



Health  
Canada

Santé  
Canada

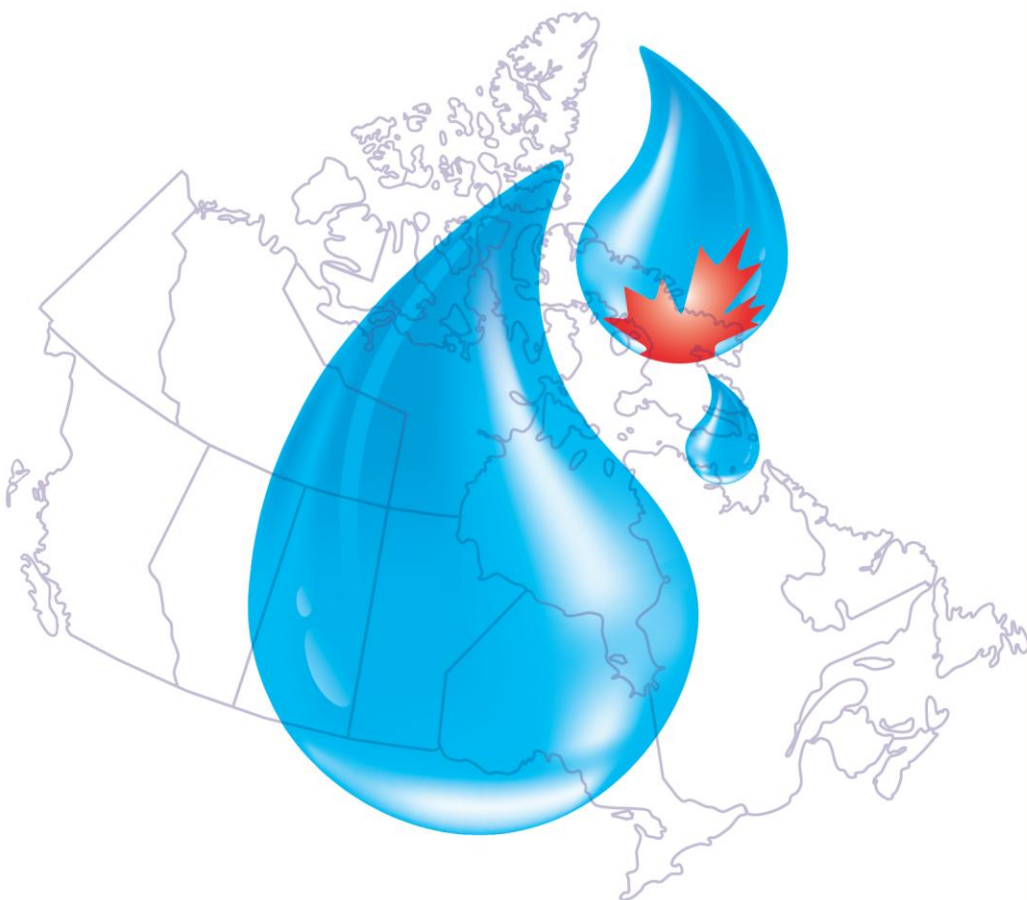
*Your health and  
safety... our priority.*

*Votre santé et votre  
sécurité... notre priorité.*

# Guidelines for Canadian Drinking Water Quality

Guideline Technical Document

**Ammonia**



Canada

***Health Canada is the federal department responsible for helping the people of Canada maintain and improve their health. We assess the safety of drugs and many consumer products, help improve the safety of food, and provide information to Canadians to help them make healthy decisions. We provide health services to First Nations people and to Inuit communities. We work with the provinces to ensure our health care system serves the needs of Canadians.***

Published by authority of the Minister of Health.

*Guidelines for Canadian Drinking Water Quality: Guideline Technical Document – Ammonia*

is available on Internet at the following address:  
[www.healthcanada.gc.ca](http://www.healthcanada.gc.ca)

Également disponible en français sous le titre :  
*Recommandations pour la qualité de l'eau potable au Canada : Document technique – L'ammoniac*

This publication can be made available on request in a variety of alternative formats.

© Her Majesty the Queen in Right of Canada,  
represented by the Minister of Health, 2013

This publication may be reproduced without permission provided the source is fully acknowledged.

Pub. Number: 130477  
Cat.: H144-13/5-2013E-PDF  
ISBN: 978-1-100-23002-3

# **Guidelines for Canadian Drinking Water Quality**

Guideline Technical Document

## **Ammonia**

**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**

**June, 2013**

This document may be cited as follows:

Health Canada (2013). Guidelines for Canadian Drinking Water Quality: Guideline Technical Document — Ammonia. Water and Air Quality Bureau, Healthy Environments and Consumer Safety Branch, Health Canada, Ottawa, Ontario. (Catalogue No H144-13/5-2013E-PDF).

The document was prepared by the Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment.

---

Any questions or comments on this document may be directed to:

Water and Air Quality Bureau  
Healthy Environments and Consumer Safety Branch  
Health Canada  
269 Laurier Avenue West, Address Locator 4903D  
Ottawa, Ontario  
Canada K1A 0K9

Tel.: 613-948-2566

Fax: 613-952-2574

E-mail: [water\\_eau@hc-sc.gc.ca](mailto:water_eau@hc-sc.gc.ca)

Other Guideline Technical Documents for the Guidelines for Canadian Drinking Water Quality can be found on the following web page: [www.healthcanada.gc.ca/waterquality](http://www.healthcanada.gc.ca/waterquality)

---

## Table of Contents

<b>Part I. Overview and Application.....</b>	<b>1</b>
1.0 Guideline.....	1
2.0 Executive summary.....	1
2.1 Health effects .....	1
2.2 Exposure .....	1
2.3 Analysis and treatment.....	2
3.0 Application of the guideline.....	2
3.1 Monitoring .....	2
<b>Part II. Science and Technical Considerations .....</b>	<b>3</b>
4.0 Identity, use and sources in the environment.....	3
4.1 Ammonia and drinking water treatment and distribution.....	4
4.1.1 Ammonia in raw water.....	4
4.1.2 Use of ammonia for chloramination .....	4
4.2 Environmental fate.....	5
4.2.1 Water.....	5
4.2.2 Air .....	5
4.2.3 Soil .....	5
5.0 Exposure .....	6
5.1 Water.....	6
5.2 Food .....	7
5.3 Air .....	8
5.4 Consumer products .....	8
5.5 Soil .....	8
6.0 Analytical methods .....	9
6.1 U.S. EPA methods and Standard Methods .....	9
6.2 Other available methods .....	10
7.0 Treatment technology .....	10
7.1 Municipal scale.....	10
7.1.1 Biological treatment (controlled nitrification).....	11
7.1.2 Breakpoint chlorination .....	13
7.1.3 Ion exchange .....	14
7.1.4 Membrane filtration .....	15
7.1.5 Combination of reverse osmosis and biological treatment.....	17
7.1.6 Air stripping.....	17
7.1.7 Emerging technologies.....	17
7.1.8 Nitrification in the distribution system .....	18
7.2 Residential scale.....	22

