1991, Olkowski *et al.,* 1991., Hamlen *et al.,* 1993, Hydack, 2003) indicate that some 20 to 40% of farms on the Canadian Prairies use drinking water containing more than 1000 ppm of sulphate. Based on our survey of several farms in Saskatchewan (Olkowski *et al.,* 1991),

some 25 to 30% of livestock operations use water with sulphate levels between 1000-1500 ppm, and in some 5 to 10% of examined farms the sulphate level in drinking water exceeded 3000 ppm. In a few instances, drinking water contained as much as 5000 to 7800 ppm of sulphate.

It has to be stressed that even relatively low levels of sulphur in water may have significant impact on total dietary sulphur intake, if the ration contains high levels of sulphur. High to excessive S concentrations in some plants occur naturally and can increase under a variety of soil management conditions (Boila *et al.,* 1987, Hardt *et al.,* 1991).

High concentrations of S are inherently present in a number of commonly used feedstuffs (NRC 1984), and subsequently excessive S content can be expected in the rations based on these ingredients. Table 9.16.2 shows several examples of feedstuffs containing high levels of S commonly used in ruminant rations.

Table 9.16.1 Examples of dietary intake of sulphur associated with water and feed in a generic animal representing cattle.

Guideline for Water†		**Guidelines for Dietary Sulphur‡**		
Water Sulphate content (mg/L)	**Estimated Water Contribution to Total Dietary Sulphur Intake (g/day)**	**Estimated Contribution of Sulphur From Normal Feed (g/day)**	**Estimated Dietary Sulphur Levels Generally Regarded as Safe and Dietary Sulphur Levels Consideration for Risk of Adverse or Toxic Effect (g/day)**	
1000 (333 mg/L S)	**10.7 to 13.3**	**16 to 20**	**Safe Levels** *(generally regarded as nutritionally balanced)*	**16 – 26**
			Excessive Levels *(possible risk of adverse metabolic effects)*	**27 – 32**
			Potentially Toxic Levels *(high risk of metabolic disturbances and/ or overt health problems)*	**>32**

Note 1: Assuming this generic animal is a beef cow (550 - 600 kg BW), in the third trimester of pregnancy, fed an average quality brome-alfalfa hay, with an ambient temperature of 20 to 25˚C, and would be eating 11 – 14 kg of feed dry matter, her water intake would be approximately 32 to 40 litres per day. Intake estimates taken from the CowBytes® ration balancing program.
Note 2: Salt or Mineral Supplements are not included in estimates of sulphur in feed.
† Guidelines for water are based on CCME 2005 recommendation.
‡Values for feed and dietary sulphur are from CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.

Table 9.16.2 Feedstuffs commonly used in ruminant livestock diets containing high concentrations of sulphur

Feed	Sulphur content % (DM)
Alfalfa	0.40
Extracted cotton seeds	0.34-0.56
Mangel beets	0.63
Sugar beets and their by-products	0.22-0.54
Soybean meal	0.49
Molasses	0.40-0.61
Rape seeds mechanically extracted	0.50
Sweet clover hay	0.47
Turnip	0.43
Yeasts	0.45-0.62
Wheatgrass	0.47
Dehydrate whey	1.12-1.15
Brewers dried grains	0.32
Wheat Distillers Dried Grains With Solubles (DDGS)	*0.44-0.65
Corn Distillers Dried Grains With Solubles (DDGS)	**0.31-1.9

NRC 1984,
* McKinnon, 2008 (Personal Communication).
.** Distillers Grains By-products In Livestock and Poultry Feeds, Nutrient Profiles Comparison Tables, University of Minnesota, http://www.ddgs.umn.edu/profiles.htm#us,
Note: In recent very dry years, in Saskatchewan, canola forage has been in use as feed for cattle. In this context it should be noted that canola forage may contain high levels of S, and thus may increase the risk of adverse effects.

In ruminant livestock, in order to assess the potential hazard associated with sulphur in water, the total intake of dietary sulphur must be taken into consideration.

An important consideration while assessing the risk of exposure is that sulphur intake by a ruminant animal depends on numerous dietary and environmental variables. The factors contributing to dietary sulphur may be extremely variable, and frequently difficult to control. As illustrated in Table 9.16.1, even under normal dietary conditions, water may be a significant contributor to the overall load of dietary S.

Dietary S at 0.4% has been recommended as the tolerance level (NAS 1980), but some sources suggest that even lower levels can be detrimental. According to Kandilis (1984), 0.3% of total dietary sulphur may cause adverse effects. Indeed, currently, the lower level appears more realistic in view of recent research findings. It is of interest to note that looking at the problems associated with dietary sulphur overload from an historical perspective, it is apparent that there is a trend indicating that the tolerance for excess dietary S continues to decline, as cattle are more and more selected for high performance characteristics.

Sulphate

Health Effects: The basic toxicity issues associated with sulphur have been studied in the past and the findings have been compiled in two major documents NRC (1974) and NAS (1980). However, the range of responses of ruminants to excess sulphur appears to be evolving. For instance, more recent research papers provided evidence that an excess of dietary sulphur in cattle and sheep causes the central nervous system disorder, cerebro-cortical necrosis (CCN), commonly also known as polioencephalomalacia (PEM). Further, the tolerance of ruminant livestock to sulphur has been decreasing over the last 3 decades. Based on personal observations since the mid 1980's, the number of outbreaks reported has been increasing over the last two decades, and these events tend to be more severe and affect a larger number of animals. Our more recent observations from the last 7 years (Olkowski *et al.,* unpublished observations) suggest the course of the disease is more acute, and mortality rates tend to be higher than in the past. The affected animals tend to die in early stages of the disease.

Acute death: In ruminants fed high levels of sulphur, sulphides in the rumen can be generated in considerable quantities. In experimental animals, death associated with excessive synthesis of sulphide in the rumen gas cap has been reported. However, such direct adverse effects associated with sulphur toxicity are not common.

Central Nervous System Disorder: In recent years, many reports implicated high levels of S in the drinking water as an etiological factor in S induced brain tissue necrosis commonly known as polioencephalomalacia or PEM (Harries 1987, Beke and Hironaka 1991, Olkowski *et al.,* 1991; Hamlen *et al.,* 1993; Gould, 1998, Peterson *et al.,* 2003; Hydack, 2003, Kul *et al.,* 2006, McKenzie *et al.,* 2008). In published reports, the morbidity and mortality associated with S induced brain lesions may be high. For instance, Peterson *et al.,* (2002) reported a 15 % incidence of PEM in cattle drinking water containing 3100 ppm of sulphates. This level of sulphate would contribute approximately 1 g of dietary sulphur per litre. Interestingly, in the recent study of Kul *et al.,* (2006) dietary sulphur at a level of 0.45% resulted in a massive outbreak of PEM.

Sulphur-related PEM may occur within 3 to six weeks following exposure to high sulphur water or diet. The course of the disease may be acute with rapid onset of signs such as blindness, recumbency, seizures, and frequently death; or sub-acute characterized by aimless wandering, head pressing, walking on obstacles due to visual impairment, and ataxia. The latter form may progress to a more severe form, with recumbency and seizures. Early treatment with thiamine may lead to recovery. The brains of animals that die of sulphur induced PEM show characteristic necrotic lesions in the cortical gray matter.

Production Effects: In recent years cattle are more likely to be affected by levels of dietary sulphur, which in the past, would not have had any effect. For example, the study of Zinn *et al.,* (1997) showed that sulphur in excess of 0.2% of dietary dry matter may have a detrimental effect on average daily gain, feed intake, and net energy value of the diet. Loneragan *et al.,* (2001) reported that sulphate concentrations greater than 583 ppm decreased feedlot performance as indicated by a reduction in average daily

gain, feed conversion and carcass characteristics. In contrast to this, in the study of Weeth and Capps (1972) water containing 1462 ppm of sulphates had no adverse effect on animal performance. Notably, the dietary contribution from water containing 1462 ppm sulphate could account for 0.2% of S intake without considering feed S content.

Several examples of recent research indicated that cattle exposed to excess dietary S perform poorly (Zinn *et al.,* 1997, Patterson and Johnson 2003, Patterson *et al.,* 2003). The production losses can be substantial. For instance, in a study on steers (Peterson et al, 2003), the average daily gain declined from 1.39 to 1.01 lb/day as the sulphates in drinking water increased from 400 to 3100 ppm.

Canadian guidelines for livestock suggest 1000 mg/L of sulphate. However, realistically, when water intake is high, sulphur intake with the drinking water containing 1000 ppm of sulphate alone may reach 0.3% dietary sulphur. As argued above, dietary intake of sulphur exceeding 0.3% may affect performance and create health hazard. In view of the recent research, Canadian guidelines for water sulphur need to be revised.

9.16.2 Metabolic Interactions

Specific Metabolic Aspects of Dietary Sulphur In Ruminants: The susceptibility of ruminant livestock to sulphur is directly related to specific metabolic features of these species. Because of the unique nature of sulphur metabolism, ruminants are at considerably higher risk of developing serious adverse reactions associated with excessive intake of sulphur. Therefore, the problems associated with sulphur in water for ruminants must be considered in the context of overall specific metabolic features of dietary sulphur.

Sulphur found in drinking water sources is most likely to occur as sulphate. In ruminants, almost all ingested sulphate is reduced to sulphide by rumen microbes. Sulphide is absorbed, and oxidised sequentially to sulphite and sulphate in the tissues, and sulphate is recycled to the rumen via saliva. Therefore, cycling of the ingested sulphur is an important component of metabolism, as well as potential adverse effects. Excess dietary S may cause a proliferation of sulphur reducing bacteria in the rumen, which may further increase the systemic pool of toxic S metabolites of dietary origin.

Excessive intake of sulphur may cause direct toxicity, but mostly the detrimental effects are associated with metabolic interference.

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Metabolic effects of high levels of dietary S are mostly associated with nutritional interaction. Excess dietary S interferes with the metabolism of several essential nutrients. These effects represent a very discrete class of nutritional S toxicity linked to specific features of S metabolism in ruminant species.

Metabolic Problems Associated with Sulphur-Nutrient Interactions: Experimental data indicate that vitamin B_1 (Thiamine) synthesis in the GI tract of ruminants is impaired by excess sulphur (Goetsch and Owens 1987, Olkowski *et al.,* 1993). Blood thiamine concentration was lower in cattle drinking high sulphate water (Gooneratne *et al.,* 1987, Olkowski *et al.,* 1991). In the situation of increased metabolic demand, thiamine deficit can occur in ruminants exposed to excess dietary S (Olkowski *et al.,* 1991).

The retention of both calcium and phosphorus was reduced by the addition of sulphate to diets (Tucker *et al.,* 1991), and this metabolic problem may be of importance in dairy cows.

Sulphate and thiosulphate inhibited the uptake of selenate (Turner *et al.,* 1990), and the possible involvement of sulphate in an increased incidence of muscular dystrophy was reported (Hintz and Hogue 1964). The effect of dietary S may be reversed by an increased supplementation of selenium (Pope *et al.,* 1979). Hence, the effect of S may be of more importance in cases of marginal adequacy of selenium.

Dietary sulphur may interact with several essential minerals. Research has shown that S, either alone or in a synergistic effect with molybdenum, can affect GI metabolism of copper, zinc, manganese, magnesium and phosphorus (Golfman and Boila 1990).

As evidenced by the research discussed above, sub-clinical effects associated with excessive intake of sulphur may represent a wide range of metabolic disturbances. In the vast majority of cases, problems resulting from excess dietary sulphur are associated with secondary metallic interaction of sulphur with essential nutrients. These effects are non-specific, secondary metabolic disturbances, and may be present as a plethora of non-specific metabolic disorders that may affect performance. The most prominent secondary metabolic effects are those associated with sulphur induced copper deficiency.

Sulphur Induced Copper Deficiency: The chronic effects of long term exposure to excess dietary S represent a very discrete class of nutritional adverse effects linked to the unique features of S metabolism in the ruminant species. Decreased bioavailability of copper is due to the formation of insoluble CuS, or if high levels of molybdenum are present along with high levels of sulphur, thiomolybdate-Cu complexes (for review see Gooneratne *et al.,* 1989).

Copper deficiency is likely the most prominent problem in cattle and sheep drinking high sulphur water. If the level of copper in the ration is marginal, animals may develop signs of copper deficiency within a few weeks. The problem is more severe if the diet is also high in molybdenum.

In essence, all signs characteristic for copper deficiency can be induced by excess dietary sulphur. However, signs of S induced copper deficiency may be variable as they depend on many metabolic variables and nutritional conditions.

Hair coat changes are among the most prominent signs indicative of possible copper deficiency (Figure 9.16.1).

The example demonstrated here represents a real field case from a SK farm where a number of animals from a commercial feedlot showed signs of poor performance that was traced to metabolic copper deficiency associated with high levels of sulphur in drinking water.

The picture on top demonstrates features typical of copper deficiency associated with high levels of sulphur in water. Notable are signs such as rough, poor quality hair, with faded color. This animal also shows signs of generally poor body condition with clear evidence of poor growth. Once the problem was identified, the herd was supplemented with copper. Within a few weeks, the entire herd showed signs of improvement. The picture on the bottom shows the same animal approximately 3 months after the copper supplementation was introduced. Notable are drastic changes in the quality and appearance of the hair coat.

Figure 9.16.1 Sulphur induced copper deficiency in beef cattle.

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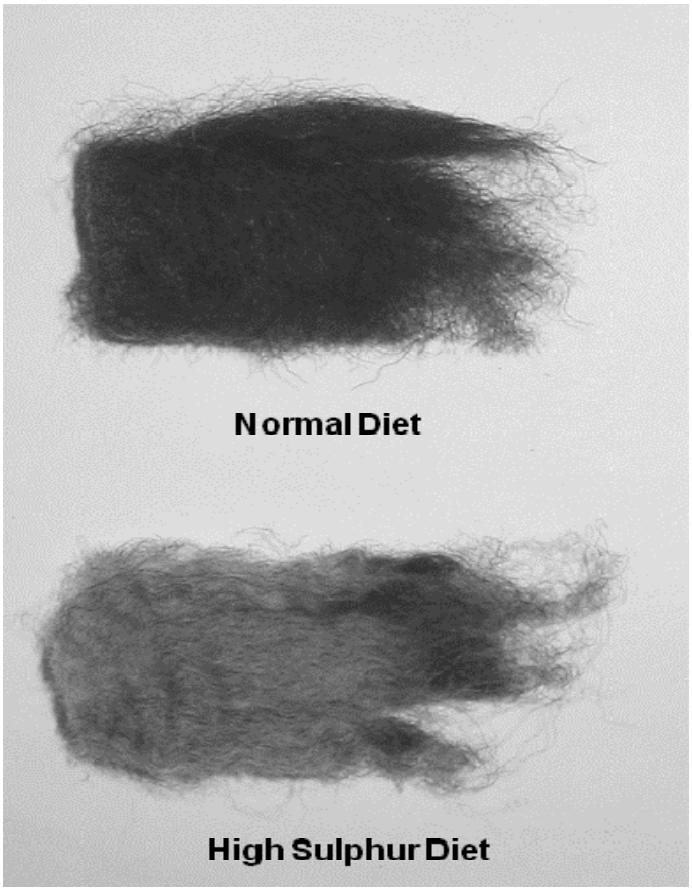
The signs of possible sulphur induced copper deficiency are clearly appreciable in sheep with black wool as demonstrated in Figure 9.16.2.



The photograph shows appearance of wool in an experimental animal that was initially fed a normal diet (top part) and subsequently when it was fed a high sulphur diet (bottom part). Diagnosis of copper deficiency was confirmed by low plasma copper level. Notably, prior to exposure to the high sulphur diet this lamb had normal, healthy, uniformly black and shiny wool. Just 6 wks after the animal was fed a high sulphur diet, the hair became rough and brittle. The change in wool color actually shows the history of metabolic changes where the tip is black (growth from the time when animal was fed normal diet), whereas below the color is gray (wool growth when the animal was fed high sulphur diet).

Figure 9.16.2 Change in wool appearance associated with sulphur induced copper deficiency in sheep.

As illustrated above, changes in the hair color where red hair turns yellowish, and black hair coat becomes brown or gray, are among the most recognizable signs of possible copper deficiency. Affected animals frequently show “spectacles” of faded hair around eyes.

Other signs of sulphur induced copper deficiency may include, scours, unthriftiness, reduced growth rate, weight loss, reduced fertility and delayed puberty, low conception and ovulation rates in cows, and reduced semen quality in bulls.

Retained placenta may also be a sign of secondary copper deficiency. Calves born from copper deficient cows, and young calves exposed to excess sulphur may display inability to suckle and in-coordination. Common features are stiff gait, heel cracks, sole abscesses, foot rot, which may be manifested as lameness. Cardiovascular disease and reduced immune response were also reported.

Copper Requirement in Cases of Sulphur Overload: Copper requirements may differ depending of the complexity of metabolic interactions. In most circumstances Cu - S interaction will be additionally complicated by other elements, with molybdenum and iron being the most likely factors. The copper, iron, molybdenum and sulphur contents of pastures and forages vary with the species, strain and maturity of the plant, the soil conditions and the fertilizers used (McFarlane *et al.,* 1990).

The feed form (e.g. hay, fresh grass, or silage) may influence the antagonisms among sulphur, copper and molybdenum, with sulphur *per se* having an enhanced influence in silages and both antagonists having reduced influence in hay, when compared with fresh grass (Langlands *et al.,* 1981, Suttle, 1977, 1983b) Suttle, 1974; Bremner *et al.,* 1987; Whitelaw *et al.,* 1979; Woolliams, C. *et al.,* 1986; Woolliams, *et al.,* 1986).

Mo and S have a very strong synergistic effect on reducing Cu availability by combining with Cu in the rumen to form an insoluble complex. In addition, high levels of Ca, Cd, Co, Fe, Hg, Mn, P, Pb, Se, Sn, and Zn may further complicate Cu utilization.

The effect of sulphur on copper metabolism can be further complicated by other elements known to affect copper homeostasis. Several of these elements such as iron, magnesium, manganese and calcium can be present in water in significant amounts along with high levels of sulphate.

Because of so many variable factors that may affect sulphur-copper interaction, it would be very difficult to accommodate all the variables in order to estimate copper requirement. Even if one considers the two factors that have the most prominent effect (i.e. synergistic effects of sulphur and molybdenum) the modeling becomes very complex. In order to illustrate the effects of molybdenum and sulphur on dietary copper requirements, we compiled relevant data from various publications. The relative changes in copper requirements associated with various levels of dietary sulphur and molybdenum are presented graphically in Figure 9.16.3.

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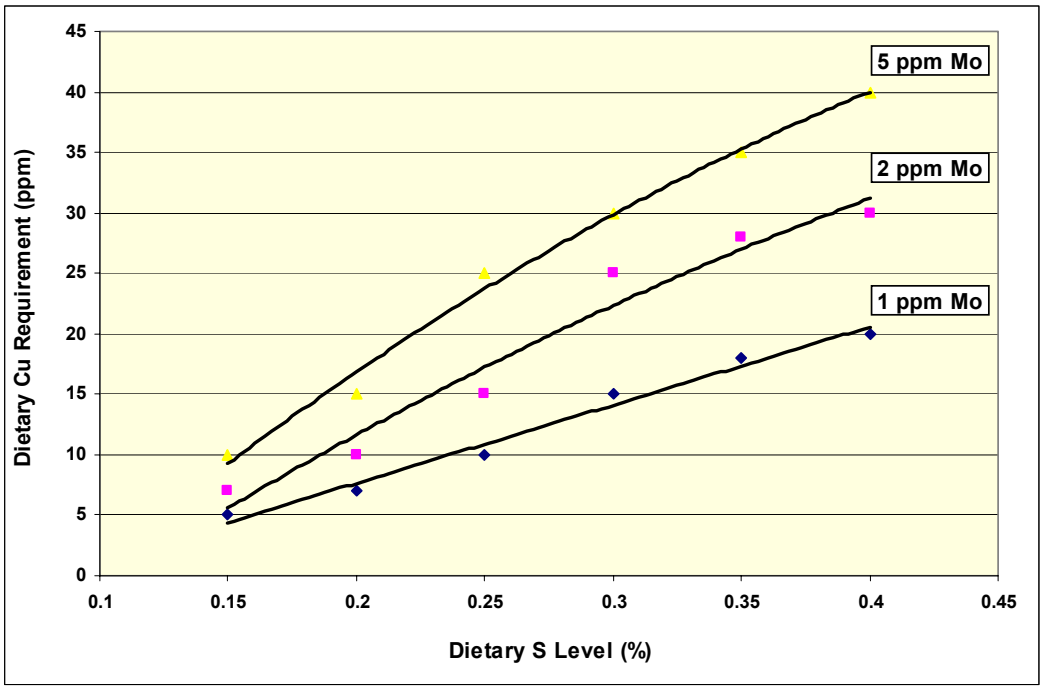

Figure 9.16.3 Relationship between dietary copper required to alleviate adverse effects under various levels of dietary molybdenum and sulphur.

There is an insufficiency of research that would provide recommendation on dietary copper required under various levels of sulphur and molybdenum. Puls (1994) recommended the following "Rule of Thumb": Cu intake should be 5 to 8 times Mo.

There are breed differences in terms of dietary copper requirements, with Simmental cattle having highest requirement, followed by Charolais, Hereford, Angus and Shorthorn, in that order. Under some circumstances, Simmentals may require twice as much Cu as Angus. However, it is important to stress that supplementation of dietary copper to offset the adverse effects of sulphur must be carried out with due care in order to avoid copper toxicity. Total dietary copper in cattle should not exceed 50 ppm. Sheep are considerably more sensitive to copper toxicity than cattle. The recommended feed copper level in sheep is between 5.0 and 10.0 ppm, but 20 ppm may be safe for a short period.

Table 9.16.3 Summary of practical information relevant to sulphur exposure in livestock.