8.0	Kinetics and metabolism.....	23
8.1	Absorption.....	23
8.2	Distribution and metabolism.....	24
8.3	Excretion.....	25
9.0	Health effects.....	25
9.1	Effects in humans.....	25
	9.1.1 Acute toxicity.....	25
	9.1.2 Subchronic and chronic toxicity and carcinogenicity.....	26
	9.1.3 Neurotoxicity.....	26
	9.1.4 Genotoxicity.....	26
9.2	Effects on experimental animals.....	27
	9.2.1 Acute toxicity.....	27
	9.2.2 Short-term exposure.....	27
	9.2.3 Long-term exposure and carcinogenicity.....	28
	9.2.4 Genotoxicity.....	28
	9.2.5 Reproductive and developmental toxicity.....	29
	9.2.6 Neurotoxicity.....	29
9.3	Mode of action.....	29
10.0	Classification and assessment.....	29
10.1	International considerations.....	30
11.0	Rationale.....	30
12.0	References.....	30
	Appendix A: List of acronyms.....	42
	Appendix B: Analytical methods for the determination of ammonia-nitrogen in drinking water	43

---

# Ammonia

## **Part I. Overview and Application**

### **1.0 Guideline**

*It is not considered necessary to establish a health-based guideline for ammonia, based on its low toxicity at concentrations found in drinking water. Levels of ammonia, either naturally present in the source water or added as part of a disinfection strategy, can affect water quality in the distribution system (e.g., nitrification) and should be monitored.*

### **2.0 Executive summary**

The production of ammonia is a normal part of metabolism, and levels occurring in the human body from metabolic processes generally exceed concentrations found in drinking water. The concentration of free ammonia entering the distribution system can lead to nitrification and the potential increase of nitrate and nitrite in drinking water. Further information can be found in the guideline technical document on nitrate and nitrite in drinking water.

This guideline technical document reviews and assesses all identified health risks associated with ammonia in drinking water. Based on this review, and taking into consideration the lack of an appropriate endpoint from the ingestion of ammonia, the lack of sufficient evidence of systemic effects in humans and limited relevant studies in experimental animals, it has been found that no health-based guideline can be derived for ammonia in drinking water.

### **2.1 Health effects**

The mode of action for ammonia is dependent on the route of exposure. Although limited information is available for the oral route of exposure, the information that does exist suggests that the oral route of exposure should be considered independently from the inhalation route of exposure. In humans, most health effects reported as a result of ammonia exposure are associated with exposure through inhalation. Although ingestion of concentrated ammonia causes irritation and damage to the mouth, throat and gastrointestinal tract, these effects are unlikely to occur at the levels of ammonia found in drinking water.

Ammonia is naturally produced and metabolized by the human body. Levels of ammonia present in the body are much greater than levels typically found in drinking water. No adverse health effects have been associated with the ingestion of ammonia at levels found in drinking water.

### **2.2 Exposure**

Since ammonia occurs naturally in the environment, humans are regularly exposed to low levels of ammonia through water, food, air, consumer products and soil. However, ammonia is produced in the intestinal tract at levels significantly higher than from all exposure media.

Ammonia is commonly found in surface water and rainwater. Groundwater generally contains low concentrations of ammonia, but some deep wells affected by specific geological formations have been shown to have high concentrations of ammonia. The level of ammonia in surface water varies regionally and seasonally and can be affected by localized anthropogenic influences, such as runoff from agricultural fields or industrial or sewage treatment discharges.

Ammonia may also be added to treated water as part of the disinfection strategy to form chloramines as a secondary disinfectant.

### **2.3 Analysis and treatment**

Ammonia can be detected in drinking water through a number of well-established methods. The choice of the method used will depend on the presence of interferences. The presence of naturally-occurring ammonia can reduce the efficiency of treatment processes. In municipal water treatment plants, ammonia can be removed through biological treatment (controlled nitrification) and physicochemical processes such as breakpoint chlorination, ion exchange and membrane filtration. It is important to minimize the levels of ammonia entering the distribution system to help prevent nitrification, water quality degradation and possible corrosion issues. At the residential level, although there are no certified residential treatment devices currently available for the reduction of ammonia levels in drinking water, treatment devices using reverse osmosis or ion exchange may be effective.

## **3.0 Application of the guideline**

*Note: Specific guidance related to the implementation of drinking water guidelines should be obtained from the appropriate drinking water authority in the affected jurisdiction.*

Free ammonia entering the distribution system can be one of the causative factors of nitrification and the potential increase of nitrate and nitrite in the distribution system. The health effects of nitrite and nitrate in drinking water and their respective MACs are fully discussed in the guideline technical document on nitrate and nitrite. Good operational practices will help prevent nitrification. These practices include limiting excess free ammonia entering the distribution system to concentrations below 0.1 mg/L (measured as ammonia-nitrogen), and preferably below 0.05 mg/L (measured as ammonia-nitrogen). Utilities using ammonia as part of their disinfection strategy should ensure that the appropriate chlorine to ammonia ratio is maintained. Changes in free ammonia concentrations can be an indication that nitrification is occurring; thus, monitoring should be conducted both at the treatment plant and in the distribution system.

### **3.1 Monitoring**

Utilities should characterize their source water to assess the presence of ammonia and the variability of ammonia levels. Utilities that are chloraminating or that have ammonia in their source water should monitor for free ammonia, in addition to other parameters (e.g., total chlorine residual, nitrite, heterotrophic plate counts), at key locations in the distribution system. It is recommended that free ammonia be monitored daily in the plant effluent. In addition, it is recommended that source water, locations such as reservoir outlets and areas with long water detention times (e.g., dead ends) be monitored weekly. Changes in the trend of priority nitrification parameters in the distribution system, such as total chlorine residual, nitrite and nitrate, should trigger more frequent monitoring of free ammonia. Utilities that undertake comprehensive preventive measures and have baseline data indicating that nitrification does not occur in the system may conduct less frequent monitoring of free ammonia.



## **Part II. Science and Technical Considerations**

### **4.0 Identity, use and sources in the environment**

Ammonia (CAS number 7664-41-7, chemical formula  $\text{NH}_3$ ) is a colourless gas at room temperature, with a penetrating, sharp, pungent odour. Ammonia gas ( $\text{NH}_3$ ) can be compressed and become a liquid under pressure. When ammonia is dissolved in water, it exists in two forms simultaneously: the non-ionized form ( $\text{NH}_3$ ) and the ammonium cation ( $\text{NH}_4^+$ ). The equilibrium between the two species is governed in large part by pH and temperature. The sum of the two forms is known as total ammonia (also referred to as free ammonia). For drinking water monitoring purposes, total ammonia refers to all of the ammonia species, including free ammonia, monochloramine ( $\text{NH}_2\text{Cl}$ ), dichloramine ( $\text{NHCl}_2$ ) and trichloramine (or nitrogen trichloride –  $\text{NCl}_3$ ). Ammonia is very soluble in water and has a high vapour pressure (Table 1). The odour threshold is 1.5 mg/L in water (Environment Canada and Health Canada, 1999; ATSDR, 2004; HSDB, 2005).