Guidelines	Interactions			Adverse Effects and Signs of Toxicity	
Recommended Maximum in Drinking Water for Livestock†	**Essential Elements**	**Toxic Metals**	**Metabolic Effects**	**Short Term, High Level Exposure**	**Long Term, Low Level Exposure**
1000 mg/L	molybdenum magnesium, iron, iodine, manganese, copper, zinc, selenium, phosphorus	NA	Sulphur, either alone or in a synergistic effect with molybdenum, can affect GI metabolism of copper, zinc, manganese, magnesium, phosphorus and vitamin B1. Sulphate and thiosulphate may inhibit the uptake of selenate. Mo and S have a synergistic effect on reducing Cu availability by combining with Cu in the rumen to form an insoluble complex. The retention of both calcium and phosphorus may be reduced by the addition of sulphate to diets (potential metabolic problem of importance in dairy cows).	In ruminants fed high levels of sulphur, sulphides in the rumen can be generated in considerable quantities. In experimental animals, death associated with excessive synthesis of sulphide in the rumen gas cap has been reported. However, such direct adverse effects associated with sulphur toxicity are not common. High levels of S in the drinking water is an etiological factor in brain tissue necrosis commonly known as polioencephalomalacia (PEM. Sulphur-related PEM may occur within 3 to six weeks following exposure to high sulphur water or diet.	The chronic effects of long term exposure to excess dietary S represent a very discrete class of nutritional adverse effects linked to the unique features of S metabolism in the ruminant species. Cu deficiency is the most prominent problem in cattle and sheep drinking high sulphur water. If the level of copper in the ration is marginal, animals may develop signs of copper deficiency within a few weeks. The problem is more severe if the diet is also high in molybdenum.

† The CCME guideline of 1,000 mg/L is commonly cited, but without considering the total burden of dietary sulphur, this recommendation is of limited value.

9.16.3 Water Types or Conditions Where High Levels Occur

Sulphates occur naturally in many minerals and are also used in the manufacturing industry. Mining and smelting operation and pulp and paper mills also use sulphates and sulphuric acid and discharge waste into surface water.

Sulphate is usually expected to be a groundwater problem but during droughts, the level of water in some dugouts can drop below the groundwater line and very poor groundwater can flow into and contaminate the dugout. When this happens, the water quality can change drastically over a matter of weeks from a source of good quality water to water that is unfit for livestock.

Sulphur contamination in surface water bodies is often found adjacent to salt affected soils. In severely saline areas forages may also become contaminated by wind-blown sulphate salts. Surface water bodies such as sloughs, ponds, dugouts, dams and lakes have a tendency to accumulate sulphur and other dissolved minerals during periods of drought. Notably, recent observation from field study (Klemmer 2008, personal communication) revealed that even in areas with normally abundant summer rainfall in southeastern Saskatchewan, mineral concentration in dugouts can double from spring to autumn due to evaporation (Klemmer, 2008 Livestock Development Specialist, Saskatchewan Ministry of Agriculture, unpublished observations).

In Saskatchewan, about 17% of the groundwater exceeds the Canadian guideline for sulphate for livestock of 1000 mg/L(Saskatchewan Watershed Authority Rural Water Quality Data Base). The highest level recorded in groundwater was 7700 mg/L and in surface water, sulphate levels have exceeded 9000 mg/L.

The following tables show the sulphate concentration in rural Saskatchewan groundwater and surface water.

Table 9.16.4 Sulphate Concentration in Saskatchewan Groundwater

Sulphate Content (mg/L)	Number of Samples Analysed	Percent of Total
<500	1774	61.3
500 to 1000	633	21.9
1000 to 2000	399	13.8
2000 to 3000	63	2.2
>3000	24	0.8

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

Table 9.16.5 Sulphate Concentration in Saskatchewan Surface Water

Sulphate Content (mg/L)	Number of Samples Analysed	Percent of Total
<500	170	54.5
500 to 1000	80	25.6
1000 to 2000	35	11.2
2000 to 3000	12	3.8
3000 to 4000	7	2.2
>5000	8	2.56

Source: Saskatchewan Watershed Authority Rural Water Quality Data Base

9.16.4 Management Considerations

In the evaluation of exposure of ruminant animals to sulphur, it is important to consider all sources, including feed, water, and environment. In milder cases, once identified, the problem of secondary metabolic disturbances in domestic livestock animals may be corrected via nutritional supplements and clinical management of the problem.

The best management solution would be providing only good quality water, so if good quality water is available, it should be used. If economically justifiable, water purification for livestock should be advocated.

However, if water purification is not a practical solution, several strategies can be developed to manage the problem. Low to moderately high levels of S in water can be managed reasonably well. Standard management procedures should include nutritional safeguards. Levels of the dietary pool, as well as reduced S compounds from the environment, should be taken into account while assessing the risk associated with water content of S compounds. If possible, the total dietary S level (from both feed and water) should be kept below 0.3% DM basis.

Preventative measures to be considered should include balancing the ration to decrease excessive intake of S and supplementation of nutrients likely affected by S. In problem areas, an attempt should be made to decrease the load of dietary S by blending feedstuff containing high levels of S with feed and mineral supplements with low S content. Dietary supplementation of copper and thiamine in quantities exceeding the normal dietary requirement may decrease the risk of adverse effects associated with sulphur.

9.16.5 Treatment Technology

Treatment technology:

- Biological methods of sulphate removal are currently under evaluation at PFRA
- Nano-Filtration or RO membranes
- Ion Exchange

See Section on water treatment for further discussion on specific treatment systems.

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10. REFERENCES

Agriculture and Agri-Food Canada – Prairie Farm Rehabilitation Administration (AAFC-PFRA). 2002. *Algae, Cyanobacteria and Water Quality.* Available from the AAFC-PFRA Water Quality Division.

Agriculture and Agri-Food Canada – Prairie Farm Rehabilitation Administration (AAFC-PFRA). 2002. *Copper Treatments for Dugouts.* Available from the AAFC-PFRA Water Quality Division.

Alfaro, E., Neathery, M.W., Miller, W.J., Crowe, C.T., Gentry, R.P., Fielding, A.S., Pugh, D.G. and Blackmon, D.M. 1988. Influence of a wide range of calcium intake on tissue distribution of macroelements and microelements in dairy calves. J. Dairy Sci. 71:1295-1300.

Allison, I. S. 1930. The problem of saline drinking waters. Science 7 1:559.

Ammerman, C. B., Chicco, C. F., Loggins, P. E. and. Arrington, L. R. 1972. Availability of different salts of magnesium to sheep. J. Anim. Sci. 34:122.

Ammerman, C.B., Arrington, L.R., Jayaswal, M.C., Shirley, R.L and Davis, G.K. 1963. Effects of dietary calcium and phosphorus on nutrient digestibility of steers. J. Anim. Sci. 22: 248-252.

Andrews, B. F., Campbell, D. R. and Thomas, P. 1965. Effects of hypertonic magnesium-sulphate enemas on newborn and young lambs. Lancet 2:64.

Annett, C. S., D'Itri, F. M., Ford, J. R. and Prince. H. H. 1975. Mercury in fish and water fowl from Lake Ball, Ontario. J. Environ. Qual. 4:219.

ANZECC, Australian and New Zealand Environment and Conservation Council. (2000).

Archibald, J. G. 1951. Molybdenum in cows' milk. J. Dairy Sci. 34: 1026.

Armstrong, G.L., Hollingsworth, J. and Morris, J.G. 1996. Emerging foodborne pathogens: *Escherichia coli* O157:H7 as a model of entry of a new pathogen into the food supply of the developed world. Epidemiol. Rev. 18: 29-51.

Arsenic. In: Guidelines for Canadian Drinking Water Quality: Guideline Technical Document Prepared by the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment Health Canada, Ottawa, Ontario. May, 2006.

Arthur, D. I., Motzok, I. and Branion, H. D. 1958. Interaction of dietary copper and molybdenum in rations fed to poultry. Poultry Sci. 37:1181.

References

Atteh, J. O. and Leeson, S. 1983. Influence of increasing the calcium and magnesium content of the drinking water on performance and bone and plasma minerals of broiler chickens. PoultrySci. 62:869-874.

Bahman, A. M., Rooke, J.A. and Topps, J.H. 1993. The performance of dairy cows offered drinking water of low or high salinity in a hot arid climate. Anim. Prod. 57:23-28.

Balnave, D. and Scott, T. 1986. The influence of minerals in drinking water on egg shell quality. Nut. Rep. Int. 34: 29–34.

Balnave, D. and Yoselewitz, I. 1987. The relation between sodium chloride concentration in drinking water and eggshell damage. Br. J. Nutr. 58:503–509.

Balnave, D. and Yoselewitz, I. 1989. The influence of saline drinking water on the activity of carbonic anhydrase in the shell gland of laying hens. Aust. J. Agric. Res. 40: 1111−1115.

Balnave, D., Yoselewitz, I. and Dixon, R.J. 1989. Physiological changes associated with the production of defective eggshell by hens receiving sodium chloride in the drinking water. Br. J. Nutr. 61: 35–43.

Balnave, D. Zhang, D. and Moreng, R.E. 1991. Use of ascorbic acid to prevent the decline in eggshell quality observed with saline drinking water. Poultry Sci. 70: 848–852.

Balnave, D., 1993. Influence of saline drinking water on eggshell quality and formation. World's Poultry Sci. J. 49: 109-111.

Balnave, D. and Muheereza, S.K. 1997. Improving eggshell quality at high temperatures with dietary sodium bicarbonate. Poultry Sci. 76:588−593.

Beke, G.J. and Hironaka, R. 1991. Toxicity to beef cattle of sulfur in saline well water: a case study. Sci. Total. Environ. 101: 281-290.

Bell, M. D., Diggs, G. B., Lowrey, R. S. and Wright. P. L., 1964. Comparison of Mo metabolism in swine and cattle as affected by stable molybdate. J. Nutr. 84:367.

Bingley, J. R. 1974. Effects of high doses of molybdenum and sulphate on the distribution of copper in plasma and in blood of sheep. Aust. J. Agric. Res. 25: 467.

Biswas, S., Talukder, G. and Sharma, A. 1999. Prevention of cytotoxic effects of arsenic by short term dietary supplementation with selenium in mice. Mutat. Res. 441: 155-160.

Blosser, T.H. and B.K. Soni. 1957. Comparative influence of hard and soft water on milk production of dairy cows. J. Dairy Sci. 40:1519.

Boila, R.J., Devlin, T.J. and Wittenberg, K.M. 1987. Geographical variation of the total sulfur content of forages grown in Northwestern Manitoba. Can. J. Anim. Sci. 67: 869-872.

Bowen, J. M., Blackman, D. M. and Heavener, J. E.1970. Effect of magnesium ions on neuromuscular transmission in the horse, steer and dog. J. Am. Vet. Med. Assoc. 157: 164.

Boyne, R. and Arthur, J.R. 1986. Effects of molybdenum or iron induced copper deficiency on the viability and function of neutrophils from cattle. Res Vet Sci. 41: 417-419.

Brackpool, C. E., Roberts, J.R. and Balnave, D. 1996. Blood electrolyte status over the daily laying cycle and the effect of saline drinking water on the availability of calcium in the blood for egg-shell formation in the laying hen. J. Anim. Physiol. Anim. Nutr. 75:214−225.

Bremner, I., Humphries, W. R,. Phillippo, M., Walker., M. J. and Morrice, P. C. 1987. Iron-induced copper deficiency in calves: Dose response relationships and interactions with molybdenum and sulfur. Anim. Prod. 45: 403-414.

Britton, J. W. and Goss, H. 1946. Chronic molybdenum poisoning in cattle. J. Am. Vet. Med. Assoc. 108:176.

Bruce-Grey-Owen Sound Health Unit. 2000. Waterborne outbreak of gastroenteritis associated with a contaminated municipal water supply, Walkerton, Ontario, May–June 2000. Canada Communicable Disease Report **26**: 170-173.

Bruning-Fann, C.S. and Kaneene, J.B. 1993. The effects of nitrate, nitrite, and N-nitroso compounds on animal health. Vet. Hum. Toxicol. 35: 237-253.

Buck, W. B., Osweiler, G. D. and Van Gelder. U. A. 1973. Clinical and Diagnostic Veterinary Toxicology. Kendall/Hunt Publishing Company, Dubuque, Iowa.

Buck, W.B., Osweiler, G.D. and Van Gelder, G.A. 1982. Nitrate toxicity. In Clinical and diagnostic veterinary toxicology, 1982. Kendall/Hunt Pubishing Company.

Byron, W.R., Bierbower, G.W., Brouwer, J.B. and Hansen, W.H. 1967. Pathologic changes in rats and dogs from two-year feeding of sodium arsenite or sodium arsenate. Toxicol. Appl. Pharmacol. 10:132-147.

Campbell, A. G., Coup, M. R., Bishop, W. H. and Wright, D. E. 1974. Effect of elevated iron intake on the copper status of grazing cattle. N. Z. J. Agric. Res. 17: 393-399.

References

Canadian Council of Resource and Environment Ministers (CCME). 1999. Protocols for Deriving Water Quality Guidelinesfor the Protection of Agricultural Water Uses (Irrigation and Livestock Water)

Canadian Council of Ministers of the Environment (CCME). 2005. Canadian water quality guidelines for the protection of agricultural water uses.

Canadian Council of Resource and Environment Ministers (CCREM). 1987. Canadian Water Quality Guidelines. Water Quality Branch, Inland Waters Directorate, Environment Canada, Ottawa.

Care, A. D. 1960. The effect on cattle of high level magnesium supplementation of their diet. Vet. Rec. 72:517.

Casarett And Doull's Essentials Of Toxicology / editors, Curtis D. Klaassen And John B. Watkins III. New York : McGraw-Hill/Medical Pub. Div., c2003.

Challis, D. J., Zeinstra, M. S. and Anderson, M. J. 1987. Some effects of water quality on the performance of high yielding dairy cows in an arid climate. Vet. Rec, 120:12-15.

Chambers, P. A., Allard, M., Walker, S. L., Marsalek, J., Lawrence, J., Servos, M., Busnarda, J., Munger, K. S., Adare, K., Jefferson, C., Kent, R. A., and Wong, M. P. 1997. Impacts of municipal wastewater effluents on Canadian waters: A review. Water Qual. Res. J. Can. 32: 659–713.

Chandra, S.V. and Shukla, G.S. 1976. Role of iron deficiency in inducing susceptibility to manganese toxicity. Arch. Toxicol. 35: 319-323.

Chapman, L. and Chan, H.M. 2000. The influence of nutrition on methyl mercury intoxication . Environ. Health Perspect. 108 (suppl 1):29-56.

Chase, C. R., Beede, D. K., Van Horn, H. H., Shearer, J. K., Wilcox, C. J. and Donovan, G. A. 2000. Responses of lactating dairy cows to copper source, supplementation rate, and dietary antagonist (iron). J. Dairy Sci. 83: 1845-1852.

Cheeke, P.R. 1998. Natural Toxicants in Feeds Forages & Poisonous Plants 2nd ED, Interstate Publishers Inc.

Chen, J. and Balnave, D. 2001. The Influence of Drinking Water Containing Sodium Chloride on Performance and Eggshell Quality of a Modern, Colored Layer Strain Poultry Sci. 80: 91–94.

Chicco, C. F., Ammerman, C. B., Feaster, J. P. and Dunavant, B. G. 1973. Nutritional interrelationships of dietary calcium, phosphorus and magnesium in sheep. J. Anim. Sci. 36:986.

Chicco, C. F., Ammerman, C. B., Hillis, W. G. and Arrington, L. R. 1972. Utilization of dietary magnesium by sheep. Am. J. Physiol. 222:1469.

Chicco, C. F., Ammerman, C. B., van Walleghem, P. A., Waldroup, P. W. and Harms, R. H. 1967. Effects of varying dietary ratios of magnesium, calcium and phosphorus in growing chicks. Poultry Sci. 46:368.

Chorus, I.E. 2001. Cyanotoxins, Occurrence, Causes, Consequences, Springer, Berlin.

Clark, J.H., Legge, A.W., Davis, C.L. and McCoy, G.C. 1989. Effect of calcium carbonate on ruminal fermentation, nutrient digestibility, and cow performance. J. Dairy Sci. 72: 493-500.

Clarke, E. G. D. and Clarke. M. L. 1975. Veterinary Toxicology. Williams & Wilkins, Baltimore, Md.

Clinical Veterinary Toxicology. 2004. Konnie H. Plumlee, Ed. St. Louis, Mo. : Mosby, c2004.

Coia, J.E. 1998. Clinical, microbiological and epidemiological aspects of *Escherichia coli* O157 infection. FEMS Immun. Med. Mic. 20: 1-9.

Combs, G.E., Berry, T.H., Wallace, H.D. and Crum, R.C. 1966. Levels and sources of vitamin D for pigs fed diets containing varying quantities of calcium. J. Anim. Sci. 25: 827-830.

Cook, G. A., Lesperance, A.L., Bohman, V. R. and Jensen, E.H. 1966. Interrelationship of molybdenum and certain factors to the development of the molybdenum toxicity syndrome. J. Anim. Sci. 25:96.

CowBytes Ration Balancing Software (Incorporates NRC Beef 2000 Model), Alberta Agriculture Food and Rural Development.

Cragle, R. G. 1973. Dynamics of mineral elements in the digestive tract of ruminants. Fed. Proc. 32: 1910.

Crawford, R.F., Kennedy, W.K. and Davison, K.L. 1966. Factors influencing the toxicity of forages that contain nitrate when fed to cattle. Cornell-Vet. 56: 3-17.

Cristaldi, L.A., McDowell, L.R., Buergelt, C.D., Davis, P.A., Wilkinson, N.S. and Martin. F.G. 2005. Tolerance of inorganic selenium in wether sheep. Small Ruminant Res. 56: 205-213.

References

Cummings, B.A., Caldwell, D.R., Gould, D.H et al : 1995. Identity and interactions of rumen microbes associated with dietary sulfate-induced polioencephalomalacia in cattle. Am. J. Vet. Res. 56: 1384-1389,

Cunningham, H.M., Brown, J.M. and Edie, A. E. 1953. Molybdenum poisoning of cattle in the Swan River Valley of Manitoba. Can. J. Agric. Sci. 33:254.

Damron, B. L. 1998. Sodium chloride concentration in drinking water and eggshell quality. Poultry Sci. 77:1488−1491.

Damron, B. L. and Kelly, L.S. 1987. Short-term exposure of laying hens to high dietary sodium chloride levels. Poultry Sci. 66: 825–828.

Damron, B.L. Fact Sheet AN 125. Animal Sciences Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida. First published February 2002.

David, G.. Renter, D.G. and Sargeant, J.M 2002. Enterohemorrhagic *Escherichia coli* O157: epidemiology and ecology in bovine production environments. Anim. Health Res. Rev. **3**: 83-94.

Davies, R. E., Reid, B. L. Kurnick, A. A. and Couch. J.R. 1960. The effect of sulphate on molybdenum toxicity in the chick. J. Nutr. 70:193.

Davis, P. A., McDowell, L.R., Wilkinson, N.S., Buergelt, C.D., Van Alstyne, R., Weldon, R.N. and Marshall, T.T. 2006. Tolerance of inorganic selenium in rangetype ewes during gestation and lactation. J. Anim. Sci. 84: 660-668.

Dick, A. T. 1953a. The effect of inorganic, sulphate on the excretion of molybdenum in sheep. Aust. Vet. J. 29:18.

Dick, A. T. 1953b. The control of copper storage in the liver of sheep by inorganic sulphate and molybdenum. Aust. Vet. J. 29:233.

Digesti, R.D. and Weeth,H.J. 1976. A defensible maximum for inorganic sulphate in drinking water of cattle. J. Anim. Sci. 42:1498-1502.

Dowe, T.W., Matsushima, J. and Arthoud, V.H. 1957. The effects of adequate and excessive calcium when fed when fed with adequate phosphorus in growing ration for beef calves. J. Anim. Sci. 16: 811-818.

Downing, J.A., Watson, S.B., McCauley, E. 2001. Predicting cyanobacteria dominance in lakes. Can. J. Fish. Aquat. Sci. 58:1905–1908.

Elbetieha, A., Bataineh, H., Darmani, H. and Al-Hamood, M.H. 2001. Effects of long-term exposure to manganese chloride on fertility of male and female mice. Toxicol. Lett. 119:193-201.

Emerick RJ. 1974. Consequences of high nitrate levels in feed and water supplies. Fed. Proc. 33:1183-1187. Review.

Emeric, R.J. 1988. Nitrate Toxicity. In The Ruminant Animal: Digestive Physiology and Nutrition. Editor; D.C. Church. pp. 480-483.

Environment Canada. Detailed surface water quality data, Northwest Territories 1980–1981, Alberta 1980–1981, Saskatchewan 1980–1981, Manitoba 1980–1981. Inland Waters Directorate (1984).

Faith. N.G., Shere, J.A., Brosch, R., Arnold, K.W., Ansay, S.E., Lee, M.S., Luchansky, J.B. and Kaspar, C.W. 1996. Prevalence and clonal nature of *Escherichia coli* O157:H7 on dairy farms in Wisconsin. Appl. Environ. Microb. 62:1519-1525.

Fan, A.M., Willhite, C.C. and Book, S.A. 1987. Evaluation of the nitrate drinking water standard with reference to infant methemoglobinemia and potential reproductive toxicity. Regul. Toxicol. Pharmacol. 1987 7:135-48. Review.

Fan, A.M. and Steinberg, V.E. 1996. Health implications of nitrate and nitrite in drinking water: an update on methemoglobinemia occurrence and reproductive and developmental toxicity. Regul. Toxicol. Pharmacol. 23: 35-43.

Feinglass, E.J. 1973. Arsenic intoxication from well water in the United States. N. Engl. J. Med., 288: 828.