**Table 1.** Physicochemical properties of ammonia

<b>Property</b>	<b>Value<sup>a</sup></b>
Molecular mass	17.03 g/mol
Solubility	421 g/L at 20°C
Boiling point	-33.4°C
Melting point	-77.7°C
Vapour pressure	882 kPa at 20°C
Water solubility	47% at 0°C and 31% at 25°C
Log <i>n</i> -octanol/water partition coefficient ( $K_{ow}$ )	Experimental data not available
Henry's law constant ( $K_{aw}$ )	0.0006 at 20°C <sup>b</sup>

<sup>a</sup> Values as reported in ATSDR (2004); HSDB (2005); <sup>b</sup> Value as reported in Crittenden et al. (2005)

Ammonia occurs in air, soil and water as a result of natural processes or industrial activities, including certain types of intensive farming. Ammonia is an important source of nitrogen, which is essential for plants and animals and plays an important role in protein synthesis (Environment Canada and Health Canada, 1999; Xia et al., 2011; Zehr and Kudela, 2011).

Ammonia produced naturally by the decay of organic materials from plants, dead animals and other organisms accounts for the largest proportion of the ammonia in the environment. The sources of ammonia in the soil are diverse, including natural or synthetic fertilizers, degradation of livestock excrement, decay of organic material from dead plants and animals, and, indirectly, from natural fixation of atmospheric nitrogen by free-living nitrogen-fixing bacteria (ATSDR, 2004; Xia et al., 2011). Common anthropogenic sources of ammonia in drinking water sources are agricultural/fertilizer runoff and wastewater effluent.

Ammonia is used in fertilizers for animal feed production and in the manufacture of fibres, plastics, explosives, paper and rubber. As a fertilizer, ammonia is applied directly onto soil on farm fields, lawns and plants (Environment Canada and Health Canada, 1999; ATSDR, 2004; Xia et al., 2011). A high percentage of the ammonia and ammonia compounds produced commercially are used for the production of fertilizers (ATSDR, 2004).

Outside of the fertilizer industry, small volumes of ammonia are consumed in several specific industrial applications: as a modifying reagent in the flotation of phosphate ores, as a corrosion inhibitor at petroleum refineries and natural gas plants, as a stabilizer in rubber production, as a curing agent in leather manufacture and as a coolant in metal processing. Ammonia is also used in municipal and industrial water treatment and in the manufacture of food and beverages, certain pharmaceuticals, household cleaners and detergents, and numerous organic and inorganic chemicals, such as cyanides, amides, amines, nitrites and dye intermediates (Camford Information Services, 2003). Treated wastewater effluent may be a potential source of ammonia and other nitrogen-containing compounds in surface waters.

The total manufacturing capacity of ammonia in Canada was estimated at 3887 kilotonnes in 1988 and 5601 kilotonnes in 2000 and remained unchanged through 2002 (most recent data available). The amount of ammonia imported by the ammonia industry is less than 1% of the Canadian market needs (Camford Information Services, 2003).

#### **4.1 Ammonia and drinking water treatment and distribution**

Ammonia is one of the unique parameters in that it is not only potentially present in source water but also, in some cases, intentionally added to drinking water. Both these situations can have important implications for the drinking water treatment and distribution systems. As the main objective of this document is to focus on the health effects related to exposure to ammonia in drinking water supplies, a full review of chloramination, nitrification or other implications related to ammonia and drinking water treatment will not be provided here.

##### *4.1.1 Ammonia in raw water*

Ammonia present in the raw water creates a high oxidant demand and decreases disinfection efficiency. The reaction between ammonia and chlorine is very rapid, and ammonia may negatively affect the removal of organic and inorganic compounds such as iron, manganese and arsenic by reducing chlorine's availability for oxidation (Lytle et al., 2007; White et al., 2009).

##### *4.1.2 Use of ammonia for chloramination*

Ammonia may also be added to treated water as part of the disinfection strategy to form chloramines as a secondary disinfectant. Where chloramination is practised, the addition of an excess amount of ammonia or an inappropriate chlorine to ammonia-nitrogen ( $\text{Cl}_2:\text{NH}_3\text{-N}$ ) weight ratio may result in the presence of free ammonia in the finished water. Ammonia may also be released as a result of chloramine demand and decay in the distribution system or may be formed from the reaction between nitrate and metal pipe surfaces (U.S. EPA, 2002; Harrington et al., 2003; Edwards and Dudi, 2004; Huang and Zhang, 2005; Zhang et al., 2009). Ammonia may also be released from the cement mortar coating of water distribution pipes and cause water quality issues in the distribution system (WHO, 2003). Free ammonia entering the distribution system can be one of the principal causative factors of nitrification, which is responsible for significant water quality degradation (U.S. EPA, 2002). Nitrification is a two-step process involving the aerobic oxidation of ammonia to nitrite by ammonia-oxidizing bacteria (AOB) and the further oxidation of nitrite to nitrate by nitrite-oxidizing bacteria (NOB) (Kirmeyer et al., 1995, 2004; U.S. EPA, 2002).

Ammonium cations and ammonia exist in equilibrium in water, depending upon the pH and temperature. At 20°C, the ammonium ion predominates in drinking water below 9.3, whereas ammonia is mainly found at or above pH 9.3 (Baribeau, 2006). A pH adjustment can be

used to influence the form of ammonia in the water (Department of National Health and Welfare, 1993). It is important to account for the ammonia concentration in the source water when establishing the ammonia dosage for chloramination (Muylywyk, 2009; Shorney-Darby and Harms, 2010).

## 4.2 Environmental fate

The physical and chemical properties of ammonia are pH dependent. Consequently, environmental fate processes that influence the transport and partitioning of ammonia will also be pH dependent. Ammonia is essential in nature's biological cycles and is necessary for making deoxyribonucleic acid (DNA), ribonucleic acid (RNA) and proteins.

### 4.2.1 Water

Ammonia is in equilibrium with the ammonium ion in water. This equilibrium is highly dependent on pH and, to a lesser extent, temperature (Weast et al., 1988). The equilibrium favours the ammonium ion in acidic or neutral waters. If present in surface waters, ammonia can partially volatilize to the atmosphere; this phenomenon is affected by pH, temperature, wind speed and the atmospheric ammonia concentration. Ammonia present in air can readily dissolve in rainwater as a result of its high water solubility. Ammonia can also be removed by microbial processes or adsorb to sediment and suspended organic material. In surface water or groundwater, ammonia can undergo sequential transformation by two processes in the nitrogen cycle: nitrification and, to a lesser extent, denitrification. Nitrite and nitrate formed from the aerobic process of nitrification can be taken up by aquatic plants or other organisms. Elemental nitrogen formed from the anaerobic process of denitrification is lost by volatilization to the atmosphere (Environment Canada and Health Canada, 1999; ATSDR, 2004). Treated wastewater effluent may be a potential source of ammonia and other nitrogen-containing compounds in surface waters (Skadsen and Cohen, 2006).

### 4.2.2 Air

Ammonia can rapidly react with acidic substances in air, such as nitric or sulphuric acid, to form ammonium aerosols (Bouwman et al., 1997), which can subsequently be removed from the atmosphere by dry or wet deposition. This removal mechanism is more important in industrialized areas, where air contains more acidic pollutants, than over rural locations (Goulding et al., 1998). Overall, dry deposition processes predominate where there are high amounts of ammonia emissions; conversely, wet deposition of particulate ammonium predominates where ammonia emissions are lower (Asman et al., 1998).