Ferguson, W. S., Lewis, A. H. and Watson, S. J. 1938. Action of molybdenum in nutrition of milking cattle. Nature (London) 141:553.

Fergusson, J.E. 1990. The heavy elements: Chemistry, environmental impact and health effects. Pergamon Press, Sydney.

Flanagan, P.R., Haist, J. and Valberg, L.S. 1980. Comparative effects of iron deficiency induced by bleeding and a low-iron diet on the intestinal absorptive interactions of iron, cobalt, manganese, zinc, lead and cadmium. J. Nutr. 110:1754-1763.

Fontenot, J. P., M. B. Wise, and K. E. Webb, Jr. 1973. Interrelationships of potassium, nitrogen and magnesium in ruminants. Fed. Proc. 32:1925.

References

Frape, D. 2004. Equine Nutrition and Feeding. Third Edition, Blackwell Publishing. pp. 108-115.

Fraser, D., J. F. Patience, P. A. Phillips, and J. M. McLeese. 1993. Water for piglets and lactating sows: Quantity, quality and quandaries. In *Recent Developments in Pig Nutrition,* 2, Cole, D. J., W. Haresign, and P. C. Garnsworthy, Eds., Nottingham University Press, Loughborough, U.K., 200-224.

Frens, A.M. 1946. Salt drinking water for cows. (as cited by Challis et al. 1987).

Fungauf, R., Vogt, H. and Penner, W. 1961. Studies on calcium tolerance in chickens. Arch. Geflugelk. 25: 82-89.

Gailer, J., George, G.N., Pickering, I.J., Prince, R.C., Younis, H.S. and Winzerling, J.J. 2002. Biliary excretion of [(GS)(2)AsSe](-) after intravenous injection of rabbits with arsenite and selenate. Chem. Res. Toxicol. 15:1466-1471.

Gainesville, FL. Maurice, D. V., 1989. Salinity of drinking water and performance of chickens. Pages 140–144 *in*: Proceedings of the Georgia Nutrition Conference, University of Georgia, Athens, GA.

Galvin, R.M. 1996. Occurrence of metals in waters: An overview. *Water SA* 22, 7–18.

Ganther, H. E. and Mertz, W. In: Trace Element Metabolism in Animals-2. University Park Press, Baltimore, Md.

Gardner, A.W. and Hall-Patch. P.K. 1962. An outbreak of industrial molybdenosis. Vet. Rec. 74:113.

Gawthorne, J. M. and Nader. C. J. 1976. The effect of molybdenum on the conversion of sulphate to suiphide and microbial-protein-sulphur in the rumen of sheep. Br. J. Nutr. 35:11.

Gengelbach, G.P. and Spears, J.W. 1998. Effects of dietary copper and molybdenum on copper status, cytokine production, and humoral immune response of calves. J. Dairy Sci. 81: 3286-3292.

Gentry, R. P., Miller, W. J., Pugh, D. G., Neathery, M. W. and Bynoum, J. B. 1978. Effects of feeding high magnesium to young dairy calves. J. Dairy Sci. 61:1750.

Gerken, H. J., Jr., and Fontenot, J. P. 1967. Availability and utilization of magnesium from dolomitic limestone and magnesium oxide in steers. J. Anim. Sci. 32:789.

Gipp, W.F., Pond, W.G and Smith, S.E. 1967. Effects of level of dietary copper, molybdenum, sulfate and zinc on body weight gain, hemoglobin, and liver storage of growing pigs. J. Anim. Sci. 26:727.

Gitelman, H. J., Kukolj, S. and Welt, L. G. 1968. Inhibition of parathyroid gland activity by hypermagnesemia. Am. J. Physiol. 215:483.

Gochfeld, M. 2003. Cases of mercury exposure, bioavailability, and absorption. Ecotox. Environ. Safe. 56: 174–179

Goetsch, A.L. and Owens, F.N. 1987. Effect of supplement sulfate (Dynamate) and thiamine-HCl on passage of thiamine to the duodenum and site of digestion in steers. Arch. Anim. Nutr. (Berlin) 37: 1075-1083.

Golfman, .LS. and Boila, R.J. 1990. Effect of molybdenum and sulfur on minerals in the digestive tract of steers. Can. J. Anin. Sci. 70: 905:920.

Goodrich, R.D. and Tillman, A.D. 1966. Effects of sulfur and nitrogen sources and copper levels on the metabolism of certain minerals by sheep. J. Anim. Sci. 25:484-491.

Goodrich, R.D. and Tillman. A. D. 1966. Cooper, sulphate and molybdenum interrelationships in sheep. J. Nutr. 90:76.

Goold, G. J. and Smith. B. 1975. The effects of copper supplementation on stock health and production. The effect of parenteral copper on the milk yield characteristics of a dairy herd with hypocuprosis. N.Z. Vet. J. 23:233.

Gooneratne, S. R., Symonds, H.W. Bailey, J. V. and Christensen, D. A. 1994. Effects of dietary copper, molybdenum and sulfur on biliary copper and zinc excretion in Simmental and Angus cattle. Can. J. Anim. Sci. 74:315-325.

Gooneratne, S.R., Buckley, W.T. and Christensen, D.A. 1989. Review of copper deficiency and metabolism in ruminants. Can. J. Anim. Sci. 69: 819-845.

Gould, D.H., Cummings, B.A. and Hamar, D.W. 1997. In vivo indicators of pathologic ruminal sulfide production in steers with diet-induced polioencephalomalacia. J. Vet. Diagn. Invest. 9: 72-76.

Gould, D.H., Dargatz, D.A., Garry, F.B., Hamar, D.W. and Ross, P.F. 2002. Potentially hazardous sulfur conditions on beef cattle ranches in the United States. J. Am. Vet. Med. Assoc. 221: 673-677.

Gould, D.H., McAllister, M.M., Savage, J.C. and Hamar, D.W. 1991. High sulfide concentration in rumen fluid associated with nutritionally induced polioencephalomalacia in calves. Am. J. Vet. Res. 52: 1164-1169.

Gould, D.H. 1998. Polioencephalomalacia. J Anim Sci. 76:309-314. Review.

References

Goyer, R.A. 1997. Toxic and essential metal interactions. Annu. Rev. Nutr. 17:37-50. Review.

Grace, N. D., Ulyatt, M. J. and Macrae, J. C. 1974. Quantitative digestion of fresh herbage by sheep. III. The movement of Mg, Ca, P, K and Na in the digestive tract. J. Agric. Sci. 82:321.

Grace, N.D., Wilson, P.R. and Quinn, A.K. 2005. Impact of molybdenum on the copper status of red deer (Cervus elaphus). N Z Vet J. 53:137-141.

Graf, G.C. and C.W. Holdaway. 1952. A comparison of "hard" and commercially softened water in the ration of lactating dairy cows. J. Dairy Sci. 35: 998.

Gray, L.E. and Laskey, J.W. 1980. Multivariant analysis of the effects of manganese on the reproductive physiology and behaviour of the male house mouse. J. Toxicol. Environ. Health, 6: 861

Grout, A. S. Veira, D. M., Weary, D. M., von Keyserlingk, M. A. G. and. Fraser D. 2006. Differential effects of sodium and magnesium sulfate on water consumption by beef cattle. J. Anim. Sci. 84:1252–1258.

Guallar, E, Sanz-Gallardo, M.I, et al., 2002. Heavy Metals and Myocardial Infarction Study Group. Mercury, fish oils, and the risk of myocardial infarction. N. Engl. J. Med. 347:1747–1754

Hallen, I.P. and Oskarsson, A. 1995. Bioavailability of lead from various milk diets studied in suckling rats. Biometals. 8:231-236.

Hammond, P.B. and Aronson, A.L 1964. Lead poisoning in cattle and horses in the vicinity of a smelter. Ann. NY Acad. Sci. 111: 595–611.

Hancock, D.D., Besser, T.E., Kinsel, M.L., Tarr, P.I., Rice, D.H. and Paros, M.G. 1994. The prevalence of *Escherichia coli* O157.H7 in dairy and beef cattle in Washington State. Epidemiol. Infect. 113: 199-207.

Hancock, D.D., Besser, T.E., Lejeune, J., Davis, M. and Rice, D.H. 2001. The control of VTEC in the animal reservoir. Int. J. Food. Microbiol. 66: 71-78.

Hancock, D.D., Besser, T.E., Rice, D.H., Ebel, E.D., Herriott, D.E. and Carpenter, L.V. 1998. Multiple sources of Escherichia coli O157 in feedlots and dairy farms in the northwestern USA. Prev. Vet. Med. 35:11-19.

Hancock, D.D., Besser, T.E., Rice, D.H., Herriott, D.E. and Tarr, P.I. 1997a. A longitudinal study of Escherichia coli O157 in fourteen cattle herds. Epidemiol. Infect. 118: 193-195.

Hancock, D.D., Rice, D.H., Thomas, L.A., Dargatz, D.A. and Besser, T.E. 1997b.
Hardt, P.F., Ocumpaugh, W.R. and Greene, L.W. 1991. Forage mineral concentration, animal performance, and mineral status of heifers grazing cereal pastures fertilized with sulfur. J. Anim. Sci. 69: 2310-2320.

Harper, G.S., King, T.J., Hill, B.D., Harper, C.M.L. and Hunter, R.A. 1997. Effect of coal mine pit water on the productivity of cattle. II. Effect of increasing concentrations of pit water on feed intake and health. Aust. J. Agric. Res. 48: 155-164

Haydock D. 2003. Sulfur-induced polioencephalomalacia in a herd of rotationally grazed beef cattle. Can. Vet. J. 44:828-829.

Health Canada. 1995. A national survey of chlorinated disinfection by-products in Canadian drinking water. Report 95-EHD-197. Ottawa, ON.

Heller, V.G. 1933. The effect of saline and alkaline waters on domestic animals. Oklahoma Agricultural Experimental Station Bulletin l 217. (as cited by Challis et al. 1987).

Hemphill, F.E., Kaeberle, M.L. and Buck, W.B. 1971. Lead suppression of mouse resistance to *Salmonella typhimurium*. Science 172: 1031–1032.

Herigstad, R. R., Whitehair, C. K., Beyer, N. Mickelsen, O. and Zabik. M. J. 1972. Chronic methylmercury toxicosis in calves. J. Am. Vet. Med. Assoc. 160:173.

Hindmarsh, J.T. and McCurdy, R.F. 1986. Clinical and environmental aspects of arsenic toxicity. CRC Crit. Rev. Clin. Lab. Sci., 23: 315.

Hintz, H.F. and Hogue, D.E. 1964. Effect of selenium, sulfur and sulfur amino acids on nutritional muscular dystrophy in the lamb. J. Nutr. 82:495-498.

Horner, R.F. 1982. Suspected ammonium nitrate fertiliser poisoning in cattle. Vet. Rec. 110: 472-474.

http://www.inspection.gc.ca/english/fssa/microchem/resid/2002_2003/anima_mvdome.shtml

Huber, J.T., Price, N.0., and Engel, R.W. 1971. Response of lactating dairy cows to high levels of dietary molybdenum. J. Anim. Sci. 32:364.

Humphries, W. R., Phillippo, M., Young, B. W. and Bremner. I. 1983. The influence of dietary iron and molybdenum on copper metabolism in calves. Br. J. Nutr. 49:77-86.

Hutton, M. and Symon, C. 1986. The quantities of cadmium, lead, mercury and arsenic entering the U.K. environment from human activities. Sci. Total Environ. 57:129.

References

International Program on Chemical safety of the United Nations (IPCS). 2002. Fluorides, Environmental Health Criteria 227. Geneva. World Heath Organization.

International Program on Chemical Safety of the United Nations (IPCS). 1995. Inorganic lead, Environmental Health Criteria 195. Geneva. World Heath Organization.

Ivan, M. and Grieve. C. M. 1976. Effects of zinc, copper and manganese supplementation of high-concentrate ration on gastrointestinal absorption of copper and manganese in Holstein calves. J. Dairy Sci. 59:1764.

James, L.F., Lazar, V.A. and Binns, W. 1966. Effects of sublethal doses of certain minerals on pregnant ewes and foetal development. Am. J. Vet. Res. 27: 132–135.

Jaster, F. H., Schuh, D. and Weguer, T.N. 1978. Physiological effects of saline drinking water on high producing dairy cows. J. Dairy Sci. 61:66-71.

Kandylis, K. 1984. Toxicology of sulfur in ruminants: review. J. Diary Sci. 67:2179-2187.

Kattnig, R.M., Pordomingo, A.J., Schneberger, A.G., Duff, G.C. and Wallace, J.D. 1992. Influence of saline water on intake, digesta kinetics, and serum profiles of steers. J. Range Manage. 45: 514-518.

Kerk, P. V. D. 1973. Metabolic disorders in sheep and cattle caused by magnesium oxide in the concentrate feed. Tijdschr. Diergeneesk. 98:1166 (via Nutr. Abstr. Rev. 44:799).

Khan, M.Z., Szarek, J., Konicki, A. and Krasnodempska-Depta, A. 1993. Oral administration of monensin and lead in broiler chick: effects on some hematological and biochemical parameters of broiler chickens. Acta Vet. Hung. 42:111-120.

Khan, M.Z., Szarek, J., Krasnodempska-Depta, A. and Konicki, A. 1993. Effects of concurrent administration of lead and selenium on some hematological and biochemical parameters of broiler chickens. Acta Vet. Hung. 41:123-137.

Kincaid, R.L. 1980. Toxicity of ammonium molybdate added to drinking water of calves. J. Dairy Sci. 63:608-610.

Kincaid, R.L. and White, C.L. 1988. The effects of ammonium tetrathiomolybdate intake on tissue copper and molybdenum in pregnant ewes and lambs. J. Anim. Sci. 66:3252-3258.

Kline, R. D., Corzo, M. A., Hays, V.W. and Cromwell. G.L. 1973. Related effects of copper, molybdenum and sulfide on performance, hematology, and copper stores of growing pigs. J. Anim. Sci. 37:936.

Kling, L.J., Soares, J.H. and Haltman, W.A. 1987. Effects of vitamin E and systemic antioxidants on the survival of mercury poisoned Japanese quail. Poultry Sci. 66: 325-331.

Koong, L..J., Wise, M. B. and Barrick, E. R. 1970. Effect ofelevated dietary levels of iron on the performance and blood constituents of calves. J. Anim. Sci. 31:422.

Kratzer, F. H. 1952. Effect of dietary molybdenum upon chicks and poults. Proc. Soc. Exp. Biol. Med. 80:483.

Krider, J. L., Albright, J. L., Plumfee, M. P., Conrad, J. H., Sinclair, C. L., Underwood, L., Jones, R. G. and Harrington, R. B. 1975. Magnesium supplementation, space and docking effects on swine performance and behavior. J. Anim. Sci. 40:1027.

Kul, O., Karahan, S., Basalan, M. and Kabakci, N. 2006. Polioencephalomalacia in cattle: a consequence of prolonged feeding barley malt sprouts. J. Vet. Med. A. 53: 123-128.

Larvor, P. 1976. Kinetics in ewes fed normal or tetany prone grass. Cornell Vet. 66:413

Lejeune, J., Besser, T.E. and Hancock, D.D. 2001. Cattle water troughs as reservoirs of *Escherichia coli* O157. Appl. Environ. Microb. 67: 3053-3057.

Lepore, P. D.and Miller. R. F. 1965. Embryonic viability as influenced by excess molybdenum in chicken breeder diets. Proc. Soc. Exp. Biol. Med. 118:155.

Lesperance, A.L. and Bohman. V. R. 1961. Criteria for measuring molybdenum toxicity. J. Anim. Sci. 20:940.

Lesperance, A.L. and Bohman. V.R. 1963. Effect of inorganic molybdenum and type of roughage on the bovine. J. Anim. Sci. 22:686.

Lewis, D. 1954. The reduction of sulfate in the rumen of sheep. Biochem. J. 56: 391-399.

Lewis, L.D. 1995. Feeding and Care of The Horse. Second Edition, A Lea & Febiger Book , Williams & Wilkins, Weverly Company.

Linder RE, Klinefelter GR, Strader LF, Suarez JD, Roberts NL. 1997. Spermatotoxicity of dichloroacetic acid. Reprod. Toxicol. 11:681-688.

Loneragan, G.H., Gould, D.H., Callan, R.J., Sigurdson, C.J. and Hamar, D.W. 1998. Association of excess sulfur intake and an increase in hydrogen sulfide concentrations in the ruminal gas cap of recently weaned beef calves with polioencephalomalacia. J. Am. Vet. Med. Assoc. 213: 1599-1604.

References

Loneragan, G.H., Wagner, J.J., Gould, D.H. Garry, F.B. and Thorens., M. A. 2001. Effects of water sulfate concentration on performance, water intake and carcass characteristics of feedlot steers. J. Anim. Sci. 79:2941.

Looper, M.L. and Waldner. D.N. 2002. Water for Dairy Cattle. Guide D-107. New Mexico State University Cooperative Extension Service.

Lopez Alonso, M., Benedito, J.L., Miranda, M., Castillo,6 C., Hernandez, J. and Shore, R.F. 2000. Toxic and trace elements in liver, kidney and meat from cattle slaughtered in Galicia (NW Spain). Food Addit. Contam. 17: 447-457.

Lopez Alonso, M., Benedito, J.L., Miranda, M., Castillo, C., Hernandez, J. and Shore, R.F. 2002. Contribution of cattle products to dietary intake of trace and toxic elements in Galicia, Spain. Food Addit. Contam. 19: 533-541.

Maenz, D.D., Patience, J.P. and Wolynetz, M.S. 1994. The Influence of the Mineral Level in Drinking Water and the Thermal Environment on the Performance and Intestinal Fluid Flux of Newly-Weaned Pigs. J. Anim. Sci. 72: 300-308.

Manassaram, D.M., Backer, L.C. and Moll, D.M. 2006. A review of nitrates in drinking water: maternal exposure and adverse reproductive and developmental outcomes. Environ. Health Perspect. 114: 320-327.

Manitoba Agriculture, Food and Rural Initiatives (MAFRI). 2004. Evaluating Water Quality for Livestock.

Manitoba Environment. 1998. *Manitoba rural water quality. Toxic blue-green algae.* Publication 2. www.cwra.org/branches/arts/manitoba/pub2page1.html.

Martin, R.G. McMeniman, N.P. & Dowsett. K.F. 1992. Milk and water intakes of foals suckling grazing mares Equine Vet. J. 24, 295-299.

Massry, S. G., Coburn, J. W. and Kleeman, C. R. 1970. Evidence for suppression of parathyroid gland activity by hypermagnesemia. J. Clin. Invest. 49:1619.

Maurice, D. V., 1989. Salinity of drinking water and performance of chickens. Pages 140−144 *in*: Proceedings of the Georgia Nutrition Conference, University of Georgia, Athens, GA.

Méranger, J.C., Subramanian, K.S. and McCurdy, R.F. 1984. Arsenic in Nova Scotian groundwater. Sci. Total Environ. 39: 49.

McDowell, L.R. 2003. Minerals in Animal and Human Nutrition. 2nd ed. Elsevier Science, Amsterdam.

McDowell, L.R. 1997. Minerals for Grazing Ruminants in Tropical Regions. IFAS, University of Florida, Cooperative Extension Service Third Edition, Gainesville, FL.

McKenzie, R.A,. Rayner, A.C,. Thompson, G.K.. Pidgeon, G.F. and Burren, B.R. 2004. Nitrate-nitrite toxicity in cattle and sheep grazing Dactyloctenium radulans (button grass) in stockyards. Aust. Vet. J. 82: 630-634.

McKenzie, R.A., Carmichael, A.M., Schibrowski, M.L., Duigan, S.A., Gibson, J.A. and Taylor, J.D. 2009. Sulfur-associated polioencephalomalacia in cattle grazing plants in the Family Brassicaceae. Aust. Vet. J. 87: 27-32.

McLeese, J. M., Patience, J. F., Wolynetz, M. S. and Christinson, G. I. 1991. Evaluation of the quality of ground water supplies used in Saskachewan swine farms. Can. J. Anim. Sci. 71: 191-206.

McLeese, J.M., Tremblay, M.L., Patience, J.F. and Christison, G.I. 1992. Water intake patterns in the weanling pig: effect of water quality, antibiotics and probiotics. Animal Production. 54: 135–142.

Méranger, J.C., Subramanian, K.S. and Chalifoux, C. 1979. A national survey for cadmium, chromium, copper, lead, zinc, calcium, and magnesium in Canadian drinking water supplies. Environ. Sci. Technol., 13: 707.

Méranger, J.C., Subramanian, K.S. and Chalifoux, C. 1981. Survey for cadmium, cobalt, chromium, copper, nickel, lead, zinc, calcium, and magnesium in Canadian drinking water supplies. J. Assoc. Off. Anal. Chem., 64: 44.

Merkley, J.W. and Sexton, T.J. 1982. Reproductive performance of White Leghorns provided fluoride. Poultry Sci. 61: 52-56

Meybeck, M. 1982. Carbon, nitrogen and phosphorus transport by world rivers. Am. J. Sci. 282: 401-450.

Meyer, H. 1990. Contributions to water and minerals metabolism of the horse, In: Advances in Animal Physiology and Animal Nutrition. pp. 1-102. Supplements to Journal of Animal Physiology and Animal Nutrition, Paul Parey, Hamburg and Berlin.

Miller, E. R., Ulirey, D. E., Zutout, C. L., Baltzer, B. V., Schmidt, D. A., Hoefer, J. A. and Luecke. R. W. 1965. Magnesium requirement of the baby pig. J. Nutr. 85:13.

Mills, C. J., Bull, R. J., Cantor, K. P., Reif, J., Hrudey, S. E., and Huston, P. 1999. Health risks of drinking water chlorination by-products: Report of an expert working group. Chron. Dis. Can. 19:91-102.

Moffitt, A. E., Jr. and Clary. J. J. 1974. Selenite-induced binding of inorganic mercury in blood and other tissues in the rat. Res. Commun. Chem. Pathol. Pharmacol. 7:593.

Moinuddin, J. F. and Lee, H. W. 1960. Alimentary, blood and other changes due to feeding MnSo MgSO and Na. Am. J. Physiol. 199:77.

Morris, E. R. and O'Dell, B. L. 1963. Relationship of excess calcium and phosphorus to magnesium requirement and toxicity in guinea pigs. J. Nutr. 81:175.

Mroz, Z., A. W. Jongbloed, N. P. Lenis, and K. Verman. 1995. Water in pig nutrition: Physiology, allowances and environmental implications. Nutr. Res. Rev. 8:137–164.

Mullen, A. L., Stanley, R. E. Lloyd, S. R. and Moghessi. A. A. 1975. Absorption, distribution and milk secretion of radionuclides by the dairy cow. IV. Inorganic radio-mercury. Health Phys. 28:685.

Mullis, L.A., Spears, J.W. and McCraw, R.L. 2003. Effects of breed (Angus vs Simmental) and copper and zinc source on mineral status of steers fed high dietary iron. J. Anim. Sci. 81: 318-322.

National Research Council, 1994. Nutrient Requirements of Poultry. 9th rev. ed. National Academy Press, Washington, DC.

National Research Council. 1974. Effects of fluorides in Animals. National Academy of Sciences, Washington, D.C.

National Research Council. 1974. Nutrients and Toxic Substances in Water for Livestock and Poultry. National Academy of Sciences, Washington, D.C.

National Research Council. 1979, Nutrient Requirements of Domestic Animals. No. 2.

Nutrient Requirements of Swine. National Academy of Sciences, Washington, D.C.

National Research Council. 1993. Health effects of ingested fluoride. National Academy of Sciences, Washington, D.C.

National Water Quality Data Bank (NAQUADAT). Water Quality Branch, Inland Waters Directorate, Environment Canada.

Neathery, M.W. and Miller, W.J. 1975. Metabolism and toxicity of cadmium, mercury, and lead in animals: a review. J. Dairy. Sci. 58: 1767-1781.

Nelson, N., Byerly, T. C. Kolbye, Jr., A. C. Kurland, L. T. Shapiro, R. E. Shibko, S. I. Stickel, W. H. Thompson, J. E. Vanden Berg, L. A. and Weissler. A. 1971. Hazards of mercury. Environ. Res. 4:1.

Newton, G. L., Fontenot, J. P., Tucker, R. E. and Polan, C. E. 1972. Effects of high dietary potassium intake on the metabolism of magnesium by sheep. J. Anim. Sci. 35:440.

Nielsen, J.B. and Andersen, O. 1995. A comparison of the lactational and transplacental deposition of mercury in offspring from methylmecury exposed mice. Toxicol. Lett. 76: 165-171.

Niles, G.A., Morgan, S., Edwards, W.C. and Lalman, D. 2002. Effects of dietary sulfur concentrations on the incidence and pathology of polioencephalomalicia in weaned beef calves. Vet. Hum. Toxicol. 44:70-72.

NRC - Mineral Tolerance of Animals 2005, 2nd Revised Ed, Committee on Minerals and Toxic Substances in diets and Water for Animals, The National Academies Press, Washington, DC.

NRC (National Research Council) 1980. Mineral tolerance of domestic animals. National Academy of Sciences, Washington, DC.

NRC. 1996. Nutrient Requirements of Beef Cattle. 7th Ed. National Academies Press. Washington, D.C.

NRC. 1998. Nutrient Requirements of Swine. 10th ed. Natl. Acad. Press, Washington, DC.

Nugara, D. and Edwards, Jr. H. M. 1963. Influence of dietary Ca and P levels on the Mg requirement of the chick. J. Nutr. 80:181.

O'K6elley, R. F. and Fontenot, J. P. 1969. Effects of feeding different magnesium levels to drylot-fed lactating beef cows. J. Anim. Sci. 29:959.

0'Kelley, R. E. and Fontenot, J. P. 1973. Effects of feeding different magnesium levels to drylot-fed gestating beef cows. J. Anim. Sci. 36:994.

Olkowski, A.A. 1992. Sulfur toxicity and sulfur-nutrient interactions in ruminants. PhD. Thesis. University of Saskatchewan. Saskatoon. Canada.

Olkowski, A.A. 1997. Neurotoxicity and secondary metabolic problems associated with low to moderate levels of exposure to excess dietary sulfur in ruminants: A review. Vet. Hum. Toxicol. 39: 355-360

Olkowski, A.A., Laarveld, B., Patience, J.F., Francis, S.I. and Christensen, D.A. 1993. The effect of sulfate on thiamine destroying activity in rumen content cultures in-vitro. Int. J. Vit. Nutr. Res. 63: 38-44.

References

Olkowski, A.A., Rousseaux, C.G. and Christensen, D. A. 1991. Association of sulfate-water and blood thiamine in beef cattle. Field studies. Can. J. Anim. Sci. 71: 825-832.

Osweiler, G.D. and Ruhr, L.P. 1978. Lead poisoning in feeder calves. JAVMA 172: 498–500.

O'Toole, D. and Raisbeck, M.F. 1995. Pathology of experimentally induced chronic selenosis (alkali disease) in yearling cattle. J. Vet. Diagn. Invest. 7:364-373.

Palmer, J.S., Wright, F.C. and Haufler. M. 1973. Toxicologic and residual aspects of an alkyl mercury fungicide to cattle, sheep and turkeys. Clin. Toxicol. 6:425.

Parizek, J.J., Kalouskova, A., Babicky, J., Benes, and Pavlik. L. 1974. Interaction of selenium with mercury, cadmium and other toxic metals. In W. G. Hoekstra, J. W. Suttie,

Parkhurst, C.R., and Thaxton. P. 1973. Toxicity of mercury to young chickens. I. Effect on growth and mortality. Poultry Sci. 52:273.

Patience J, Beaulieu A, Gillis D. 2004. The impact of ground water high in sulphates on the growth performance, nutrient utilization, and tissue mineral levels of pigs housed under commercial conditions J. Swine Health Prod. 12: 228-236.

Patience, J. F., and M. S. Wolynetz. 1990. Influence of dietary undetermined anion on acid-base status and performance in pigs. J. Nutr. 120:579-587.

Patience, J. F., J. F. Umboh, R. K. Chaplin, and C. M. Nyachoti. 2005. Nutritional and physiological responses of growing pigs exposed to a diurnal pattern of heat stress. Livest. Prod. Sci. 96:205-214.

Patience, J. F., R. E. Austic, and R. D. Boyd. 1987a. Effect of dietary electrolyte balance on growth and acid-base status in swine. J. Anim. Sci 64:457-466.

Patterson, H. H., Johnson, P. S. Patterson, T.R., Young, D.B. and Haigh, R. 2002. Effects of water quality on animal health and performance. Proc. West. Sec. Amer. Soc. Anim. Sci: 53:217-220.

Patterson, H.H., Johnson, P.S., Young, D.B. and Haigh, R. 2003. Effects of water quality on performance and health of growing steers. SDSU, Beef 2003-15.

Peirce, A. W. 1959. Studies on salt tolerance of sheep. II. The tolerance of sheep for mixtures of sodium chloride and magnesium chloride in the drinking water. Aust. J. Agric. Res. 10:725.

Peterson, H. 2000. Water Quality Requirements for Saskatchewan's Agri-Food Industry. Prepared for Agriculture and Agri-Food Canada-Prairie Farm Rehabilitation Administration.

Pfeiffer, A., H. Henkel, M. W. A. Verstegen, and I. Philipczyk. 1995. The influence of protein intake on water balance, flow rate and apparent digestibility of nutrients at the distal ileum in growing pigs. Livest. Prod. Sci. 44:179-187.

Phillippo, M., Humphries, W. R. and Garthwaite, P. H. 1987. The effect of dietary molybdenum and iron on copper status and growth in cattle. J. Agric. Sci. 109:315–320.

Pike. R. L., and Brown, M. L. 1975. Nutrition: An integrated approach, 2nd ed. John Wiley & Sons, New York.

Pilliner, S. 1999. Horse Nutrition and Feeding Second Edition. Blackwell Science pp.49-54.

Piper, R.C., Miller, V.L. and Dickenson. E. O. 1971. Toxicity and distribution of mercury in pigs with acute methylmercurialism. Am. J. Vet. Res. 32:263.

Pless, C. D., Fontenot, J. P. and Webb, Jr. K. E. 1973. Effect of dietary calcium and phosphorus levels on magnesium utilization in sheep. Va. Polytech. Inst. State Univ. Res. Div. Rep. 153:104.

Ponnapakkam, T.P., Bailey, K.S., Graves, K.A. and Iszard, M.B. 2003. Assessment of male reproductive system in the CD-1 mice following oral manganese exposure. Reprod. Toxicol. 17:547-551.

Potter, S. and Matrone. G. 1974. Effect of selenite on the toxicity of dietary methyl mercury and mercuric chloride in the rat. J. Nutr. 104:638.

Pourreza, J., Nili, N. and Edriss, M.A. 1994. Relationship of plasma calcium and phosphorus to the shell quality of laying hens receiving saline drinking water. Br. Poultry Sci. 35:755-762.

Pourreza, J., Nili, N. and Edriss, M.A. 2000. Effect of saline drinking water on eggshell quality of Leghorn and native hens. J. Agric. Sci. Tech., Iran. 2:3-8.

Prabowo, A., Spears, J. W. and Goode, L. 1988. Effects of dietary iron on performance and mineral utilization in lambs fed a forage based diet. J. Anim. Sci. 66:2028–2035.

Prankel, S.H., Nixon, R.M., Phillips, C.J. 2005. Implications for the human food chain of models of cadmium accumulation in sheep. Environ. Res. 97:348-358.

Puls, R. 1994. Mineral Levels in Animal Health: Diagnostic Data, 2nd edn. Sherpa International, Clearbrook, British Columbia.

Puschner, B., Galey, F.D., Johnson, B., Dickie, C.W., Vondy, M., Francis, T., Holstege, D.M. 1998. Blue–green algae toxicosis in cattle. Journal of the American Veterinary Medical Association 213, 1605-1607.

Raisbeck, M.F, Siemion, R.S. and Smith, M.A. 2006. Modest copper supplementation blocks molybdenosis in cattle. J. Vet. Diagn. Invest. 18: 566-572.

Rayssiguier, Y., Garel, J. M. M., Prat, J. and Barlet, J. P. 1977. Plasma parathyroid hormone and calcitonin levels in hypocalcaemic magnesium deficient calves. Ann. Rech. Vet. 8:267.

Reid, J. T., Pfau, K. 0., Salisbury, R. L., Bender, C. B. and Ward. G. M. 1947. Mineral metabolism studies in dairy cattle. I. The effect of manganese and other trace elements on the metabolism of calcium and phosphorus during early lactation. J. Nutr. 34:661.

Reid, R. L., Post, A. J. and Jung, G. A. 1970. Mineral composition of forages. W. Va. Univ. Agric. Exp. Stn. Bull. 589T.

Renter, D.G., Sargeant, J.M., Oberst, R.D. and Samadpour, M. 2003. Diversity, frequency, and persistence of *Escherichia coli* O157 strains from range cattle environments. Appl. Environ. Microb. 69: 542-547.

Ridder, W.E. and Oehme, F.W. 1974. Nitrates as an environmental, animal, and human hazard. Clin. Toxicol. 7:145-159.

Roberts, J. R., and Balnave, D. 1992. The physiological basis of poor eggshell quality in laying hens: The effect of saline drinking water on electrolyte balance and renal function. J. Anim. Physiol. Anim. Nutr. 68:197-204.

Roh, J.K., Bradley, Jr., R.L. Richardson, T and Weckel. K.G. 1975. Distribution and removal of added mercury in milk. J. Dairy Sci. 58:1782.

Rook, J. A. F. and Storry, J. E. 1962. Magnesium in the nutrition of farm animals. Nutr. Abstr. Rev. 32: 1055.

Rosenfeld, I. and Beath, O.A. 1964. Selenium: Geobotany, Biochemistry, Toxicity and Nutrition. Academic Press, New York, 411 pp.

Ross, D. B. 1970. The effect of oral ammonium molybdate and sodium sulfate given to lambs with high liver copper concentrations. Res. Vet. Sci. 11:295.

Roush, W. B. and Mylet, M. 1986. Effect of water softening, watering devices, and dietary salt level on the performance of caged Single Comb White Leghorn laying hens. Poultry Sci. 65:1866-1871.

Sallmen, M. 2001. Exposure to lead and male fertility. Int. J. Occup. Med. Environ. Health. 14:219-222.

Salonen, J.T., Seppanen, K., Nyyssonen, K., Korpela, H., Kauhanen, J., Kantola, M., Tuomilehto, J., Esterbauer, H., Tatzber, F. and Salonen, R. 1995. Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in eastern Finnish men. Circulation 91:645–655.

Sargeant, J.M., Gillespie, J.R., Oberst, R.D., Phebus, R.K., Hyatt, D.R., Bohra, L.K and Galland, J.C. 2000. Results of a longitudinal study of the prevalence of *Escherichia coli* O157:H7 on cow-calf farms. Am. J. Vet. Res. 61: 1375-1379.

Sargeant, J.M., Sanderson, M.J., Griffin, D.D. and Smith, R.A. 2004. Factors associated with the presence of Escherichia coli O157 in feedlot–cattle water and feed in the Midwestern USA. Prev. Vet. Med. 66: 207-237.

Saskatchewan Watershed Authority (SWA) Rural Water Quality Data Base. A data base for water samples from farms and small communities. Obtained from SWA in August, 2008.

Sell, J. L. 1977. Comparative effects of selenium on metabolism of methylmercury by 8chickens and quail: Tissue distribution and transfer into eggs. Poultry Sci. 56:939.

Sell, J. L. and Davidson, K.L. 1975. Metabolism of mercury, administration as methyl-mercuric chloride or mercuric chloride by lactating ruminants. J. Agric. Food Chem. 23:803.

Sell, J. L., Guenter, W. and Sifri. M. 1974. Distribution of mercury among components of eggs following the administration of methylmercuric chloride to chickens. J. Agric. Food Chem. 22:248.

Senturk, S. and Cihan, H. 2004. Salt poisoning in beef cattle. Vet. Hum. Toxicol. 46:26-27

Sharma, R.P., Street, J.C., Shupe, J.L and Bourcier, D.R. 1982. Accumulation and depletion of cadmium and lead in tissues and milk of lactating cows fed small amounts of these metals. J. Dairy Sci. 65:972-979.

Shere, J.A., Kaspar, C.W., Bartlett, K.J., Linden, .SE., Norell, B., Francey, S. and Schaefer, D.M. 2002. Shedding of *Escherichia coli* O157:H7 in dairy cattle housed in a confined environment following waterborne inoculation. Appl. Environ. Microb. 68: 1947-1954.

Shupe, J.L., Miner, ML., Harris, L.E. and Greenwood, D.A. 1962. Relative effects of feeding hay atmospherically contaminated by fluoride residue, normal hay plus calcium

fluoride, and normal hay plus sodium fluoride to dairy heifers. Am. J. Vet. Res. 23: 777-787.

Shupe, J.L., Harris, L.E., Greenwood, D.A., Butcher, J.E. and Nielsen, H.M. (1963a) The effect of fluorine on dairy cattle. 5 . Fluorine in the urine as an estimator of fluorine intake. Am. J. Vet. Res. 24: 300-306.

Shupe, J.L., Miner, ML., Greenwood, D.A., Harris, L.E. and Stoddard, G.E. 1963b. The effect of fluorine on dairy cattle. 2. Clinical and pathologic effects. Am. J. Vet. Res. 24: 964-979.

Shupe, J.L. 1980. Clinicopathologic features of fluoride toxicosis in cattle. J. Anim. Sci. 51: 746-757.

Smart. M. B., Cohen, R., Christensen, D. A. and Williams, C. M. 1986. The effects of sulphate removal from drinking water on the plasma and liver copper and zinc concentrations of beef cows and their calves. Can. J. Anim. Sci. 66:669-680.

Smart, M.E., Cymbaluk, N.F. and Christensen, D.A. 1992. A review of copper status of cattle in Canada and recommendations for supplementation. Can. Vet. J. 33:163-170.

Smith, B.P., Fisher, G.L., Poulos, P.W. and Irwin, M.R. 1975. Abnormal bone development and lameness associated with secondary copper deficiency in young cattle. J. Am. Vet. Med. Assoc. 166:682.

Smith, S.W. and Wright. H. 1975. Effect of dietary Mo on Cu metabolism. Evidence of the involvement of Mo in abnormal binding of Cu to plasma protein. Clin. Chem. Acta 62:55.

Solomon, H., Miroa, J. and Ben-Chedalia, D. 1995. Performance of high producing cows offered drinking water of high and low salinity in the Arava desert. J. Dairy Sci. 78:620-624.

Spears, J.W. 1990. Ionophores and nutrient digestion and absorption in ruminants. J. Nutr. 120:632-638.

Standish, J. F., Ammerman, C. B., Palmer, A. Z. and Simpson. C. F. 1971. Influence of dietary iron and phosphorus on performance, tissue mineral composition and mineral absorption in steers. J. Anim. Sci. 33:171-178.

Standish, J. F., Ammerman, C. B., Simpson, C. F., Neal, F.C. and Palmer, A. Z. 1969. Influence of graded levels of dietary iron as ferrous sulphate, on performance and tissue mineral composition of steers. J. Anim. Sci. 29:496–503.

Standish, J.F., Ammerman, C.B., Wallace, N.D. and Combs. G. E. 1975. Effect of high

State University Cooperative Extension Service.

Stoddard, G.E., Bateman, G.Q., Harris, L.E., Shupe, J.L. and Greenwood, D.A. 1963. Effects of fluorine in dairy cattle. IV. Milk production. J. Dairy Sci. 46:720:726.

Stoewsand, B.S., Bache, C.A. and Lisk. D.J. 1974. Dietary selenium protection of methylmercury intoxication of Japanese quail. Bull. Environ. Contam. Toxicol. 11:152.

Stuart, L.D. and Oehme, F.W. 1982. Environmental factors in bovine and porcine abortion. Vet. Hum. Toxicol. 24:435-441.

Suttie, J.W., Miller, R.E and Phillips, P.H. 1957. Studies of the effects of dietary NaF on dairy cows. 1. The physiological effects and the developmental symptoms of fluorosis. J. Nutr. 63: 211-224.

Suttie, J.W., Phillips, PH. and Miller, R.E. 1958 . Studies of the effects of dietary sodium fluoride on dairy cows. 3. Skeletal and soft tissue fluorine deposition and fluorine toxicosis. J. Nutr. 65: 293-304.

Suttie, J.W., Gesteland, R. and Phillips, R.H. 1961. Effects of dietary sodium fluoride on dairy cows. 6. In young heifers. J. Dairy Sci. 44: 2250-2258.

Suttie, J.W. and Faltin, E.C. 1973. Effects of sodium fluoride on dairy cattle: influence of nutritional state. Am. J. Vet. Res. 34: 479-483.

Suttle, N.F. 1974. Effects of organic and inorganic sulfur on the availability of dietary copper to sheep. Br. J. Nutr. 32: 559-568.

Suttle, N. F. 1975. The role of organic sulfur in the copper-molybdenum-S interrelation ship in ruminant nutrition. Br. J. Nutr. 34:411.

Suttie, J.W. and Kolstad, D.L. 1977. Effects of dietary fluoride ingestion on ration intake and milk production. J. Dairy Sci. 60:1568-1573.

Suttie, J.W., Carlson, JR. and Faltin, P.C. 1977. Effects of alternating periods of high- and low-fluoride ingestion on dairy cattle. J. Dairy Sci. 55: 790-804.

Suttie, J.W. 1978. Effects of fluorides on animals. In: First International Minerals Conference, St Petersburg, Florida. International Minerals and Chemical Corporation, Illinois, p. 87.

Suttie, J.W. 1980. Nutritional aspects of fluoride toxicosis. J. Anim. Sci. 51: 759-766.

Suttle, N.F. 1983. Assessing the mineral and trace element status of feeds. In: Robards, G.E. and Packham, R.G. (eds) Proceedings of the Second Symposium of the

International Network of Feed Information Centres. Commonwealth Agricultural Bureaux, Farnham Royal, UK, pp. 211-237.

Suttle, N.F., Abrahams, P. and Thornton, I. 1984. The role of a soil x dietary sulfur interaction in the impairment of copper absorption by soil ingestion in sheep. J. Agric. Sci. Cambridge 103: 81-86.

Suttle, N.F. and Peter, D.W. 1985. Rumen sulphide metabolism as a major determinant of the availability of copper to ruminants. In: Mills, CF., Bremner, I. and Chesters, J.K. (eds) Proceedings of the Fifth International Symposium on Trace Elements in Man and Animals, Aberdeen. Commonwealth Agricultural Bureaux, Farnham Royal, UK, pp. 367-370.

Suzuki, K., X. C. Cheng, H. Kano, T. Shimizu, and Y. Sato. 1998. Influence of low protein diets on water intake and urine and nitrogen excretion in growing pigs. Anim. Sci. Technol. 69:267-270.

Thacker, P.A. 2001. Water in Swine Nutrition. pp. 381-398. In: Swine Nutrition, 2nd Edition. Eds: Lewis, A.J. Southern. L.L. CRC Press.

Thaxton, P., Cogburn, L.A. and Parkhurst. C.R. 1973. Dietary mercury as related to the blood chemistry in young chickens. Poultry Sci. 52:1212.

Thaxton, P., Young, P.S., Cogburn, L.A. and Parkhurst. C.R. 1974. Hematology of mercury compounds in young chickens. Bull. Environ. Contam. Toxicol. 12:46.

Thaxton, P., Parkhurst, C.R., Cogburn, L. A. and Young, P.S. 1975. Adrenal function in chickens experiencing mercury toxicity. Poultry Sci. 54:578.