### 4.2.3 Soil

Ammonia contained in soil or sediments may volatilize to the atmosphere, adsorb to particulate matter or be taken up by plants and microorganisms as a nutrient source and converted to organic nitrogen compounds. It can be rapidly transformed to nitrate by the microbial population through nitrification (Atlas and Bartha, 1998). The nitrate formed will either leach through the soil or in turn be assimilated by plants or other microorganisms. Ammonia at natural concentrations in soil is not believed to have a very long half-life. In fact, following application of an ammonia-containing fertilizer to a soil, the amount of ammonia in that soil decreased to low levels in a few days. However, very high localized concentrations of ammonia (spill or excessive application of fertilizers) inhibit nitrogen transformation by microbial processes. Under these conditions, other physical and chemical processes, including

binding to soil particles and volatilization to the atmosphere, will dictate the fate of ammonia, until the concentration returns to background levels (Atlas and Bartha, 1998).

## 5.0 Exposure

As ammonia occurs naturally in the environment, humans are regularly exposed to low levels of ammonia through exogenous sources (air, soil, food, consumer products and water), estimated to be less than 20 mg/day (IPCS, 1986). However, it should be noted that the endogenous production of ammonia in the intestinal tract is significantly greater (> 4 g/day) (Summerskill and Wolpert, 1970).

### 5.1 Water

Ammonia is commonly found in surface water and rainwater. The level of ammonia in surface water varies regionally and seasonally and can be affected by localized anthropogenic influences, such as runoff from agricultural fields or industrial or sewage treatment discharges. The ammonia concentrations in rivers and bays are usually less than 6 mg/L; higher levels may indicate anthropogenic pollution (Bouwer and Crowe, 1988).

Because of the cation exchange capacity of soil, groundwater generally contains low concentrations of ammonia; natural levels are usually below 0.2 mg/L (Bouwer and Crowe, 1988). However, deep wells affected by some geological formations have been shown to have high concentrations of ammonia (Schilling, 2002). Some utilities across the United States and Canada have reported elevated levels of ammonia in groundwater sources above 2 mg/L (Schilling, 2002; Rezania, 2010; Schulz, 2010). A study of 119 wells in Minnesota found that ammonia levels averaged 0.86 mg/L, with a maximum concentration of 7.15 mg/L (Rezania, 2010; Schulz, 2010).

Ammonia can be present in drinking water as a result of its addition for the purpose of disinfection with chloramines (Bouwer and Crowe, 1988).

Monitoring data (1998–2008) for 1119 raw water samples collected from Alberta communities indicated that the ammonia content in 60% of the samples was below the method detection limit (MDL) of the analytical methods used (0.001–0.050 mg/L), with an average concentration of 0.20 mg/L. Although a maximum concentration of 20.8 mg/L was determined at one location, less than 1% of the concentrations were above 2 mg/L. Ammonia was detected in less than 9% of the 227 surface water (dam) samples collected in Calgary (2000–2010), with a maximum concentration of 0.12 mg/L; no major seasonal variations were observed. Ammonia was not detected (MDL of 0.02–0.04 mg/L) in the treated water. From 1998 to 2010, 1286 chloraminated water samples were taken at two Alberta water reservoirs. The samples collected and analysed over this period had average and maximum concentrations of ammonia of 0.20 mg/L and 0.53 mg/L, respectively, for both reservoirs. A 2004 facility assessment indicated that 40 groundwater systems with naturally occurring ammonia had concentrations ranging from 0.2 to 1.5 mg/L with an average of 0.6 mg/L (Alberta Environment, 2010).

Data from 1605 samples, including groundwater and surface water, collected from various locations in Nova Scotia (1999–2009) showed no detectable levels of ammonia in 86% and 94% of the groundwater and surface water samples, respectively. Average concentrations were 0.09 mg/L and 0.06 mg/L for groundwater and surface water, respectively, with a maximum of 9.5 mg/L detected in a drilled well sample and 0.38 mg/L in surface water (Nova Scotia Environment, 2010).

Monitoring data collected from 2002 to 2010 in Manitoba, indicated that 91% of the 931 tested raw surface water samples and 83% of the 640 treated surface water samples had detectable levels of ammonia (MDL of 0.003 mg/L). Ammonia concentrations ranged from 0.003 mg/L to 4.42 mg/L (average of 0.34 mg/L) and from 0.003 to 3.62 mg/L (average of 0.19 mg/L) in the raw and treated surface water, respectively. The data indicated that 84% of the raw well water samples showed detectable ammonia concentrations in the range of 0.01 to 2.2 mg/L (average of 0.56 mg/L). Ammonia concentrations ranged from 0.41 to 0.69 mg/L (average of 0.59 mg/L) in 4 of the 8 treated well water samples. The remaining 4 treated well water samples had no detectable ammonia. Monitoring data for 10 wells (2002-2008) indicated ammonia concentrations from these wells ranged from 0.05 to 2.44 mg/L (average of 1.0 mg/L). However, no information on the water characteristics (raw vs. treated) was provided for these wells.

Ammonia was detected (MDL of 0.003 mg/L) in 393 raw and treated water samples analysed between 2009 and 2011 in Manitoba, however, the source of the water (i.e., well or surface water) was not provided. An average concentration of 0.29 mg/L and a maximum concentration of 3.55 mg/L were seen in the raw water, whereas an average concentration of 0.11 mg/L and a maximum concentration of 2.58 mg/L were found in the treated water (Manitoba Water Stewardship, 2011).

Ammonia was detected (MDL of 0.02–0.05 mg/L) in all but 50 of 393 well water samples analysed (1998–2010) in Saskatchewan, with an average concentration of 1.19 mg/L and a maximum concentration of 8.1 mg/L. Ammonia was not reported in 23% of the 465 treated water samples analysed over the same period, when the calculated average concentration was 1.17 mg/L. A maximum of 6% of the distributed water samples appeared to contain more than 2 mg/L of ammonia. The origin of the relatively high ammonia concentrations is not clearly identified. More research is needed to establish any potential links with various factors (e.g., land use, geology, use of ammonia-based fertilizers in agriculture) (Saskatchewan Ministry of Environment, 2010).

Drinking water samples collected on First Nations reserves across Canada over a 6- to 8-year period were analysed for ammonia. The average values were generally below 1 mg/L, with annual maximum concentrations ranging from 0.24 mg/L in the Atlantic provinces to 2.6 mg/L in Manitoba and 4.4 mg/L in Saskatchewan (Health Canada, 2008a). A national survey conducted in 2009-2010 analyzed 130 samples each of raw and treated water for ammonia. Data reported that the ammonia concentrations were detected in 25% of raw water samples and in 20.8% of treated water samples. An average concentration of 0.4 mg/L total ammonia (maximum of 3.3 mg/L) and an average concentration of 0.55 mg/L total ammonia (maximum of 2.9 mg/L) were found in the raw and treated samples, respectively (Health Canada, 2012).

IPCS (1986) reported that the average human intake of ammonia from a groundwater source would be 0.36 mg/day, based on a daily consumption of 2 litres of water at an average total ammonia concentration of 0.18 mg/L.

## 5.2 Food

Ammonia is found in food at negligible concentrations. Exposure to ammonia from food ingestion is primarily due to the use of various ammonium salts as food stabilizers, leavening agents and flavourings (Environment Canada and Health Canada, 1999; ATSDR, 2004). Some jurisdictions have set restrictions on levels of ammonium salts allowable in processed foods. For example, minor amounts of ammonium compounds (< 0.001–3.2%) can be added to foods as acid regulators, stabilizers, flavouring substances and fermentation aids (IPCS, 1986). The U.S. Food and Drug Administration has set maximum allowable levels of ammonium bicarbonate



(0.04–3.2%) in processed foods for baked goods, grains, snack foods and reconstituted vegetables; 2.0% ammonium carbonate in baked goods, gelatins and puddings; 0.6–0.8% ammonium hydroxide in baked goods, cheeses, gelatins, and puddings; and 0.01% monobasic ammonium phosphate in baked goods (ATSDR, 2004). The estimated exposure from these food additives is 18 mg/day.

### 5.3 Air<sup>1</sup>

Ammonia exists naturally in the air at levels of 1-5 ppb (ATSDR, 2004). In urban areas, air contains up to 20  $\mu\text{g}/\text{m}^3$  of ammonia; in intensive livestock rearing areas, air may contain levels as high as 300  $\mu\text{g}/\text{m}^3$ . Industrial activity may cause local and regional elevations in emissions and atmospheric concentrations of ammonia. For example, Denmead et al. (1982) reported an ammonia concentration as high as 300 ppb over a field during the application of gaseous ammonia fertilizer. Over cattle feedlots, atmospheric ammonia concentrations have been measured between 520 and 2160  $\mu\text{g}/\text{m}^3$  (Hutchinson et al., 1982). If it is assumed that ammonia and ammonium ion concentrations in non-urban air are 2 and 6  $\mu\text{g}/\text{m}^3$  and 24 and 25  $\mu\text{g}/\text{m}^3$  in urban air, respectively, the intake of total ammonia by an individual through inhalation would be 0.1–0.5 mg/day. The common occupational limit of ammonia in air is 25 parts per million (ppm), (IPCS, 1986).

Occupational exposure routes include industries that commonly produce, use or transport ammonia, especially if there are no adequate safety and/or venting systems. Workers in agricultural and farm settings with inadequate ventilation or in intensive livestock rearing facilities with enclosed spaces with high concentrations of animals are populations with potentially high exposures to ammonia (ATSDR, 2004).

### 5.4 Consumer products

Exposure to ammonia can result from the use of various consumer products. Ammonia is commonly used in household and industrial settings. It is often used in cleaning products, detergents and floor waxes. Industrial cleaning solutions usually contain higher levels of ammonia (up to 25%) compared with household cleaners, which typically contain levels of ammonia between 5% and 10% (ATSDR, 2004).

### 5.5 Soil

Soil typically contains about 1–5 ppm of ammonia. The levels of ammonia vary throughout the day, as well as from season to season. Generally, ammonia levels in soils are highest in the summer and spring when bacterial activity is increased. The sources of ammonia in the soil are diverse, including natural or synthetic fertilizers, degradation of livestock excrement, decay of organic material from dead plants and animals and, indirectly, natural fixation of atmospheric nitrogen by free-living nitrogen-fixing bacteria (ATSDR, 2004).

---

<sup>1</sup> Conversion factor in air: 1 part per billion (ppb)  $\approx$  0.696  $\mu\text{g}/\text{m}^3$  at 25°C and 101.3 kPa.

## 6.0 Analytical methods

There are several methods for the analysis of ammonia in drinking water, based on colorimetry, titration and potentiometry procedures. The major factors that influence the selection of an analytical method are the expected ammonia concentration and the presence of interferences such as glycine, urea, cyanates, hydrazine and amines.

In the colorimetric method, the intensity of colour developed in the reaction between ammonia and phenol is proportional to the ammonia concentration in the sample. The titration procedure uses a colour indicator, and the amount of acid used to titrate is proportional to the amount of ammonia present. The ammonia-selective electrode method is probably the easiest to perform. The change in electrical potential at the electrode is proportional to the ammonia concentration. Compared with titrimetric methods, this method applies over a larger range of concentrations and is more sensitive.

Preliminary distillation can be used to prepare samples when they contain interfering ions or when the samples are turbid. In treated drinking water, if the sample is likely to contain residual chlorine, a dechlorinating agent should be used to remove the chlorine before analysis.

### 6.1 U.S. EPA methods and Standard Methods

The U.S. Environmental Protection Agency (EPA) has three methods for the determination of ammonia in drinking water (U.S. EPA, 1983, 1993):

- Method 350.1 is a semi-automated colorimetric method (phenate method), based on the reaction between ammonia and phenol, where the intensity of the reaction product (indophenol blue) is proportional to the ammonia concentration. The method is suitable for ammonia concentrations ranging from 0.01 to 2.0 mg NH<sub>3</sub>-N/L. (U.S. EPA, 1993). The MDL is not stated in the method, but is identified as 0.01 mg/L by the National Environmental Methods Index (NEMI, 2010).
- Method 350.2, which uses either colorimetry (nesslerization) or titrimetry procedures, has MDLs of 0.05 mg/L and 1.0 mg/L, respectively (NEMI, 2010). Thus, titrimetry procedures are preferred for ammonia concentrations greater than 1.0 mg NH<sub>3</sub>-N/L, whereas colorimetry procedures are preferred for concentrations below 1.0 mg NH<sub>3</sub>-N/L (U.S. EPA, 1983).
- Method 350.3 determines ammonia concentration potentiometrically using an ion-selective electrode equipped with a hydrophobic gas-permeable membrane. The method is suitable for ammonia concentrations ranging from 0.03 mg/L to 1400 mg NH<sub>3</sub>-N/L and has an MDL of 0.03 mg/L (U.S. EPA, 1983).

In addition, the following Standard Methods are recognized for the determination of ammonia in drinking water (NEMI, 2010):

- Method 4500-NH<sub>3</sub> C (APHA et al., 2005) is a titrimetric method equivalent to U.S. EPA Method 350.2. The volume of sample aliquot for distillation can be adjusted to match the known or expected range of ammonia concentration. The applicable range for ammonia determination using this method is from 5 to 100 mg NH<sub>3</sub>-N/L.
- Method 4500-NH<sub>3</sub> D (APHA et al., 2005), which uses an ammonia-selective electrode, is equivalent to U.S. EPA Method 350.3 and is applicable to the measurement of 0.03 to 1400 mg NH<sub>3</sub>-N/L. Method 4500-NH<sub>3</sub> E is an alternative ammonia-selective electrode method. This method uses the prior addition of a known concentration of ammonia and is applicable when the relationship between the ammonia concentration and the associated potential measured is linear.

- Method 4500-NH<sub>3</sub> F (APHA et al., 2005) is a phenate method similar to U.S. EPA Method 350.1. The intensity of the blue indophenol dye, a reaction product, is proportional to the concentration of ammonia and is measured at 640 nm using a spectrophotometer. This method has a linear analytical response to 0.6 mg NH<sub>3</sub>-N/L.
- Methods 4500-NH<sub>3</sub> G and 4500-NH<sub>3</sub> H (APHA et al., 2005) are two continuous-flow automated versions of the phenate method, where the blue colour is intensified with sodium nitroprusside and nitroferricyanide, respectively. Method 4500-NH<sub>3</sub> G is applicable in the range of 0.02 to 2.00 mg NH<sub>3</sub>-N/L.