The Ruminant Animal: Digestive Physiology and Nutrition. 1993. D. C. Church, editor. Prospect Heights, IL : Waveland.

Thilsted, J.P., Hibbs, C.M., Dowds, S.J. and Meibohm, A. 1981. Sodium salt toxicosis in beef cows resulting from consumption of saline water. Proceedings of the Annual-Meeting of the American Association of Veterinary Laboratory Diagnosticians. 24: 229-235.

Thilsted, J.P., Hibbs, C.M., Dowds, S.J. and Meibohm, A. 1981. Sodium salt toxicosis in beef cows resulting from consumption of saline water. Proceedings of the Annual-Meeting of the American Association of Veterinary Laboratory Diagnosticians. 24: 229-235. .

Thirunavukkarasu, O.S. and Viraraghavan, T. 2003. Arsenic in drinking water: Health effects and removal technologies. In: Aquatic arsenic toxicity and treatment. T. Murphy and J. Guo (eds.). Backhuys Publishers, Leiden, The Netherlands. pp. 129-138.

Thirunavukkarasu, O.S., Viraraghavan, T. and Subramanian, K.S. 2001. Removal of arsenic in drinking water by iron oxide-coated sand and ferrihydrite - Batch studies. Water Qual. Res. J. Can. 36: 55–70.

Thirunavukkarasu, O.S., Viraraghavan, T., and Subramanian, K.S. 2003. Arsenic removal from drinking water using iron oxide-coated sand. Water. Air. Soil. Pollut. 142: 95–111.

Thirunavukkarasu, O.S., Viraraghavan, T., and Subramanian, K.S. 2003. Arsenic removal from drinking water using granular ferric hydroxide. Water SA, 29: 161–170.

Thomas, J. W. 1965. Mechanisms responsible for grass tetany, p. 14. In Proc. Ga. Nutr. Conf. Feed Manuf.

Thomas, J. W. and Moss. S. 1951. The effect of orally administered molybdenum on growth, spermatogenesis and testes histology of young dairy bulls. J. Dairy Sd. 34:939.

Thomson, A.B, Olatunbosun, D. and Valverg, L.S. 1971. Interrelation of intestinal transport system for manganese and iron. J. Lab. Clin. Med. 7:642-655.

Thomson, A.B. and Valberg, L.S. 1972. Intestinal uptake of iron, cobalt, and manganese in the iron-deficient rat. Am. J. Physiol. 223:1327-1329.

Tucker, W.B., Hogue, J.G., Waterman DF et al. 1991. Role of sulfur and chloride in the dietary cation-anion balance equation for lactating dairy cattle. J. Anim. Sci. 69:1205-1213.

Underwood, E. J. 1976. Molybdenum in animal nutrition. In W. Chappel and K. Peter son, eds. The Biology of Molybdenum. Marcel Dekker, Inc., New York.

Underwood, E. J. 1977. Trace Elements in Human and Animal Nutrition. 4th ed. Academic Press, New York.

Underwood, E.J. and Suttle, N.N. 1999. Lead. In: The mineral nutrition of livestock. 3rd Edition. CABI Publishing, NY.

United States Geological Services. (2005). National Water-Quality Assessment Program.Reston, VA: U.S. Geological Survey Available: http://water.usgs. gov/nawqa/

Uthus, E.O. 2001. High dietary arsenic exacerbates copper deprivation in rats. J. Trace Elem. Exp. Med. 14: 43-55.

Van Donkersgoed, J., Berg, J., Potter, A., Hancock, D., Besser, T., Rice, D., LeJeune, J. and Klashinsky, S. 2001. Environmental sources and transmission of *Escherichia coli* O157 in feedlot cattle. Can. Vet. J. 42: 714-720.

Vanderveen, J.E. and Keener. H.A. 1964. Effects of molybdenum and sulfate sulphur Veeramachaneni DN. 2000. Deteriorating trends in male reproduction: idiopathic or environmental? Anim. Reprod. Sci. 60-61:121-130. Review.

Vermeulen, B., De Backer, P. and Remon, J.P. 2002. Drug administration to poultry. Adv. Drug Deliv. Rev. 54:795-803. Review.

Veterinary toxicology. 2001. Joseph D. Roder, Ed.. Boston : Butterwoth-Heinemann.

Vodela, J.K, Lenz, S.D., Renden, J.A, McElhenney, W.H. and Kemppainen, B.W. 1997a. Drinking water contaminants (arsenic, cadmium, lead, benzene, and trichloroethylene). 1. Interaction of contaminants with nutritional status on general performance and immune function in broiler chickens. Poultry Sci. 76:1474-1492.

Vodela, J.K, Lenz, S.D., Renden, J.A, McElhenney, W.H. and Kemppainen, B.W. 1997b. Drinking water contaminants (arsenic, cadmium, lead, benzene, and trichloroethylene). 2. Effects on reproductive performance, egg quality, and embryo toxicity in broiler breeders. Poultry Sci. 76:1493-1500.

Vyskocil, A. and Viau, C. 1999. Assessment of Molybdenum Toxicity in Humans. J. Appl. Toxicol. 19, 185–192.

Wagner, S.L., Maliner, J.S., Morton, W.E., and Braman, R.S. 1979. Skin cancer and arsenical intoxication from well water. Arch. Dermatol., 115: 1205.

Walsh, T. and O'Moore. L.B. 1953. Excess of molybdenum in herbage as a possible contributory factor in equine osteodystrophia. Nature (London) 171:1166.

Ward, J. D., Spears, J. W. and Gengelbach, G. P. 1995. Differences in copper status and copper metabolism among Angus, Simmental and Charolais cattle. J. Anim. Sci. 73:571-577.

Ward, M.H, deKok, T.M., Levallois, P., Brender, J., Gulis, G., Nolan, B.T. and Van Derslice, J. 2005. International Society for Environmental Epidemiology. Workgroup report: Drinking-water nitrate and health--recent findings and research needs. Environ. Health Perspect. 113:1607-1614.

Water Quality Guidelines. http://www.mfe.govt.nz/publications/water/anzecc-water-quality guide-02.

Weeth, H. J., and L. H. Hunter. 1971. Drinking of sulfate water by cattle. J. Anim. Sci. 32:277-281.

Weeth, H.J. and Capps, D.L. 1972. Tolerance of growing cattle for sulfate-water. J. Anim. Sci. 34: 256-260.

Weiss, B., Clarkson, T.W. and Simon, W. 2002. Silent latency periods in methylmercury poisoning and in neurodegenerative disease. Environ. Health Perspect. 110 (Suppl 5): 851–854.

Welsh, S. 0. 1979. The protective effect of vitamin E and N, N'-diphenyl-p phenylenediamine (DPPD) against methylmercury toxicity in the rat. J. Nutr. 109:1673.

Welsh, S. 0. and Soares, J. H. Jr. 1975. The effects of selenium and vitamin E on methyl mercury toxicity in the Japanese quail. Fed. Proc. 34:913 (Abstr).

Wheeler, S.M., Brock, T.B. and Teasdale, D. 1985. Effects of adding 30 mg fluoride/l drinking water given to pregnant ewes and their lambs upon physiology and wool growth. Journal of Agricultural Science, Cambridge 105:715-726.

Whittier, P. C., and Freeman, R. M. 1971. Potentiation of metastatic calcification in vitamin D-treated rats by magnesium. Am. J. Physiol. 220:209.

WHO. *Inorganic Mercury.* Environmental Health Criteria, vol. 118. Geneva: World Health Organization, 1991
WHO. *Methyl Mercury.* Environmental Health Criteria, vol. 101. Geneva: World Health Organization, 1990

Wilkinson JM, Hill J, Phillips CJ. 2003. The accumulation of potentially-toxic metals by grazing ruminants. Proc. Nutr. Soc. 62:267-277.

Winks, W.R. 1963. Safe waters for stock. Queensland Agr. J. 89: 723-728.

Wirth J.J., Rossano, M.G., Daly, D.C., Paneth, N., Puscheck, E., Potter, R.C. and Diamond, M.P. 2007. Ambient manganese exposure is negatively associated with human sperm motility and concentration. Epidemiology. 18: 270-273.

Wright, P.L. and Bell, M.C. 1966. Comparative metabolism of Se and tellurium in sheep and swine. Am. J. Physiol. 211: 6-10.
Yoselewitz, I., Balnave, D. and Dixon, R.J. 1988. Factors influencing the production of defective egg shells by laying hens receiving sodium chloride in the drinking water. Nutr. Rep. Int. 38:697-703.

Yoselewitz, I., and Balnave, D. 1989a. The influence of saline drinking water on the activity of carbonic anhydrase in the shell gland of laying hens. Australian J. Agric. Res. 40: 1111-1115.

Yoselewitz, I., and Balnave, D. 1989b. Egg shell quality responses of pullets given saline drinking water at different ages. Br. Poultry Sci. 36:715-718.

Yoselewitz, I., and Balnave, D. 1989c. Responses in egg shell quality to sodium chloride supplementation of the diet and/or drinking water. Br. Poultry Sci. 36:273–284.

Yoselewitz, I., Klein, E., I. Malka, I., Pinchasov, Y., Levav, N. and Katz, T. 1993. Influence of drinking water quality on layer performance and eggshell quality. Pages 58−59 *in*: Proceedings of the 31st Annual Convention, Israel Branch, World's Poultry Sci. Assoc.

Yoshizawa, K., Rimm, E.B., Morris, J.S., Spate, V.L, Hsieh, C.C., Spiegelman, D., Stampfer, M.J. and Willett, W.C. 2002. Mercury and the risk of coronary heart disease in men. N. Engl. J. Med. 347:1755-1760.

Zaki, A., Ait Chaoui, A., Talibi, A., Derouiche, A.F., Aboussaouira, T., Zarrouck, K., Chait, A. and Himmi, T. 2004. Impact of nitrate intake in drinking water on the thyroid gland activity in male rat. Toxicol. Lett. 147:27-33.

Zalups, R.K. and Lash, J.H. 1994. Advances in understanding the renal transport and toxicity of mercury. J. Toxicol. Environ. Health. 42:1-44.

Zhang, D., R. E. Moreng, and D. Balnave, 1991. Reproductive performance of artificially inseminated hens receiving saline drinking water. Poultry Sci. 70:776-779

Zimmerman, D.R., Speer, V.C., Hays, V.W. and Catron, D.V. 1963. Effects of calcium and phosphorus levels on baby pig performance. . J. Anim. Sci. 22: 658-663.

Zinn, R.A and Shen, Y. 1996. Interactions of dietary calcium and supplemental fat on digestive function and growth performance in feedlot steers. J. Anim. Sci. 74: 2303-230

Zinn, R.A., Alvarez, E., Mendez, M., Montano, M., Ramirez, E. and Shen, Y. 1997. Influence of dietary sulfur level on growth performance and digestive function in feedlot cattle. J. Anim. Sci. 75:1723-1728.

Zmudzki, J., Branton, G.R., Womac, C.W., Rowe, L.D. and Wagner, B. 1986. Lactose and milk replacer influence on lead absorption and lead toxicity in calves. Bull. Environ. Contam. Toxicol. 36:356-363.

11. APPENDIX A

Summary of Canadian Water Quality Guidelines for the Protection of Agricultural Water Uses *Update October 2005*

**Printed with permission from Canadian Council of Ministers of the Environment, 2005. Canadian water quality guidelines for the protection of agricultural water uses: Summary table. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.*

Canadian Water Quality Guidelines for the Protection of Agricultural Water Uses

SUMMARY TABLE

Update October 2005

Summary of Canadian water quality guidelines for the protection of agricultural water uses.

	Irrigation water		Livestock water	
Parameter[a]	**Concentration ($\mu g \cdot L^{-1}$)**	**Date**[b]	**Concentration ($\mu g \cdot L^{-1}$)**	**Date**[b]
Aldicarb	54.9[c]	1993	11[c]	1993
Algae, blue-green [See Blue-green algae]				
Aluminum[d]	5000	1987	5000	1987
Aniline[d]	Insufficient data	1993	Insufficient data	1993
Arsenic[e]	100[f]	1997	25[f]	1997
Atrazine	10[f]	1989	5[f, g]	1989
Beryllium[d]	100	1987	100[f]	1987
2,2-Bis(*p*-chlorophenyl)-1,1,1-trichloroethane [See DDT (total)]				
Blue-green algae (Cyanobacteria)[d]			Avoid heavy growths	1987
Boron[d]	500–6000[h]	1987	5000	1987
Bromacil	0.2[f]	1997	1100[f]	1997
Bromoform [See Halogenated methanes, Tribromomethane]				
Bromoxynil	0.33[i]	1993	11[f]	1993
Cadmium	5.1[i, j]	1996	80	1996
Calcium[d]			1 000 000	1987
Captan	Insufficient data	1991	13[f, i]	1991
Carbaryl	Insufficient data	1997	1100	1997
Carbofuran	Insufficient data	1989	45	1989
Carbon tetrachloride [See Halogenated methanes, Tetrachloromethane]				
Chlordane[d]			7[l, m]	1987
Chloride[d]	100 000–700 000[k]	1987		
Chlorinated benzenes				
Monochlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,2-Dichlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,3-Dichlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,4-Dichlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,2,3-Trichlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,2,4-Trichlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,3,5-Trichlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,2,3,4-Tetrachlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,2,3,5-Tetrachlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
1,2,4,5-Tetrachlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997

Continued.

Continued.

	Irrigation water		Livestock water	
Parameter[a]	**Concentration ($\mu g \cdot L^{-1}$)**	**Date[b]**	**Concentration ($\mu g \cdot L^{-1}$)**	**Date[b]**
Pentachlorobenzene[d]	Insufficient data[n]	1997	Insufficient data[n]	1997
Hexachlorobenzene	Insufficient data[n]	1997	0.52[f, n]	1997
Chlorinated ethanes[d]				
1,2-Dichloroethane	Insufficient data	1991	5[f]	1991
1,1,1-Trichloroethane	Insufficient data	1991	Insufficient data	1991
1,1,2,2-Tetrachloroethane	Insufficient data	1991	Insufficient data	1991
Chlorinated ethenes[d]				
1,1,2-Trichloroethene (Trichloroethylene; TCE)	Insufficient data	1991	50[f]	1991
1,1,2,2-Tetrachloroethene (Tetrachloroethylene; PCE)	Insufficient data	1993	Insufficient data	1993
Chlorinated methanes [See Halogenated methanes]				
Chloroform [See Halogenated methanes, Trichloromethane]				
4-Chloro-2-methyl phenoxy acetic acid [See MCPA]				1995
Chlorothalonil	5.8[f] (other crops)	1994	170[f]	1994
Chlorpyrifos	Insufficient data	1997	24[f]	1997
Chromium				
Trivalent chromium (Cr(III))	4.9[f, n]	1997	50[f, n]	1997
Hexavalent chromium (Cr(VI))	8.0[n]	1997	50[f, n]	1997
Cobalt[d]	50	1987	1000	1987
Coliforms, fecal[d]	100/100 mL	1987		
Coliforms, total[d]	1000/100 mL	1987		
Colour			Narrative	1999
Copper[d]	200–1000[o]	1987	500–5000[p]	1987
Cyanazine	0.5[f]	1990	10[f]	1990
Cyanobacteria [See Blue-green algae]				
DDT (total) (2,2-Bis(*p*-chlorophenyl)-1,1,1-trichloroethane; Dichloro diphenyl trichloroethane)[d]			~~30~~[l, m]	1987
Deltamethrin	Insufficient data	1997	2.5	1997
Dibromochloromethane [See Halogenated methanes]				
Dicamba	0.006	1993	122	1993
Dichlorobenzene [See Chlorinated benzenes]				
Dichlorobromomethane [See Halogenated methanes]				
Dichloro diphenyl trichloroethane [See DDT (total)]				
Dichloroethane [See Chlorinated ethanes]				

Continued.

Continued.

Parameter[a]	Irrigation water		Livestock water	
	Concentration ($\mu g \cdot L^{-1}$)	Date[b]	Concentration ($\mu g \cdot L^{-1}$)	Date[b]
Dichloromethane [See Halogenated methanes]				
Diclofop-methyl	0.18	1993	9[f]	1993
Diethylene glycol [See Glycols]				
Dimethoate	Insufficient data	1993	3[f]	1993
Diisopropanolamine	2 000[f]	2005	Insufficient data	2005
Dinoseb	16[j]	1992	150	1992
Dissolved solids, total [See Total dissolved solids (salinity)]				
Endrin[d]			~~0.2~~[l, m]	1987
Ethylbenzene[d, e]	Insufficient data	1996	2.4	1996
Ethylene glycol [See Glycols]				
Fecal coliforms [See Coliforms, fecal]				
Fluoride[d]	1000	1987	1000–2000[q]	1987
Glycols[d]				
Ethylene glycol	Insufficient data	1997	Insufficient data	1997
Diethylene glycol	Insufficient data	1997	Insufficient data	1997
Propylene glycol	Insufficient data	1997	Insufficient data	1997
Glyphosate[d]			280	1989
Halogenated methanes[d]				
Monochloromethane (Methyl chloride)	Insufficient data	1992	Insufficient data	1992
Dichloromethane[d] (Methylene chloride)	Insufficient data	1992	50[f]	1992
Trichloromethane[d] (Chloroform)	Insufficient data	1992	100[g]	1992
Tetrachloromethane[d] (Carbon tetrachloride)	Insufficient data	1992	5[f]	1992
Monobromomethane (Methyl bromide)	Insufficient data	1992	Insufficient data	1992
Tribromomethane[d] (Bromoform)	Insufficient data	1992	100[g]	1992
Dichlorobromomethane[d]	Insufficient data	1992	100[g]	1992
Dibromochloromethane[d]	Insufficient data	1992	100[g]	1992
Heptachlor (Heptachlor epoxide)[d]			~~3~~[l, m]	1987
Hexachlorobenzene [See Chlorinated benzenes]				
Hexachlorocyclohexane (Lindane)[d]			4	1987
Iron[d]	5000	1987		
Lead[d]	200	1987	100	1987
Lindane [See Hexachlorocyclohexane]				
Linuron	0.071[f]	1995	Insufficient data	1995
Lithium[d]	2500	1987		

Continued.

Continued.

	Irrigation water		Livestock water	
Parameter[a]	**Concentration ($\mu g \cdot L^{-1}$)**	**Date**[b]	**Concentration ($\mu g \cdot L^{-1}$)**	**Date**[b]
Manganese[d]	200	1987		
MCPA (4-Chloro-2-methyl phenoxy acetic acid; 2-Methyl-4-chloro phenoxy acetic acid)	0.025[i]	1995	25[f]	1995
Mercury[d]			3	1987
Methyl bromide [See Halogenated methanes, Monobromomethane]				
Methyl chloride [See Halogenated methanes, Monochloromethane]				
2-Methyl-4-chloro phenoxy acetic acid [See MCPA]				
Methylene chloride [See Halogenated methanes, Dichloromethane]				
Metolachlor	28[f]	1991	50[f]	1991
Metribuzin	0.5[f]	1990	80	1990
Molybdenum[d]	10–50[r]	1987	500	1987
Monobromomethane [See Halogenated methanes]				
Monochlorobenzene [See Chlorinated benzenes]				
Monochloromethane [See Halogenated methanes]				
Nickel[d]	200	1987	1000	1987
Nitrate + nitrite[d]			100 000	1987
Nitrite[d]			10 000	1987
Organotins[d]				
Tributyltin	Insufficient data	1992	250	1992
Tricyclohexyltin	Insufficient data	1992	250[f]	1992
Triphenyltin	Insufficient data	1992	820[f, i]	1992
PCE [See Chlorinated ethenes, 1,1,2,2-Tetrachloroethene]				
Pentachlorobenzene [See Chlorinated benzenes]				
Phenol[d]			2	1987
Phenoxy herbicides[d]			100	1987
Picloram[d]	Insufficient data	1990	190	1990
Propylene glycol [See Glycols]				
Selenium[d]	20–50[s]	1987	50	1987
Simazine	0.5[f]	1991	10[f]	1991
Sulfolane	500[f]	2005	Insufficient data	2005
Sulphate[d]			1 000 000	1987
TCE [See Chlorinated ethenes, 1,1,2-Trichloroethene]				
Tebuthiuron	0.27[f] (cereals)	1995	130[f]	1995

Continued.

Continued.

Parameter[a]	Irrigation water		Livestock water	
	Concentration ($\mu g \cdot L^{-1}$)	Date[b]	Concentration ($\mu g \cdot L^{-1}$)	Date[b]
Tetrachlorobenzene [See Chlorinated benzenes]				
Tetrachloroethane [See Chlorinated ethanes]				
Tetrachloroethene [See Chlorinated ethenes]				
Tetrachloroethylene [See Chlorinated ethenes, 1,1,2,2-Tetrachloroethene]				
Tetrachloromethane [See Halogenated methanes]				
Toluene[d, e]	Insufficient data	1996	24	1996
Total coliforms [See Coliforms, total]				
Total dissolved solids (salinity)[d]	500 000–3 500 000[t]	1987	3 000 000	1987
Toxaphene[d]			~~5~~ l, m	1987
Triallate[d]	Insufficient data	1992	230[f]	1992
Tribromomethane [See Halogenated methanes]				
Tributyltin [See Organotins]				
Trichlorobenzene [See Chlorinated benzenes]				
Trichloroethane [See Chlorinated ethanes]				
Trichloroethene [See Chlorinated ethenes]				
Trichloroethylene [See Chlorinated ethenes, 1,1,2-Trichloroethene]				
Trichloromethane [See Halogenated methanes]				
Tricyclohexyltin [See Organotins]				
Trifluralin	Insufficient data	1992	45[f]	1992
Triphenyltin [See Organotins]				
Uranium[d]	10[f]	1987	200	1987
Vanadium[d]	100	1987	100	1987
Zinc[d]	1000–5000[u]	1987	50 000	1987

[a]Unless otherwise indicated, supporting documents are available from the Guidelines and Standards Division, Environment Canada.