## 6.2 Other available methods

ASTM Method D1426-03 A is equivalent to U.S. EPA Method 350.2. Method D1426-03 B uses an ion-selective electrode procedure as in U.S. EPA Method 350.3 and Standard Methods 4500-NH<sub>3</sub> D and 4500-NH<sub>3</sub> E (ASTM, 2003).

The U.S. Geological Survey reported that Methods I-2522-90 and I-2525-89, which are colorimetric methods, use a rapid flow analyser, coupled with a potentiometric recorder (NEMI, 2010). Ammonia reacts with hypochlorite and salicylate ions in the presence of ferricyanide ions to form the salicylic analogue of indophenol, which is measured at 660 nm. Method I-2525-89 is applicable to low ionic strength water and an ammonia concentration range of 0.002–0.30 mg/L, whereas Method I-2522-90 applies to a concentration range of 0.01–1.5 mg/L. No substances found in natural water appear to interfere with Method I-2525-89; however, sulphide, bromide, nitrite, calcium or magnesium in highly alkaline waters may interfere with ammonia determination using Method I-2522-90.

Direct-reading instruments are being made available commercially. The analysers are designed under electrical conductivity, potentiometry or colorimetry principles, as well as aerosol formation detection systems and infrared photoacoustics.

## 7.0 Treatment technology

### 7.1 Municipal scale

Generally, conventional water treatment processes (coagulation, flocculation and clarification) have only a small effect on reducing the levels of ammonia in drinking water. Some removal may occur if ammonia is sorbed to colloidal particles (Department of National Health and Welfare, 1993; Kurama et al., 2002).

Treatment technologies and strategies to remove ammonia in drinking water include biological treatment (controlled nitrification) and physicochemical processes such as breakpoint chlorination, ion exchange, membrane filtration and air stripping.

Free chlorine and chloramine are two secondary disinfectants used for distributed water. The type of disinfection method used by utilities may influence the treatment technology to remove ammonia from drinking water. Some utilities form chloramine as a strategy to remove naturally occurring ammonia in the raw water supply.

The selection of an appropriate treatment process for a specific water supply will depend on many factors, including the characteristics of the raw water supply, the source and the concentration of ammonia (including variation), the operational conditions of the specific treatment method and the utility's treatment goal.



### 7.1.1 *Biological treatment (controlled nitrification)*

Biological treatment processes are based on the ability of microorganisms (non-pathogenic bacteria) to catalyse the biochemical oxidation or reduction of drinking water contaminants and produce biologically stable water (Rittmann and Snoeyink, 1984). Biological treatment processes have been used in Europe for several years for the removal of ammonia from drinking water (Goodall, 1979; Rittmann and Snoeyink, 1984; Rogalla et al., 1990; Janda and Rudovský, 1994) and have more recently gained acceptance for use in North America (Andersson et al., 2001; Lytle et al., 2007; White et al., 2009; McGovern and Nagy, 2010).

Several authors have reported on full-scale biological treatment to oxidize ammonia in the source water, achieving an oxidation rate greater than 90% (Rittmann and Snoeyink, 1984; Rogalla et al., 1990; Janda and Rudovský, 1994; Andersson et al., 2001; Hossain et al., 2007; Lytle et al., 2007; White et al., 2009). The nitrification process is regarded as the pathway to oxidize ammonia in the biological treatment. As ammonia-oxidizing bacteria (AOB) and nitrite-oxidizing bacteria (NOB) (i.e., nitrifiers) are slow-growing organisms, biologically active filters require a period of colonization before efficient ammonia removal is reached. During this period, ammonia breakthrough and nitrite formation can have adverse impacts on water quality (Lytle et al., 2007; McGovern and Nagy, 2010). Based on pilot study results, Lytle et al. (2007) reported that a colonization to obtain complete nitrification can be achieved in new filters in less than 3 months. This was achieved by constantly running aerated raw water through the filters to promote bacterial regrowth. In order to have complete nitrification a stoichiometric oxygen ( $O_2$ ) demand of 4.33 mg  $O_2$ /mg  $NH_4^+$ -N is required. At ammonia concentrations exceeding this oxygen demand, the biological treatment process requires a constant oxygen feed (Lytle et al., 2007; White et al., 2009).

The process may increase the level of nitrate and may release bacteria into the finished water. The finished water typically requires polishing (e.g., granular activated carbon [GAC] filtration) and post-treatment, such as disinfection, to ensure that neither undesirable organisms nor growth products pass into the distribution system (Wilczak, 2006a).

Critical factors that ensure optimized performance for biological treatment include high dissolved oxygen concentrations, phosphorus, optimal temperature for the selected biomass, a large surface area for accumulating the slow-growing nitrifying biomass, appropriate hydraulic loading rates and maintenance of a long solids retention time (a biomass hold-up in the filter) (Rittmann and Snoeyink, 1984; Bablon et al., 1988; Janda and Rudovský, 1994; Kors et al., 1998; Andersson et al., 2001; Kihn et al., 2002; Hossain et al., 2007; Lytle et al., 2007).

There are different configurations for biological water treatment processes. Most of the systems operate in a fixed biofilm configuration, which includes a biogrowth support medium for the bacterial activity (Rittmann and Snoeyink, 1984; Rogalla et al., 1990; Muramoto et al., 1995; Kors et al., 1998; Andersson et al., 2001; Lytle et al., 2007). Other systems operate in a suspended growth mode, where bacteria are hydraulically maintained in suspension within a reactor such as a fluidized bed filter (Goodall, 1979; Gauntlett, 1981). Gauntlett (1981) reported that fluidized beds had a higher reaction rate per unit volume, shorter residence time, better bacterial control and an absence of blocking or channelling compared with the fixed bed configurations. A pilot-scale study using a fluidized bed achieved an ammonia reduction greater than 95 % of an influent concentration of 3 mg  $NH_3$ -N/L (Gauntlett, 1981).

Lytle et al. (2007) reported achieving an ammonia removal of greater than 95% using biological treatment in a full-scale plant (average 0.6 million gallons per day [MGD] [2270 m<sup>3</sup>/day]). The plant was designed for iron removal, and the filters had been in operation since the 1980s. Three parallel gravity flow sand filters, each operated with a hydraulic loading rate of 2

gallons per minute [gpm] per square foot (4.9 m/h), were capable of reducing an influent ammonia concentration of 1.11 mg NH<sub>3</sub>-N/L in pre-aerated groundwater to below the detection limit of 0.1 mg NH<sub>3</sub>-N /L in the blended post-filtration water. Filtered water was chlorinated and had a free chlorine residual of 0.9 mg Cl<sub>2</sub>/L and a stable pH (Lytle et al., 2007). The authors reported a rise in the nitrate-nitrogen concentration (NO<sub>3</sub>-N) from below 0.04 mg/L to 1.11 mg/L in the filtered water. No nitrite was detected in the filtered water, confirming a complete oxidation of ammonia to nitrate through the filters.

Sand covered with manganese dioxide has been reported to be an effective support for the attachment of nitrifying bacteria. Pilot-scale and full-scale studies reported that sand filters coated with manganese oxides achieved an ammonia oxidation in the range of 95–98% (Janda and Rudovský, 1994; Stembal et al., 2005). Two water treatment plants, each using a single sand filter coated with manganese dioxide, demonstrated a reduction of influent ammonia concentrations of 3.82 and 1.76 mg/L in pre-aerated groundwater to 0.21 and 0.08 mg/L in finished water, respectively, using an air:water ratio of 50. Each filter operated with a hydraulic loading rate up to 5 m/h. The authors observed ammonia breakthrough and nitrite in the finished water (concentrations not specified) when the filters operated at hydraulic loading rates above 5 m/h. The authors suggested that the ammonia removal occurred by nitrification and by sorption on hydrated manganese dioxide (Janda and Rudovský, 1994). Another full-scale study using sand filters coated with manganese dioxide demonstrated that a two-step nitrification process, each step consisting of aeration/filtration, was capable of reducing an average influent ammonia concentration of 4.38 mg/L to 0.13 mg/L in the finished water (Janda and Rudovský, 1994). Muramoto et al. (1995) reported complete oxidation of an average influent ammonia concentration of 0.48 mg/L in a full-scale biological activated carbon filter with an empty bed contact time (EBCT) of 15 minutes.

Andersson et al. (2001) and Kihn et al. (2002) investigated the impact of temperature on controlled nitrification. The studies used open superstructure (i.e., chemical activated) and closed superstructure (i.e., physical activated) GAC filters. The filters had been in service since 1990 for open superstructure GAC and since 1984 for closed superstructure GAC. Each filter operated with hydraulic loading rates in the range of 3.9–5.0 m/h and EBCT between 20 and 30 minutes. Both filters were fed with pre-filtered and ozonated water with influent ammonia concentrations in the range of 0.02 to 0.12 mg NH<sub>4</sub><sup>+</sup>-N/L. The study reported a 98% and a 90% ammonia removal for the open superstructure and for the closed superstructure GAC filters, respectively, at temperatures of 16°C and higher. Both filters achieved up to 30% ammonia oxidation at temperatures below 4°C (Andersson et al., 2001). This lowered oxidation rate is most likely due to the fact that low temperatures decrease the bacterial activity (Bablon et al., 1988; Groeneweg et al., 1994; Andersson et al., 2001; Kihn et al., 2002; Hossain et al., 2007).

A full-scale study compared a single-medium (sand) filter with a dual-media (sand and GAC) filter for the removal of an influent ammonia concentration below 0.2 mg NH<sub>4</sub><sup>+</sup>/L (0.15 mg NH<sub>3</sub>-N/L) at low temperature. The dual-media filter showed no ammonia breakthrough at a temperature of 2°C, whereas the single-medium filter allowed approximately 20% of the influent ammonia to pass through. However, the dual-media filter provided no advantages over the single-layer filter at temperatures greater than 7°C (Bablon et al., 1988).

As nitrite is an intermediate compound in the oxidation of ammonia to nitrate in biological filters, utilities should ensure that their system is optimized such that the biological process is complete and nitrite is not present in the treated water.

### 7.1.2 Breakpoint chlorination

Breakpoint chlorination can eliminate ammonia from water through the formation of a free chlorine residual. Breakpoint chlorination is described as a process in which chlorine demand is satisfied, combined chlorine compounds are destroyed, ammonia is oxidized to form nitrogen gas and free chlorine residual is achieved when additional chlorine is added. The process requires frequent monitoring of ammonia concentrations and the various forms of chlorine (combined, total chlorine and free chlorine residual) to ensure that breakpoint chlorination is achieved at all times. It is necessary to generate a breakpoint curve for every plant and to monitor the fluctuation of ammonia to ensure that breakpoint chlorination is always achieved.

Utilities use breakpoint chlorination to remove excess ammonia in the source water and to control nitrification episodes in the distribution system. In distribution systems, breakpoint chlorination can be an effective method to control ammonia-oxidizing bacterial activity in the short term, but it may not prevent the establishment of nitrifying biofilm on return to chloramination (Kirmeyer et al., 1995; Odell et al., 1996; Zhang and DiGiano, 2002; Pintar and Slawson, 2003).

Breakpoint chlorination requires chlorine doses approximately 8–10 times higher (on a weight basis) than the ammonia concentration to achieve a free chlorine residual. The process is a series of reactions in which monochloramine is formed first. The reaction rate of monochloramine formation depends on pH, temperature and the chlorine-to-ammonia-nitrogen ( $\text{Cl}_2:\text{NH}_3\text{-N}$ ) weight ratio, preferably in the range of 3:1 to 5:1. Once monochloramine is formed and  $\text{Cl}_2:\text{NH}_3\text{-N}$  is greater than 5:1, breakpoint chlorination proceeds through two main groups of reactions: 1) disproportionation (acid-catalysed reactions) of monochloramine to form dichloramine and 2) decomposition of dichloramine. Both groups of reactions require an excess of free chlorine (Kirmeyer et al., 2004). Dichloramine undergoes a series of decomposition and oxidation reactions to form nitrogen-containing products, including nitrogen, nitrate, nitrous oxide gas and nitric oxide (AWWA, 2006). Trichloramine, or nitrogen trichloride, is an intermediate during the complete decomposition of chloramines. Its formation depends on pH and the  $\text{Cl}_2:\text{NH}_3\text{-N}$  weight ratio and may appear after the breakpoint (Kirmeyer et al., 2004; Hill and Arweiler, 2006; Randtke, 2010). At  $\text{Cl}_2:\text{NH}_3\text{-N}$  of 7.6:1, the free ammonia is oxidized to nitrogen and chlorine is reduced to chloride. An increase of the  $\text{Cl}_2:\text{NH}_3\text{-N}$  weight ratio greater than 7.6:1, free chlorine is the predominant chlorine residual.

The reaction rate of breakpoint chlorination is determined by the formation and decay rates of dichloramine, reactions that are highly dependent on pH. Ideally, the reaction takes place at a pH in the range of 7.0–8.0 (Kirmeyer et al., 2004). The theoretical  $\text{Cl}_2:\text{NH}_3\text{-N}$  weight ratio for breakpoint chlorination is 7.6:1; the actual  $\text{Cl}_2:\text{NH}_3\text{-N}$  ratio varies from 8:1 to 10:1, depending on pH, temperature and the presence of reducing agents. The presence of iron, manganese, sulphide and organic chlorine demand compounds will compete with the free chlorine added, potentially limiting the chlorine available to react with ammonia (Kirmeyer et al., 2004; AWWA, 2006; Muylwyk, 2009). A contact time of 30 minutes or longer is necessary for the reaction to go to completion (Kirmeyer et al., 2004; Hill and Arweiler, 2006). The breakpoint ratio should be determined experimentally for each water supply (Hill and Arweiler, 2006).