[b]The guidelines dated 1987 have been carried over from *Canadian Water Quality Guidelines* (CCREM 1987) and no fact sheet was prepared. The guidelines dated 1989 to 1997 were developed and initially published in CCREM 1987 as appendixes on the date indicated. They are published as fact sheets in this document. Other guidelines dated 1997 and those dated 1999 are published for the first time in this document.

[c]Concentration of total aldicarb residues.

[d]No fact sheet created.

[e]The technical document for the guideline is available from the Ontario Ministry of the Environment.

[f]Interim guideline.

[g]During the initial development of this guideline, insufficient data were available to derive a livestock watering guideline value. Therefore, the Canadian drinking water quality guideline (Health and Welfare Canada 1987) was adopted. Since then, this value has been revised by Health Canada (1996). This revised drinking water quality guideline in now adopted as the guideline for livestock water.

[h]Boron guideline
=500 $\mu g \cdot L^{-1}$ for blackberries
=500–1000 $\mu g \cdot L^{-1}$ for peaches, cherries, plums, grapes, cowpeas, onions, garlic, sweet potatoes, wheat, barley, sunflowers, mung beans, sesame, lupins, strawberries, Jerusalem artichokes, kidney beans, and lima beans
= 1000–2000 $\mu g \cdot L^{-1}$ for red peppers, peas, carrots, radishes, potatoes, and cucumbers
= 2000–4000 $\mu g \cdot L^{-1}$ for lettuce, cabbage, celery, turnips, Kentucky bluegrass, oats, corn, artichokes, tobacco, mustard, clover, squash, and muskmelons
= 4000–6000 $\mu g \cdot L^{-1}$ for sorghum, tomatoes, alfalfa, purple vetch, parsley, red beets, and sugar beets
= 6000 $\mu g \cdot L^{-1}$ for asparagus

[i]Guideline value slightly modified from CCREM 1987 + Appendixes due to re-evaluation of the significant figures.

[j]Guideline is crop-specific (see fact sheet).

[k]Chloride guideline
Foliar damage
= 100–178 $mg \cdot L^{-1}$ for almond apricots and plums
= 178–355 $mg \cdot L^{-1}$ for grapes, peppers, potatoes, and tomatoes
= 355–710 $mg \cdot L^{-1}$ for alfalfa, barley, corn, and cucumbers
>710 $mg \cdot L^{-1}$ for cauliflower, cotton, safflower, sesame, sorghum, sugar beets, and sunflowers

Rootstocks
=180–600 $mg \cdot L^{-1}$ for stone fruit (peaches, plums, etc.)
=710–900 $mg \cdot L^{-1}$ for grapes

Cultivars
= 110–180 $mg \cdot L^{-1}$ for strawberries
= 230–460 $mg \cdot L^{-1}$ for grapes
= 250 $mg \cdot L^{-1}$ for boysenberries, blackberries, and raspberries

[l]This guideline (originally published in *Canadian Water Quality Guidelines* [CCREM 1987]) is no longer recommended and the value is withdrawn. A water quality guideline is not recommended. Environmental exposure is predominantly via sediment, soil, and/or tissue, therefore, the reader is referred to the respective guidelines for these media.

[m]This substance meets the criteria for Track 1 substances under the national CCME Policy for the Management of Toxic Substances (PMTS) (i.e., persistent, bioaccumulative, primarily result of human activity, and CEPA-toxic or equivalent) and should be subject to virtual elimination strategies. Guidelines can serve as action levels or interim management objectives towards virtual elimination.

[n]Substance has been re-evaluated since CCREM 1987 + Appendixes. Either a new guideline has been derived or insufficient data existed to derive a new guideline.

[o]Copper guideline
= 200 $\mu g \cdot L^{-1}$ for cereals
= 1000 $\mu g \cdot L^{-1}$ for tolerant crops

[p]Copper guideline = 500 $\mu g \cdot L^{-1}$ for sheep, 1000 $\mu g \cdot L^{-1}$ for cattle, 5000 $\mu g \cdot L^{-1}$ for swine and poultry.

[q]Fluoride guideline = 1000 $\mu g \cdot L^{-1}$ if feed contains fluoride

[r]Molybdenum guideline = 50 $\mu g \cdot L^{-1}$ for short-term use on acidic soils

[s]Selenium guideline
= 20 $\mu g \cdot L^{-1}$ for continuous use
= 50 $\mu g \cdot L^{-1}$ for intermittent use

[t]Total dissolved solids guideline
= 500 $mg \cdot L^{-1}$ for strawberries, raspberries, beans, and carrots
= 500–800 $mg \cdot L^{-1}$ for boysenberries, currants, blackberries, gooseberries, plums, grapes, apricots, peaches, pears, cherries, apples, onions, parsnips, radishes, peas, pumpkins, lettuce, peppers, muskmelons, sweet potatoes, sweet corn, potatoes, celery, cabbage, kohlrabi, cauliflower, cowpeas, broadbeans, flax, sunflowers, and corn
= 800–1500 $mg \cdot L^{-1}$ for spinach, cantaloupe, cucumbers, tomatoes, squash, brussels sprouts, broccoli, turnips, smooth brome, alfalfa, big trefoil, beardless wildrye, vetch, timothy, and crested wheat grass
= 1500–2500 $mg \cdot L^{-1}$ for beets, zucchini, rape, sorghum, oat hay, wheat hay, mountain brome, tall fescue, sweet clover, reed canary grass, birdsfoot trefoil, perennial ryegrass
= 3500 $mg \cdot L^{-1}$ for asparagus, soybeans, safflower, oats, rye, wheat, sugar beets, barley, barley hay, and tall wheat grass

[u]Zinc guideline
= 1000 $\mu g \cdot L^{-1}$ when soil pH < 6.5
= 5000 $\mu g \cdot L^{-1}$ when soil pH > 6.5

References:

CCREM (Canadian Council of Resource and Environment Ministers). 1987. Canadian water quality guidelines. Prepared by the Task Force on Water Quality Guidelines.

Health and Welfare Canada. 1987. Guidelines for Canadian drinking water quality. 3d ed. Prepared by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Advisory Committee on Environmental and Occupational Health.

Health Canada, 1996. Guidelines for Canadian drinking water quality. 6th ed. Prepared by the Federal–Provincial Subcommittee on Drinking Water of the Federal–Provincial Committee on Environmental and Occupational Health.

Reference listing:

Canadian Council of Ministers of the Environment. 2005. Canadian water quality guidelines for the protection of agricultural water uses: Summary table. Updated October 2005. In: Canadian environmental quality guidelines, 1999, Canadian Council of Ministers of the Environment, Winnipeg.

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Agriculture and Agri-Food Canada
Agriculture et Agroalimentaire Canada
University of Saskatchewan
Saskatchewan Agriculture

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Well Water Fact Sheet
Office of the Chief Medical Officer of Health
Manitoba
Building for the Future
March 2001

What water treatment devices are available?

Water treatment devices for drinking water can be divided into two groups, according to function - point-of-use and point-of-entry devices.

Point-of-use devices are portable, plumbed-in or faucet-mounted and are used to treat the water at a single tap for drinking and cooking only. Point-of-entry devices are installed on the main water supply and treat all the water entering the home.

Chlorinators, iodinators and ultraviolet light (UV) devices are most practical when it is necessary to disinfect water that serves a whole dwelling. Chlorine and iodine kill most disease-causing organisms and require short to moderate contact times.

NOTE: Chlorine or iodine treatment alone may not provide adequate protection against protozoa such as *Giardia and Cryptosporidium*. If protozoa are present or suspected, it is recommended that the water be first passed through a filter with an absolute 1 micron or smaller pore size to remove these parasites and then chemically treated with chlorine or iodine to kill bacteria and viruses. Iodine disinfection of drinking water should be reserved for emergency and occasional use (e.g., at a weekend cottage or in recreational vehicles). Iodine should not be used for long-term continuous disinfection because it is physiologically active and ingestion in excessive amounts may be harmful.

UV devices are effective against bacteria and viruses, add nothing to water and produce no taste or odour; in addition if the water is clear, exposure to UV light is required only for a few seconds. They do not, however, ensure the safety of the water beyond the point of application, so that flushing of the system is recommended after periods of non-use. Point-of-use UV light devices are also available. A pre-filter, however, should always be employed to remove protozoan cysts and reduce turbidity, thus improving the effectiveness of the UV light.

Ceramic or glass fibre filters handle smaller amounts of water and are useful when water from just one tap is to be treated for drinking and cooking, or to provide drinking water while camping, boating or hiking. Such filters can remove bacteria and protozoa from mildly contaminated waters. They are not suitable for removing viruses or for treating highly contaminated water. Therefore, when treating surface waters, it is recommended that these filters be used in conjunction with disinfection. Portable glass fibre or ceramic filters with iodine- releasing resins are available to disinfect water for campers or for travellers in countries where the safety of the drinking water is questionable. Some iodine-releasing devices contain an activated carbon filter to remove excess iodine from the water.

Distillers and ozonators are point-of-use devices suitable where electric power is available and where there is sufficient space to install the equipment. Distillation is commonly used to reduce the levels of all chemicals in drinking water. Distillation devices are effective for the removal of inorganic chemicals, including heavy metals and some organic chemicals, but are often combined with activated carbon for the removal of certain “volatile” chemicals (e.g., trihalomethanes, tetrachloroethylene). The boiling process also kills any microorganisms (viruses, bacteria and protozoa) present in the water. There are no known beneficial or harmful health effects associated with the ingestion of demineralized or distilled water.

Ozonators produce small quantities of ozone, a strong oxidizing agent that is effective in killing pathogens over a short period of time. Ozonation produces no taste or odour in the water. The process is dependent on good mixing of ozone with the water. Unlike chlorine and iodine, ozone does not protect the water after application. Ozonation is often combined with activated carbon filtration to achieve more complete water treatment.

What else can I do to be safe?

- When camping, canoeing or hiking, you should assume that all waters contain disease-causing organisms and you should disinfect the drinking water before use. Care must also be taken to avoid ingestion of untreated water during other activities such as brushing your teeth.
- Wells should be analyzed at least annually for microbiological contamination. Drinking water should contain 0 (zero) total coliform bacteria per 100 ml. If well water does not comply with this guideline, it should be disinfected using one of the methods described above.
- As most disinfection systems require clear water to ensure maximum efficiency, it may be necessary to combine two specific devices — one to remove various organic or inorganic compounds or to reduce sediments in the water and one to reduce microbiological contamination. Ultimately, the best approach to ensure complete disinfection of water intended for human use and consumption is a multi-barrier one consisting of collecting water from the cleanest source possible, followed by filtration and disinfection.

The importation and sale of materials, such as water treatment devices and disinfectants that come in contact with drinking water, falls under the jurisdiction of the federal government. At present, there is no specific legislation governing these products in Canada.

Drinking Water Fact Sheets

How Do I Know If My Well Water Is Safe?

How Do I Test My Well Water?

What Do I Do When a Boil Water Advisory Is Issued?

How Do I Disinfection My Well?

What are the Guidelines for Food Establishments During a Boil Water Advisory?

What Water Treatment Devices are Available?

Where can I get more information?

For further information on well water safety, please contact HealthLinks at 788-8200 or 1-888-315-9257, or contact the nearest office of Manitoba Conservation or The Manitoba Water Services Board at the numbers listed below.

Manitoba Conservation

Winnipeg	204-945-0675
Fax	204-945-1211
Brandon	204-726-6064
Fax	204-726-6567
Virden	204-748-2321
Fax	204-748-2388
Steinbach	204-346-6060
Fax	204-326-2472
Selkirk	204-785-5030
Fax	204-785-5024
Lac du Bonnet	204-345-1447
Fax	204-345-1415
Flin Flon	204-687-1625
Fax	204-687-1623
The Pas	204-627-8307
Fax	204-623-1773
Killarney	204-523-5285
Fax	204-523-4626
Dauphin	204-622-2030
Fax	204-622-2306
Swan River	204-734-3436
Fax	204-734-5151
Winkler	204-325-1750
Fax	204-325-1758
Portage la Prairie	204-239-3188
Fax	204-239-3185
Thompson	204-677-6704
Fax	204-677-6652

The Manitoba Water Services Board

Brandon	204-726-6079
Fax	204-726-6290
Dauphin	204-622-2116
Fax	204-622-2298
Beausejour	204-268-6059
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Information Compiled by the Drinking Water Coordinating Group

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September 2017

Avian Advice

...helping ensure the efficient production of top quality poultry products in Arkansas and beyond.

Clean Water Lines for Flock Health

By Dr. Susan Watkins
Center of Excellence for Poultry Science
Division of Agriculture

DIVISION OF AGRICULTURE
RESEARCH & EXTENSION
University of Arkansas System

Co-Editor Karen Christensen, Associate Professor/Extension Specialist

Co-Editor F. Dustan Clark, Extension Poultry Health Veterinarian

Clean, safe and sanitized water is crucial in assuring flocks perform their best. Before implementing a daily water sanitation program, it is important to thoroughly clean the water distribution system.

Line cleaning is necessary because low levels of sanitizer placed in dirty water lines can result in biofilm sloughing causing clogging of the drinkers.

Another impact of adding sanitizers is a reaction with the biofilm resulting in an off taste to the water thus causing birds to "back off" of the water.

Effectively cleaning the water system (including the drinker lines) helps remove biofilm and scale build-up that can act as a food source and hiding place for harmful pathogens such as E. coli, Pseudomonas or even Salmonella.

In fact; some bacterial pathogens, such as, Salmonella can live for weeks in water line biofilm resulting in a continuous source of contamination.

In addition, proper line cleaning can help with prevention of calcium scale deposits which can reduce pipe volume as much as 70-80%.

The use of cleaning products present some dangers since, many of the popular water additive products such as acids and performance enhancers can create conditions favorable for the growth of yeasts and molds, if they are present. Yeasts and molds can actually thrive in low pH water resulting in a gooey slime that will clog drinkers and generally create disaster in water systems. The bottom line is water systems must be properly cleaned between flocks.

Getting Started:

The first step to assure proper cleanliness of water lines is to answer the following questions:

1. *What is the water source?*

Untreated well water is the most vulnerable for formation of slime or biofilm in the drinker lines. Most municipal or rural water supplies contain a minimum of 0.2 ppm free chlorine which greatly reduces bacteria growth.

2. *What is the mineral content of the water supply?*

The minerals calcium and magnesium are the sources of a hard white build-up called scale. Water in a system that contains more than 60 ppm of either or both these minerals and a pH above 7 has an increased possibility for scale buildup in the system. This scale needs to be removed with an acid cleaner designed for nipple drinker systems. Other common mineral contaminants are iron, manganese and sulfur. Iron results in a rusty brown to red colored residue, while manganese and sulfur can form black colored residues. If the water smells like rotten eggs, then the culprit is not sulfur but hydrogen sulfide (a by-product of sulfur loving bacteria and the lines will need to be cleaned with a strong sanitizer). If the filters at the beginning of the water lines are rusty or black colored, then a strong acid cleaner should be used after the sanitizer flush.

3. *What products have been used in the water system?*

If additives such as vitamins, electrolytes, sugar based products, mineral based performance enhancers or weak concentrations of water acidifiers have been used frequently, quite possibly a biofilm is present. Once a biofilm is established in a water system, it is 10-1000 times harder to clean. It is important to play it safe and use strong sanitizer cleaners.

4. *Have there been health issues flock after flock such as E. coli, necrotic enteritis or respiratory challenges that do not respond to good management, clean-out or down-time?*

The culprit for these problems may be hiding and thriving in the water supply, especially water regulators and drinker lines. Cleaning with a strong sanitizer is definitely an option that might help.

Choosing a Product

After identifying the type of cleaning that will be most beneficial, the next step is to choose a product that will not damage the equipment. Currently there are several acid products that can be used for scale removal. Check with your local animal health product supplier for options. Just remember that in order for the product to be effective in removing scale, it needs to drop the water pH below 5 but should not drop the pH below 4 to prevent equipment damage.

While a strong bleach solution might be effective in removing biofilm, the potential damage it can do to the regulators and nipple drinkers makes this a poor option; the same is true for many cleaners that might otherwise be good poultry barn disinfectants. Iodine is not very effective against biofilms so it is a poor choice. Currently there are several sanitizer products

available for cleaning drinker systems, but some of the most effective products which are not damaging to the drinker systems are the concentrated, stabilized hydrogen peroxides. The active ingredients in these products are different from over-the-counter hydrogen peroxide because the stabilizer keeps the sanitizer from converting to water and oxygen before it finishes the cleaning job. There are also several chlorine dioxide products available, but they are most effective if an acidifier is present which may require dual injectors or a way to safely mix the products prior to injection. A third product used by the industry is household ammonia. A quick test on algae showed that one ounce of ammonia per gallon of water was as effective as a 3% ammonia solution. However it is strongly recommended that the equipment manufacturer be consulted before use.

The most important fact to remember is biofilms or established growth of bacteria, molds and fungus in water systems can only be removed with cleaners that contain sanitizers. It also should be a product and concentration that will not damage the equipment. Pay close attention to any product safety recommendations and follow them accordingly.

Cleaning the system

After the birds are removed from the house, clean the system. First flush the lines with water. Use a high pressure flush if available. This will remove any loose sediment from the lines. Make sure the standpipes are working properly to assure any air build-up that may occur during the cleaning process will be released from the lines.

Next, determine how the cleaner will be injected. If a medicator is used, it may not provide the concentration of cleaner necessary, therefore use the strongest product available to overcome the dilute injection rate of the medicator. A very effective alternative is mixing the cleaner in a 55 gallon barrel and then using a small submersible pump (1/12th horse power) to pump the product either into individual lines or through the water tap where the medicator attaches to the water line. A third option is pumping the cleaner from the well room through a variable injection pump which will pump solutions stronger than a 1:128 rate. This is a good idea because it cleans the water lines going to the poultry house, a possible source of contaminants. However, if the distribution lines are very dirty then the dirt in them will be sent into the poultry house water lines and therefore will require extra flushing of the lines. Use this option only if there is a faucet in the poultry barn that can be used to flush the water lines before water reaches the nipple drinker lines. In a 400 foot poultry house it takes approximately 7 gallons of water per line. So eight 180 foot lines will require approximately 56 gallons of prepared cleaning solution. Once the drinker lines are filled with the cleaning solution, let it stand as long as possible with 72 hours being ideal. Use a broom to sweep the nipple drinkers in order to get the cleaning product down into the drinkers. However check with the product manufacturer to assure this will not damage the equipment. After the lines are cleaned, if mineral build-up is an issue, then re-flush the lines with the acid cleaner.

Keeping the System Clean

Cleaning the water lines between flocks is only half the battle. Even with a thorough cleaning, if a significant number of bacteria, fungi or yeasts are still present, then the biofilm has the potential to return completely in 2-3 days. Therefore the last step is to establish a daily water sanitation program. This will benefit both the birds and the water system.

Quick Guide to Cleaning Water Lines and Starting Chicks

1. After birds are gone, flush all water lines with plain water to loosen biofilm and remove any sediment. Make sure standpipes and drain hoses are working. Use safety glasses and plastic/rubber gloves.
2. Utilize the Qwik Blend Pump (attaches where Medicator connects to water line) to inject a 3% solution of ProxyClean, HydroClean, Siloxicide, CID 2000 or Sanidate.
    a. Determine amount of product to use:
    b. The Qwik Blend adds 4 oz to each gallon of water so 1 gallon of product treats 32 gallons.
    c. Every 100 feet of water line holds ~ 2.5 gallons of water
3. Flush product into each line
4. Activate nipple drinkers with a broom or by hand (wear gloves)
5. Leave in lines:
    a. Proxyclean, HydroClean or Siloxicide- 24 hours minimum; 48 to 72 hours is even better.
    b. CID 2000 or Sanidate- 4-8 hours
6. Flush cleaner from lines with water that contains a sanitizer level birds can drink
    a. Proxyclean-2-4 ounces/gallon-this is stock solution then administer with medicator at a rate of 1 ounce per gallon of water (1:128); Use the higher rate for dirty water, lower rate for cleaner water
7. For farms with hard water (more than 110 ppm combined calcium and magnesium)
    a. Skip step 6 and do the following:
    - Fill lines with a solution of citric acid or other low pH product approved for use with water lines and let stand in lines for 24 hours.
    - Acid stock solution: Mix 4-6 packs of citric acid per gallon of water to make a stock solution (The more scale in water the more acid should be added to the stock solution). The final pH of the water should be less than 6 with 5 pH ideal for scale removal. Mineral Clean or Proxor are excellent descaler products as well.

1. Final flush before new flock arrives. (Water birds will start on)
    a. Prepare one of following stock solutions. Add with medicator or peristalic pump at rate of 1:128
        - Bleach stock solution: 4-6 ounces bleach in a gallon of water
            - Goal: 2-4 ppm of free chlorine in the drinking water
        - Hydrogen peroxide stock solution: 2-3 ounces of product in a gallon of water
            - Goal: 25-75 ppm of H_2O_2 in the drinking water
2. Maintain water sanitation for at least first 7-14 days
    a. If starting birds on chlorine, flush water lines once a day.
    b. If starting birds on stabilized hydrogen peroxide solution (Proxyclean, CID, Sanidate), sanitizer should remain effective in water lines for up to 5 days but flushing in fresh product every 2-3 days could still be beneficial.

DO NOT ADD CHLORINE WHEN ADMINISTERING VACCINES, MEDICATIONS, VITAMINS OR COPPER SULFATE, DO NOT MIX CHLORINE AND OTHER PRODUCTS IN THE SAME STOCK SOLUTION

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Evaluation of Different Hydrogen Peroxide Products for Maintaining Adequate Sanitizing Residual in Water

by Tyler Clark, Brookee Dean and Susan Watkins
University of Arkansas, Division of Agriculture

INSIDE

Introduction

A clean, safe water supply is essential in poultry production. Yet even producers who take every precaution to ensure that their water supply is safe may experience problems with high bacteria counts and biofilms in their water lines. Thus, it is important to understand the capabilities of water sanitation products, particularly those products capable of reducing or destroying biofilms (Hancock et al., 2007).

Hydrogen peroxide has been used as an antimicrobial agent since the early 1800's. It was used as a disinfectant in milk as early as 1904 and is presently approved by the Food and Drug Administration (FDA) for packaging and surface sterilization in the food industry (Schurman, 2001). Hydrogen peroxide has shown to be effective against biofilms (Carpentier and Cefr, 1993).

Hydrogen peroxide (H_2O_2) is a weak acid that works as an oxidizer similar to chlorine. The key by-products formed when hydrogen peroxide is used are water and oxygen which makes it a good choice for treating water with high levels of organic matter such as ponds or rivers. The hydrogen peroxide found in drugstores or pharmacies is only a 3% concentration, while the products commonly used for water disinfection range from 16 to 34% with 50% H_2O_2 products available for use in removing biofilms from water systems between flocks. Hydrogen peroxide can also be used to oxidize iron, manganese and sulfur which can then be removed with filtration.

The Environmental Protection Agency (EPA) guidelines recommend 25-50 ppm of residual H_2O_2 in drinking water. However, water disinfection products use different stabilizing systems, which brings us to the questions we are attempting to address here:

1. How much of the different H_2O_2 concentrates is required to make a 25-50 ppm residual in water? and;
2. How long do different sources of H_2O_2 remain effective once they are blended into a stock solution and added to water?

Materials and Methods

The following four products were tested: hydrogen peroxide (35%), HydroLine Cleaner® (34% stabilized), Proxy-Clean® (50% stabilized), and Oxy Blast Plus® (34% stabilized). It is important to note that the HydroLine Cleaner®, Proxy-Clean® and Oxy Blast® all contain

EVALUATION — cont'd on page 2

additional proprietary ingredients used for stabilization and enhancing effectiveness. Oxy Blast® also has NSF International approval as a drinking water additive.

Each product was mixed with tap water to make four separate stock solutions of: 1 ounce/gallon (oz/gal), 2 oz/gal, 4 oz/gal, and 6 oz/gal for each product. The tap water was tested for residual chlorine before mixing and measured 0 ppm. Next 1 milliliter (ml) of each stock solution was added to 128ml of tap water to create a 1:128 solution. This simulated the ounce of each stock solution that would be added to a gallon of water (128 ounces) by a medicator injecting at a 1:128 rate. After creating each of the final solutions, the parts per million (ppm) of hydrogen peroxide was tested using Oxy Blast® Peroxide Test Strips which measures H_2O_2 residual from 0 to 100 ppm. Each solution was covered and then tested again on days 1, 2, 3 ,4 and 5 post preparation.

Results

The data in Table 1 indicate that under the conditions of this trial none of the products tested provided 25-50ppm at the 1 oz/gal stock solution level. At 2 oz/gal stock solution, hydrogen peroxide and Proxy-Clean® produced 25ppm H_2O_2 solution, while a 4 oz/gal stock solution of HydroLine® was required to produce the same concentration. A 2 oz/gal stock solution of Oxy Blast® produced 50ppm concentration of H_2O_2.

Assuming the products tested contained the listed percentages of hydrogen peroxide and no activity was lost in the dilution process, initial H_2O_2 activity for the 2 oz/gal stock solution concentration should have been 42.7, 41.5, 61.0 and 41.5 ppm for hydrogen peroxide, Hydroline®, Proxy-Clean® and Oxy Blast®, respectively . However, the data in Table 1 suggest that in 41.5, 75.9 and 59% of the H_2O_2 activity was lost in the initial dilution of hydrogen peroxide, HydroLine® and Proxy-Clean®, respectively. These data suggest that, while effective, the activity of hydrogen peroxide can be quickly lost. Therefore, it is imperative that label directions be followed when using such products

By day one or 24 hours post mix of solutions, the hydrogen peroxide at 2 oz/gal had decreased a residual H_2O_2 activity of 10ppm and held this concentration till day 5 when it was decreased to 5 ppm. The hydrogen peroxide at 4 oz/gal dropped to 50 ppm by day 2 and then to 25 ppm by day 3 and dropping further by day 5 to 10 ppm. HydroLine® at 4 oz/gal gave a 25 ppm residual reading till Day 3 when it dropped to 10 ppm and then finished day 5 with a 5 ppm reading. The Proxy-Clean® 2 oz/gal gave a 25 ppm reading till day 2 and then on day 3 it had dropped to 10 ppm for the rest of the measurement time period. The Oxy Blast® 2 oz/gal mixture dropped to 25 ppm by day 1 and this held till day 3 when the residual dropped to 10 ppm. These

Table 1. Residual H_2O_2 Activity from Different Products over a 5 Day Period

Product; Concentration	Day 0	Day 1	Day 2	Day 3	Day 4	Day 5
	←——H_2O_2 Concentration (ppm)——→					
35% H. Perox.; 1oz/gal	10	10	10	10	10	5
35% H. Perox.; 2oz/gal	25	10	10	10	10	5
35% H. Perox.; 4oz/gal	≥100	50	25	25	25	10
35% H. Perox.; 6oz/gal	≥100	50	25	25	10	10
HydroLine®; 1oz/gal	2	2	0.5	0.5	0.5	0.5
HydroLine®; 2oz/gal	10	25	10	10	10	5
HydroLine®; 4oz/gal	25	25	25	10	10	5
HydroLine®; 6oz/gal	≥100	50	25	25	10	10
Proxy-Clean®; 1oz/gal	25	10	10	5	5	2
Proxy-Clean®; 2oz/gal	25	25	25	10	10	10
Proxy-Clean®; 4oz/gal	≥100	≥100	50	25	25	10
Proxy-Clean®; 6oz/gal	≥100	≥100	≥100	50	25	25
Oxy Blast®; 1oz/gal	10	10	10	10	10	5
Oxy Blast®; 2oz/gal	50	25	25	10	10	5
Oxy Blast®; 4oz/gal	≥100	≥100	50	25	10	10
Oxy Blast®; 6oz/gal	≥100	≥100	50	25	10	10

results suggest that hydrogen peroxide, Proxy-Clean® and Oxy Blast® at a 2 oz/gal stock solution concentration should be adequate for providing a 25-50 ppm residual for at least 24 hours.

The data shown in Figure 1 compare the average residual H_2O_2 activity for stabilized and unstabilized hydrogen peroxide products over all concentrations tested in this trial. While both product types began and were about the same concentration on days 3, 4 and 5 of the test, stabilized products maintained higher concentrations than unstabilized products on days 1 and 2. These data suggest that stabilized hydrogen peroxide products offer some additional residual H_2O_2 activity when compared to unstabilized products but, the additional residual activity is transient, lasting no more than one or perhaps two days.

Summary

Mixing hydrogen peroxide products to obtain a solution with a 25-50 ppm residual H_2O_2 in the drinking water required a stock solution of at least 2 oz/gal with most products. However, since hydrogen peroxide products can rapidly lose potency, it is recommended that fresh stock solutions be made every 2-3 days. Although stabilized hydrogen peroxide products offer some additional residual H_2O_2 activity over unstabilized products, this activity lasts no more than two days. Finally, it is important to note that not all the products are labeled as drinking water additives so please take this into consideration when choosing water sanitizer products and follow label direction.

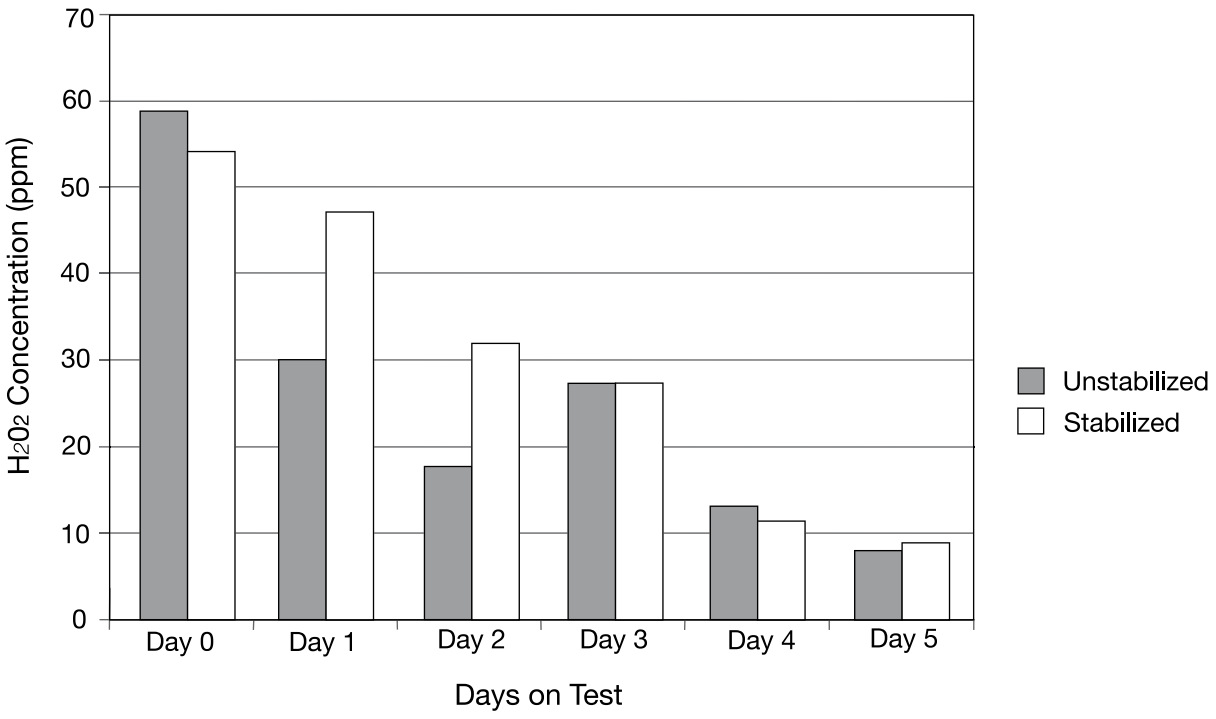
References

Carpentier, B. and O. Cefr, 1993. Biofilms and their consequences, with particular reference to hygiene in the food industry. J. Applied Bacteriol. 75:499-511.

Hancock, A., J. Hughes and S. Watkins, 2007. In search of the ideal water line cleaner. Avian Advice 9(1):1-4.

Schurman, J. J. 2001. Antibacterial activity of hydrogen peroxide against Escherichia coli O157:H7 and Salmonella spp in fruit juices, both alone and in combination with organic acids. Thesis submitted to Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of Master of Science.

Figure 1. Residual H_20_2 Activity of Stabilized And Unstabilized Hydrogen Peroxide Products[1]



[1]The data represent the average concentrations obtained when 1, 2, 4 and 6 oz/gal solutions were diluted 1 to 128.

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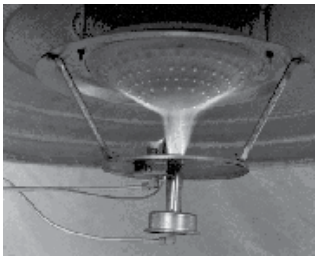
How Much Moisture Do Brooders Add to Poultry Houses?



Introduction

The vast majority of poultry growers use unvented heating units, i.e. brooders or space furnaces, to heat their poultry houses, using propane or natural gas as fuel sources. Record high propane/natural gas prices over the last two years have led a number of producers to explore the possibility of using biomass furnaces to provide heat in their poultry houses. A number of alternative heating systems exist with a price range of less than $10,000 to over $60,000 (Czarick, et al., 2008). Generally alternative heating systems are considered profitable if they are able to replace approximately 85% of the propane use, but conventional brooder/space heating systems must still supply heat during peak demand (Wimberly, 2008).

While the main benefit of biomass furnaces lies in its potential fuel saving, an overall improvement in air quality in the house as a result of introducing "dry heat" is an additional benefit reported by furnace vendors and some growers. This claim is based on the fact that unvented heating units such as brooders or space heaters release water vapor as they generate heat, while vented systems leave the combustion byproducts outside and introduce heat into the houses by heat exchangers. Unvented propane heaters are estimated to add 0.000078 pounds of water vapor for each BTU heat generated (ASHRAE, 1985). Natural gas releases slightly more water vapor than propane per unit of heat generated. If "dry heat" releases less water vapor into the poultry house, this is likely to lower in-house ammonia and ventilation requirements because of drier litter conditions. However, water vapor from unvented conventional heaters is only a portion of the moisture load added to the house, and this portion varies both within a flock and among flocks in a year. It may represent a high proportion of the moisture load during the brooding stage in cold weather when feed and water consumption are low, but much less of the load as birds get older. We decided to study the relative contribution of moisture to housing environment and potential significance of the "dry heat" benefit based on available scientific data so that growers are equipped to make wise investment decisions with respect to the relative importance of "dry heat."

Materials and Methods

This analysis was conducted based on weekly propane usage, feed consumption and water intake data collected from 18 winter flocks (flocks placed in November, December and January) raised at the Applied Broiler Research Farm (ABRF). When we did this study we assumed that, when relatively low levels of heating were required during mild weather, because of convenience and system efficiency, propane heating systems would be favored over biomass furnaces.

Moisture loads in poultry houses consist of moisture generated by birds and water vapor generated by propane heaters. Moisture generation by birds included water intake from drinkers, water in the feed (assume feed moisture content of 13%) and metabolic water generated through the digestion of feed. Yet some of the water in poultry houses is retained in the bodies of the birds. Therefore, the amount of water retained by the birds (water retention) was calculated.

Several assumptions were made to conduct the analysis:

1. Each 40 by 400 house was assumed to have 20,000 birds at placement, even though the actual bird number of each flock varied by target market weight and season;
2. Water was assumed to make up 80% of live weight of birds. This assumption was used to calculate the proportion water in the house that was retained by the birds (water retention);
3. One BTU of propane generates 0.000078 (7.80×10^{-5}) lbs of water vapor;
4. One gallon of propane generates 92,000 BTU.

Further analysis was made on daily propane use during the first two weeks of the most recent five winter flocks raised in 2006, 2007 and 2008, and compared to daily moisture loads added by birds.

Results and Discussion

On average, birds drank between 1.5 to 2.1 pounds of water for every pound of feed consumed. Water consumption from drinkers was found to represent a majority of water added to the house. An average of 19% of the water in the house was retained by the birds. This means that 81% (range of 75 to 85%) of the water that entered houses was released back into the house environment, by respiration and excretion (Figure 1).

If unvented propane heaters account for a large portion of the moisture added to poultry houses, it seems logical to assume that moisture addition problems would be worst in the winter months. Yet, analysis of propane consumption data from winter flocks revealed that unvented burning of propane generated an average of 23% of total moisture loads in the first week of brooding, 11% of the moisture in the second week, and 5% or less in the remaining weeks (Table 1, Figure 2). Still, a major portion of the fuel combusted over the life of the flock is expended maintaining house temperatures of 85 to 90°F during these early weeks. In addition, the overall growth rate and settlement status may well be determined during these early weeks (Tabler, 2000; Tabler, 2003). Therefore, daily propane usage data from the five most recent winter flocks was analyzed to get a better picture of moisture loads within the first two weeks of chick placement.

Figure 3 shows that moisture generated by propane burning represented 84 and 41% of the total load on days 1 and 2, respectively. The percentage of moisture from burning propane decreased as birds grew, and stabilized at around 11% during the second week of age. The dry heat from vented furnaces is clearly beneficial during the early days after bird placement when propane consumption is very high. Calculations show that on average the moisture load could be reduced by 20% during the first week. While this reduction in moisture load would translate to drier litter conditions, and may allow the grower to reduce ventilation rates, it is important to remember that total moisture loads increase dramatically as birds grow, and moisture generated by birds remains the main reason for ventilation. While the benefits of dry heat from biomass furnaces become smaller as birds grow, it is also important to recognize that energy efficiency is also related to litter preparation between flocks. Growers that skip or short cut may save time, but those who take the extra time to do the job right will likely find dividends in the settlement check (Tabler et al., 2008).

Summary

Several potential environmental and economic benefits have been reported for biomass furnace systems. While these benefits are often valid, it is important to see the whole picture. Vented furnaces produce dry heat that is reported to reduce in-house ammonia levels, decrease ventilation rates, improve litter quality and produce a healthier environment within the house (Wimberly, 2008). Moisture load calculations based on propane usage data collected at the Applied Broiler Research Farm indicate that when using vented biomass furnace, about 23% less moisture can be added to the indoor environment during the first week of brooding, when birds are very sensitive to house conditions and maintaining elevated temperatures requires the combustion of large amounts of propane. However, as birds grow bigger, more moisture is added by feeding and drinking, which represent more than 90% of in-house water inputs from second week on.

Table 1. Weekly Moisture Loads Generated by Birds and Unvented Propane Heaters

Week	1	2	3	4	5	6	7
Water generation from unvented burning (gal/wk)	322	405	299	172	103	82	79
Water from birds (gal/wk)	1078	3206	5772	8443	10926	12964	14319
Proportion from propane (%)	23	11	5	2	1	1	1

MOISTURE — continued on pg. 6

References

ASHRAE. 1985. ASHRAE Handbook, 1985 Fundamentals. American Society of Heating, Refrigerating and Air-Conditioning Engineers. Atlanta, GA.

Czarick, M. B. Fairchild, and D. Dartnell 2008. Alternative heating system … an overview. University of Georgia Corporative Extension Service, Poultry Housing Tips. December 2008.

Tabler, T. 2000. Brooding chicks in colder weather. Avian Advice 2(1):3-4.

Tabler, T. 2003. Early feed intake and bird performance. Avian Advice 5(1):13-15.

Tabler, T., S. E. Watkins and F. T. Jones. 2008. Litter preparation between flocks: management is the key. Avian Advice 10(4):4-7.

Wimberly, J. 2008. A review of biomass furnaces for heating poultry houses in the Northwest Arkansas region. Winrock International, Little Rock, AR.

Figure 1. Weekly Water Released and Retained (reflected as weight gain) per 1000 Birds as a Result of Feed and Water Intake.

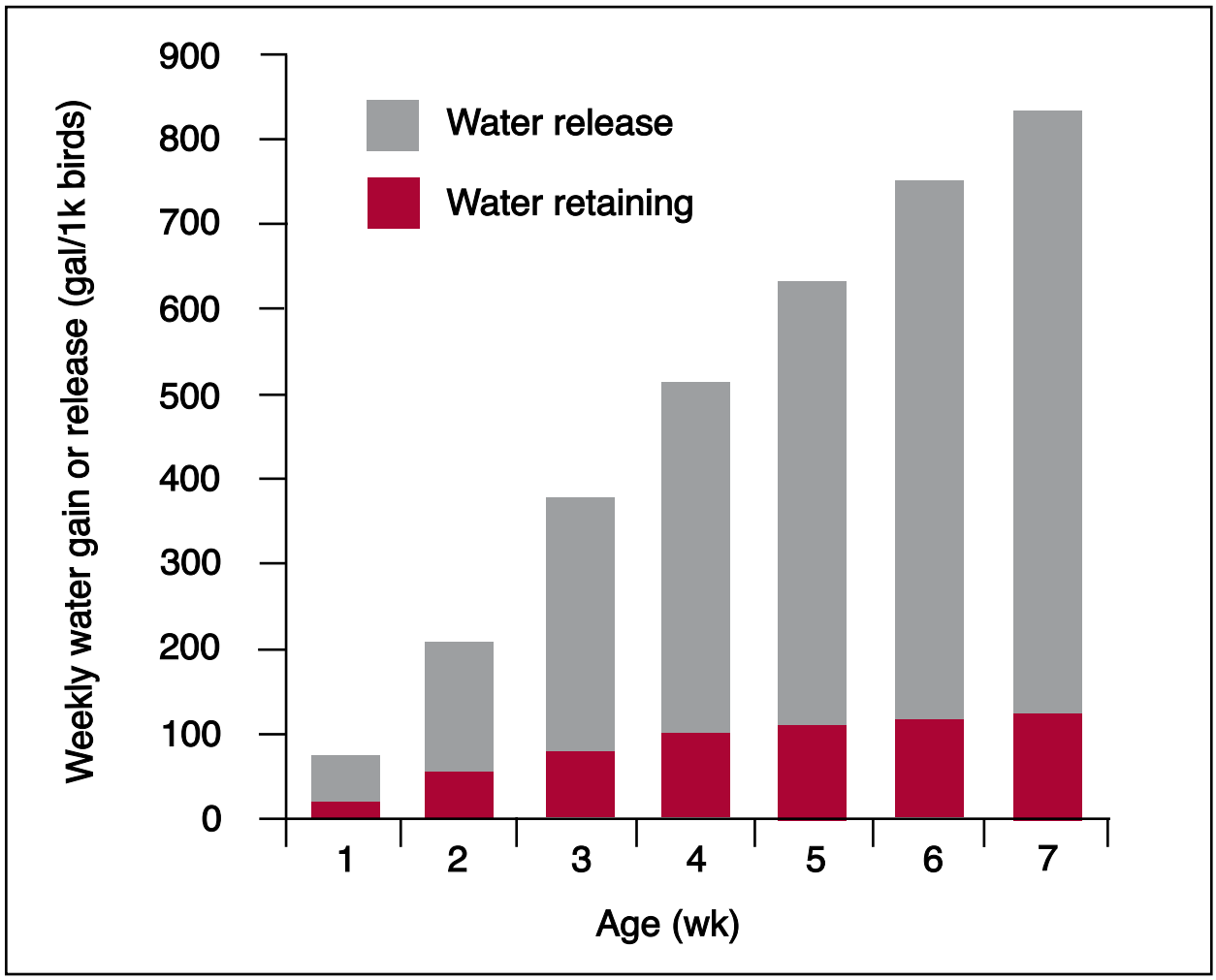


Figure 2. Weekly Moisture Addition from Water Released by Birds and Generated by Propane Heaters (analyzed for 18 winter flocks, per house basis)

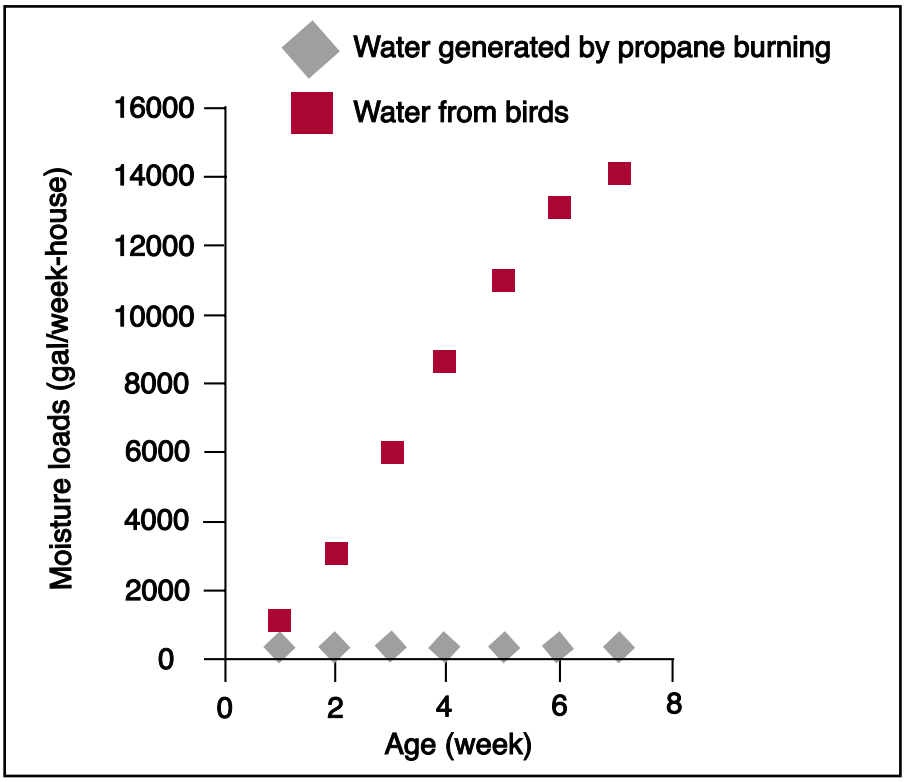
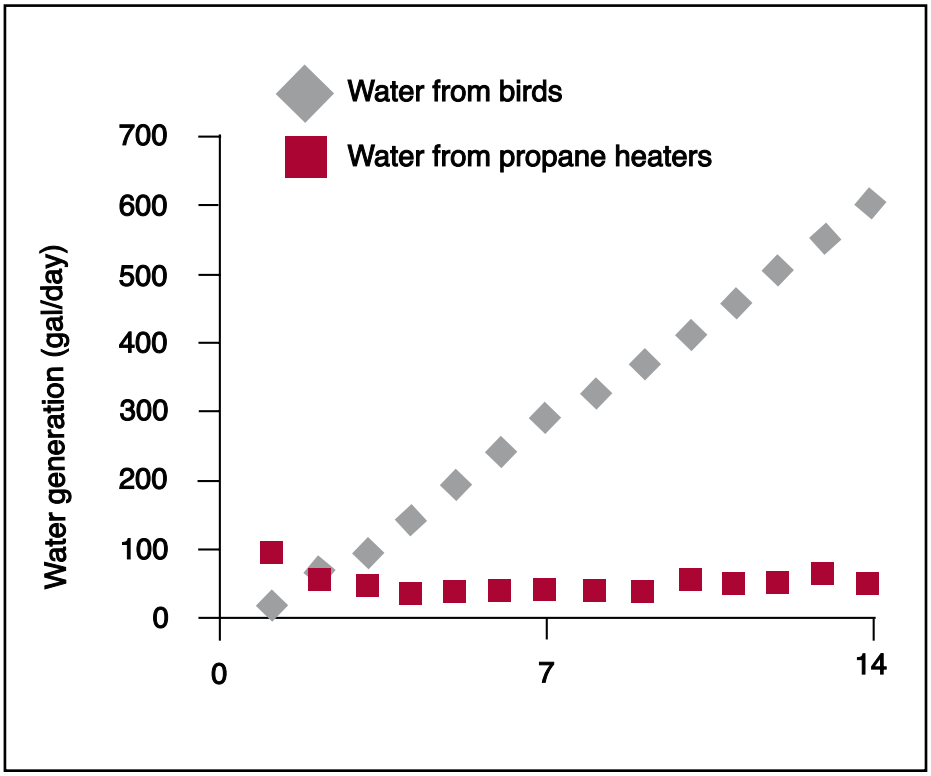


Figure 3. Daily Moisture Addition from Water Released by Birds and Generated by Propane Heaters during the First Two Weeks after Chick Placement (analyzed on 5 winter flocks per house basis)



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How Does Taste Influence Water Consumption in Broilers?

Background

Early studies suggest that birds are much more sensitive to flavors in water than in feed (Kare and Pick, 1960). This sensitivity to flavors in water may be due to the fact that birds consume almost twice as much water as feed. However, the issue of taste is much more complex than it may seem because humans perceive taste differently than many other animal species.

To illustrate this point one researcher compared the responses of different animals to a sucrose (sugar) solution, and its equivalent in saccharine. Most humans said that both solutions are sweet and pleasant tasting and laboratory rats had a similar reaction. Calves drank much more of the sucrose than humans did, but drank little of the saccharine. Chickens and dogs drank the sugar but found the saccharine very offensive. Cats did not respond to either of the solutions. The point of this illustration is we, as humans, cannot use our own sense of taste to predict how animals will respond (Kare, 1970).

Chickens, in fact, prefer water that is cold and slightly acid in taste rather than sweet (Kare, 1970). Although chicks have only a fraction of the number of taste buds found in other animals (Figure 1), birds have a well defined sense of taste and will reject certain flavors (Kare et al., 1957). In addition, the taste buds in chickens are in different locations as compared to other animals. In humans, and many other animal species, most taste buds are on the tongue; but in the chicken, taste buds are distributed primarily on the back part of the roof of the mouth, with only 2 to 4% being located on the tongue (Ganchrow and Ganchrow, 1985). In fact, the taste buds in chickens are so far back in the mouth that by the time the bird can taste something, it is almost too late to change its mind about swallowing it (Kare, 1970). Yet, the sense of taste is more than just how feed or water feels in the mouth of the bird. The sense of taste is all the sensation a bird experiences after consumption.

In general, the sense of taste guides an animal as to what it should eat. For example, chickens given a thiamin deficient diet and offered two solutions, one with and one without thiamin, will choose to drink a solution containing thiamin. While humans perceive xylose as about 70% as sweet as sucrose (sugar), chickens will drink little xylose, which has been found to cause cataracts in some bird species (Kare, 1970). These and similar choices suggest that taste is often the basis on which the bird seeks to meet its nutritional needs (Roura et al., 2008). However, the problem is still more complicated.

Water to humans is wet and tasteless, but to birds, water has a distinct taste. Therefore, water in itself is a strong stimulus for the bird and flavors tested in water solutions are actually perceived by the bird as mixtures of flavors (Beidler, 1961; Kare, 1970; Gentle, 1985). Although flavor perceptions in many animals also involve the perception of odors, in birds odors in their immediate environment have little apparent affect. Yet, temperature of water can be critical for birds. When presented with two choices of water, one at room temperature

and the other a degree or two above their body temperature, birds will suffer from acute thirst rather than drink the warmer water. On the other hand, birds will readily consume water at temperatures close to freezing. This may be due to the fact that birds are well insulated with feathers, which protect them from the cold, but allow little or no means to dissipate excess body heat.

Practical applications

The data in Figure 2 were collected by Kare et al., (1957), who tested acceptance of water containing various flavors by placing two chick watering jars in each pen. One jar contained untreated water and the other contained flavored water. The researchers compared the amount of water consumed from the two jars to measure the acceptance or rejection of flavors by the birds. Some flavors (strawberry, alfalfa, nutmeg, honey, molasses, mushroom, and wild cherry) were rejected outright, while birds would drink certain flavors (butter pecan, butterscotch, raisin, coconut, grenadine, oil of patchouli, and colocynth pulp) sparingly at first, but gradually accept the flavor as illustrated by Figure 2. Other than the novelty of knowing how flavored water influences the taste of chickens, is there a practical application for this information? Absolutely. The taste of water due to either natural or added materials can dramatically influence consumption, particularly in young birds.

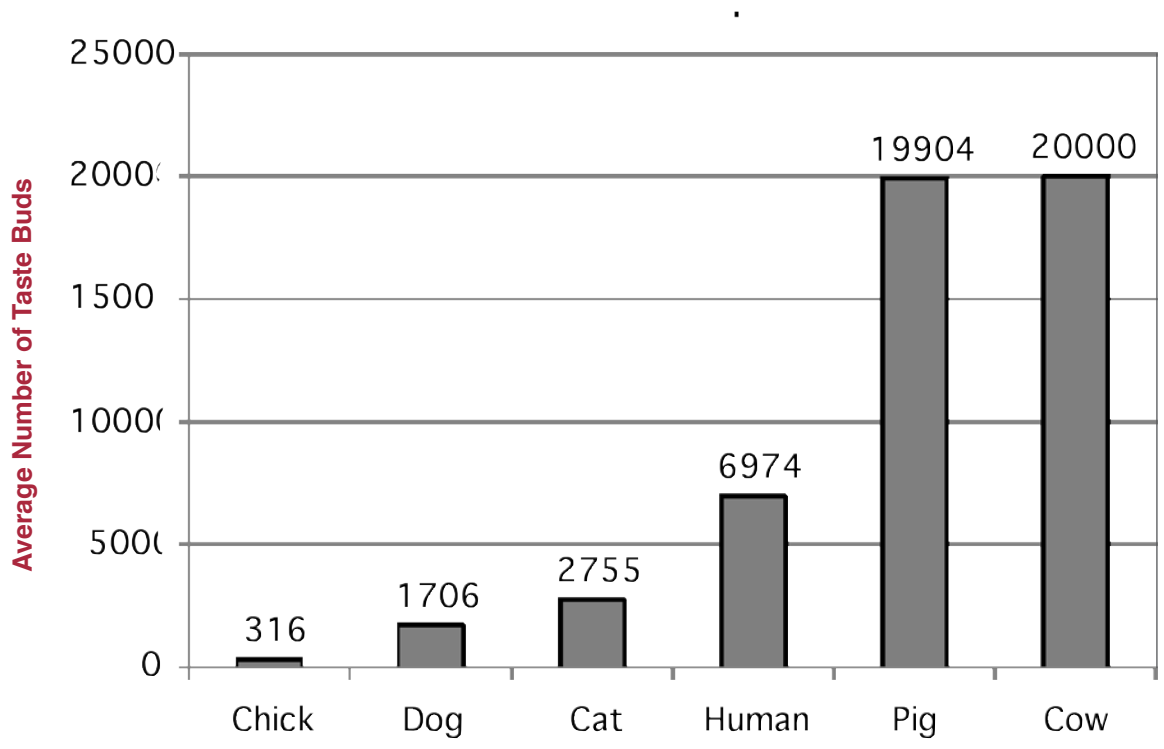
We witnessed firsthand the effects of differences in water consumption in young birds at the U of A Applied Broiler Research Farm when we tried a different water acidifier (Figure 3). The three flocks grown on product B were lighter at settlement than previous flocks grown on product A. Yet, overall water consumption data for these flocks showed no difference. However, data for the first week showed lower water consumption for flocks grown on product B as compared to product A and it took almost 21 days before the birds returned to consumption seen on product A. We were fortunate that we were raising a heavier bird and the additional time given to the birds to become acclimated to product B allowed us to make up some performance by the time they went to market. However, growers raising smaller weight birds would not have the luxury of making up for poor early water and feed consumption.

How can growers identify water consumption challenges?

If birds don't eat they don't gain weight. Since feed and water consumption are closely correlated (1 pound of feed consumed for approximately 1.67 pounds of water consumed) it is critical to pay attention to water consumption and head off problems before they start. As illustrated in Figures 2 and 3, when birds gradually accept water with certain flavors

TASTE — continued on page 10

Figure 1. Number of Taste Buds in Various Animal Species[1]



[1]Adapted from Roura et al, 2008

particularly early in the life of the flock, detection may be much more difficult, but the losses can be just as real (Tabler, 2003). In view of this situation, the following suggestions are offered:

1. Closely monitor water consumption, particularly early in the flock. Install meters in both the front and back of the house. Readings from these meters provide crucial information to determine if birds are properly spread through the house as well as determine if water lines are correctly adjusted. At about the same time each day, record water meter readings starting from day one of the flock. Identifying and solving water issues can more than pay for the cost of meters.
2. Develop water usage patterns. Since water consumption will likely vary from farm to farm, develop average water consumption charts for your farm. Compare each flock's consumption numbers to the average you have developed and pay particular attention early in the life of the flock.
3. Be aware that not all water supplies and water additives are compatible to the bird's taste. Pay close attention to water usage when trying new products to assure that there is no decrease in water usage. Make a note of products which the birds appear to like due to increased consumption which is not accompanied by flushing in the birds.

Conclusion

The factors influencing the sense of taste in birds are complex and not completely understood. However, it is clear that the taste of water can influence both feed and water consumption. By monitoring water usage and understanding what normal water usage patterns are for each day of age, producers can identify challenges and correct them before profits are lost.

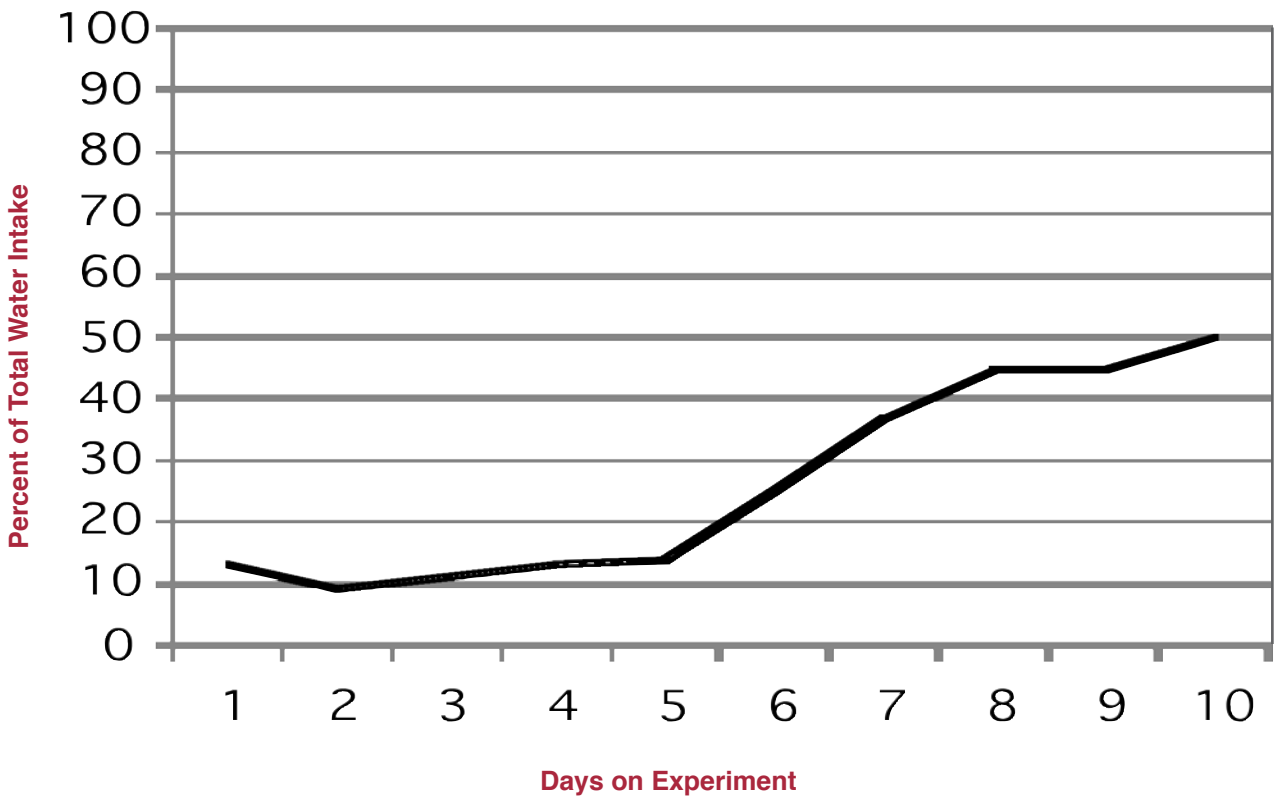
References

Beidler, L. M. 1961. The chemical senses. Ann. Rev. Psychol. 12:363-388.

Ganchrow, D. and J. R. Ganchrow. 1985. Number and distribution of taste buds in the oral cavity of hatchling chicks. Physiol. Behav. 34(6):889-894.

Gentle, M. J. 1985. Sensory involvement in the control of food intake in poultry. Proc. Nutr. Soc. 44:313-321.

Figure 2. Daily Water Consumption in Chickens Provided Flavored Water[1,2]



[1]Adapted from Kare et al. 1957 The Sense of Taste in the Fowl. Poultry Science 36:129-138
[2]Birds were given a choice of unflavored water or water containing 4 parts per thousand butter pecan flavor, these data represent the percentage of flavored water consumed.

Kare, M. R. 1970. The chemical senses of birds. Bird Control Seminars Proceedings http://digitalcommons.unl.edu/cgi/viewcontent.cgi?article=1183&context=icwdmbirdcontrol Accessed 3/24/09

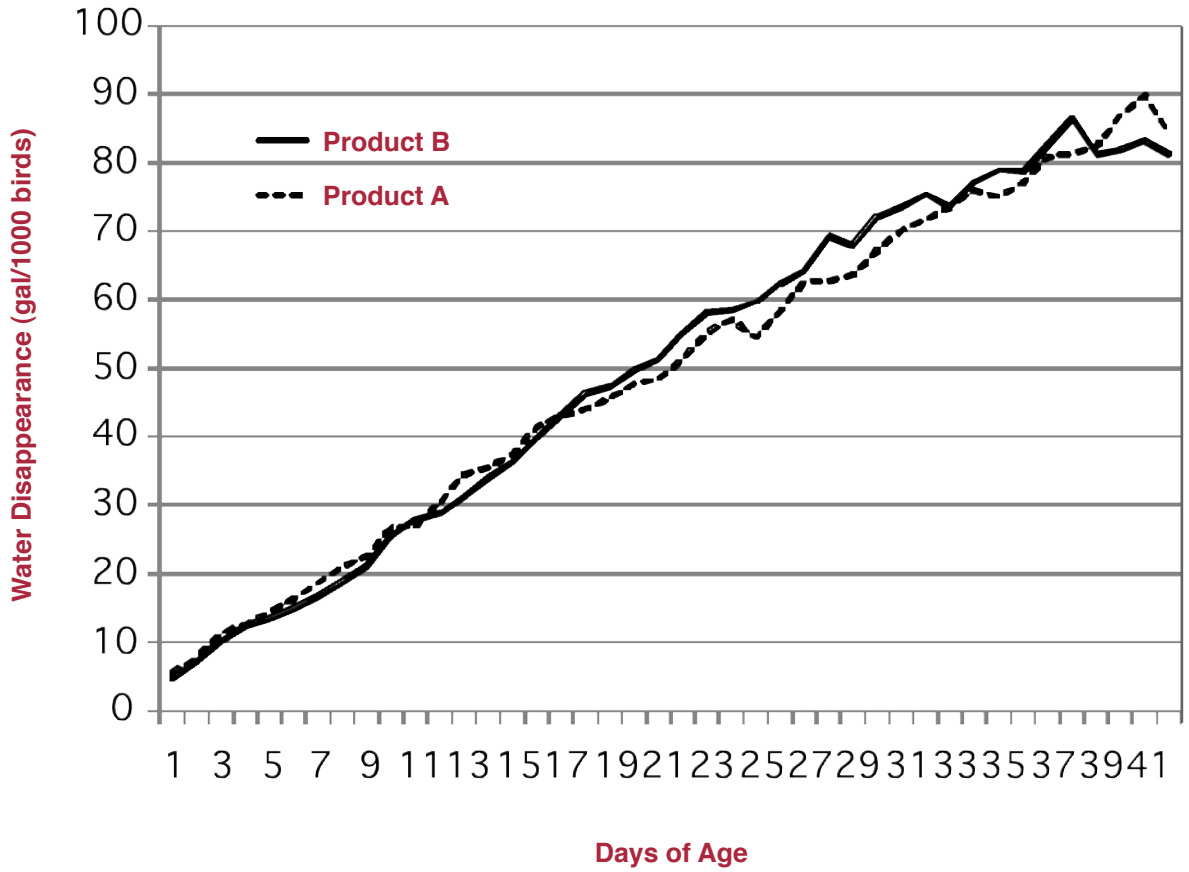
Kare, M. R., R. Black and E. G. Allison. 1957. The sense of taste in the fowl. Poultry Sci. 36:129-138.

Kare, M. R. and H. L. Pick. 1960. The influence of the sense of taste on feed and fluid consumption. Poultry Sci. 39:697-706.

Roura, E., B. Humphrey, G. Tedo and I. Ipharraguerre. 2008. Unfolding the codes of short-term feed appetence in farm and companion animals: A comparative oronasal nutrient sensing biology review. Can. J. Animal Sci. 88:535-558.

Tabler, G. T. 2003 Early feed intake and bird performance. Avian Advice 5:13-15

Figure 3. Water Usage With Different Water Acidifier Products.



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