Chlorine compounds certified to NSF International (NSF)/American National Standards Institute (ANSI) Standard 60 should respect the maximum use limit (MUL) stated in the standard. This ensures that any potential trace contaminants do not exceed their respective health-based limits even if used at the maximum dose stated for the additive. The responsible

authority may choose to allow a utility to exceed the MUL in order to achieve breakpoint chlorination and disinfection goals. As exceeding the MUL could invalidate the certification, consultation with the body that has certified the chlorine compound is recommended. This will help ascertain what potential trace contaminants might be present and thus help determine what additional monitoring of hypochlorite-related contaminants might be triggered when the MUL is exceeded. As breakpoint chlorination requires relatively high concentrations of chlorine, this can cause other problems, such as the formation (or increased concentrations) of disinfection by-products in the presence of organic matter. However, efforts to limit the formation of disinfection by-products must not compromise the effectiveness of disinfection. An advanced treatment, such as GAC adsorption, may be considered following breakpoint chlorination to remove resulting taste and odour compounds as well as chlorination by-products (Janda and Rudovský, 1994; Wilczak, 2006a).

Breakpoint chlorination, relative to nitrification control in the distribution system, is not considered an effective long-term strategy. Utilities should consider more permanent control strategies, such as changes in operation or engineering improvement (Kirmeyer et al., 1995; Hill and Arweiler, 2006).

### 7.1.3 Ion exchange

Ion exchange is a physicochemical process that employs an exchange of ions (cations or anions) in the water to be treated with ions sorbed at the solid phase of the natural or synthetic resins. Cation exchange is capable of removing ammonia from drinking water.

Studies have investigated natural zeolites, such as clinoptilolite, bentonite, sepiolite and mordenite (Hodi et al., 1995; Demir et al., 2002; Park et al., 2002; Weatherley and Miladinovic, 2004; Wang et al., 2007), and synthetic resins (Lin and Wu, 1996; Abd El-Hady et al., 2001) for the removal of ammonium ions from water. Factors such as pH, pretreatment of the natural zeolites, media particle size, influent ammonium concentration and competing cations, such as calcium, magnesium and potassium, in the water affect the efficiency of ammonium removal. Ion exchange processes do not result in a constant percentage of removal of contaminants (e.g., ammonium ion) with time, because they will break through as the resin reaches its capacity. Once the resin's capacity is reached, contaminant concentrations will increase in the finished water, and the resin must be regenerated. Ion exchange technology may be inconvenient for a treatment plant with a capacity above 80 000 m<sup>3</sup>/day because of the large footprint required for the ion exchange columns (Kurama et al., 2002).

Clinoptilolite is the most abundant natural zeolite and has been shown to have a high selectivity for ammonium ion. Although it has been applied primarily in wastewater treatment, this technique has recently been studied for the reduction of ammonium concentrations in drinking water. Studies reported that the pretreatment of natural clinoptilolite increased both the ion exchange capacity of the clinoptilolite and the ammonium removal efficiency in aqueous solutions (Haralambous et al., 1992; Turan and Celik, 2003; Vassileva and Voikova, 2009; Siljeg et al., 2010).

Laboratory-scale and pilot-scale cation exchange experiments have been shown to reduce ammonia concentrations in drinking water. This technology seems to be effective when natural zeolites are used as the cation exchange material and the water has a low hardness (Haralambous et al., 1992; Weatherley and Miladinovic, 2004).

A pilot-scale study (Gaspard et al., 1983) evaluated the capability of clinoptilolite to remove ammonium ions in tap water. An average influent concentration of 2.25 mg NH<sub>4</sub><sup>+</sup>/L (1.75 mg NH<sub>4</sub><sup>+</sup>-N/L) was reduced to a predefined breakthrough level of 0.5 mg NH<sub>4</sub><sup>+</sup>/L (0.39 mg

NH<sub>4</sub><sup>+</sup>-N/L), achieving an ion exchange capacity of 0.108 milliequivalents of ammonium ion per gram of clinoptilolite (1.47 mg NH<sub>4</sub><sup>+</sup>-N/g) and 750 bed volumes (BV).

A laboratory column study using sodium clinoptilolite (Na<sup>+</sup>-clinoptilolite) achieved an exchange capacity of 0.47 mg NH<sub>4</sub><sup>+</sup>/g clinoptilolite (0.37 mg NH<sub>4</sub><sup>+</sup>-N/g) and 600 BV at pH of 8.26. An average influent concentration of 0.86 mg NH<sub>4</sub><sup>+</sup>/L (0.67 mg NH<sub>4</sub><sup>+</sup>-N/L) in groundwater was reduced to 0.15 mg NH<sub>4</sub><sup>+</sup>/L (0.12 mg NH<sub>4</sub><sup>+</sup>-N/L) (Hodi et al., 1995).

In another laboratory study, Weatherley and Miladinovic (2004) evaluated the performance of Na<sup>+</sup>-clinoptilolite and Na<sup>+</sup>-mordenite for ammonium removal from aqueous solution. The experiments were conducted with feed concentrations from 1.0 mg/L NH<sub>4</sub><sup>+</sup>/L (0.78 mg NH<sub>4</sub><sup>+</sup>-N/L) to 200.0 mg NH<sub>4</sub><sup>+</sup>/L (155.6 mg NH<sub>4</sub><sup>+</sup>-N/L) while maintaining the pH below 7.5. Equilibrium data demonstrated that Na<sup>+</sup>-clinoptilolite achieved a 98.8% reduction of an influent concentration of 10 mg NH<sub>4</sub><sup>+</sup>/L (7.8 mg NH<sub>4</sub><sup>+</sup>-N/L), in the absence of other ions in solution. However, in the presence of 40 mg/L each of calcium, magnesium and potassium, the resin achieved reduction of 93.7%, 94.7% and 95.9% of ammonia, respectively. Similarly, equilibrium data for Na<sup>+</sup>-mordenite showed that a reduction of 92.3% of an influent concentration of 10 mg NH<sub>4</sub><sup>+</sup>/L (0.78 mg NH<sub>4</sub><sup>+</sup>-N/L) was achieved in the absence of other ions in solution. However, in the presence of 40 mg/L each of calcium, magnesium and potassium, Na<sup>+</sup>-mordenite achieved 91.8%, 92.2% and 86.3% ammonia reductions, respectively. The presence of calcium, magnesium and potassium thus decreased the ammonium removal efficiency for both zeolites (Weatherley and Miladinovic, 2004).

Laboratory column tests (Turan and Celik, 2003) studied the impact of ammonia (form not specified) concentration on the ion exchange capacity of clinoptilolite and the effectiveness of clinoptilolite regeneration on column performance. The results showed that an increase in influent ammonia concentrations decreased the ammonia reductions. Initial concentrations of 10, 15 and 20 mg/L were reduced by 96%, 94% and 87%, respectively, after 12 hours of operation. The study reported that natural clinoptilolite achieved a 65.0% reduction of an initial ammonia concentration of 10 mg/L after 23 hours of operation, whereas twice-regenerated clinoptilolite achieved a 98.0% reduction under the same operating conditions.

Abd El-Hady et al. (2001) evaluated a synthetic strong acid cationic resin for removing ammonium ions in laboratory experiments. Three initial ammonium concentrations of 10 mg NH<sub>4</sub><sup>+</sup>/L (7.8 mg NH<sub>4</sub><sup>+</sup>-N/L), 5 mg NH<sub>4</sub><sup>+</sup>/L (3.9 mg NH<sub>4</sub><sup>+</sup>-N/L) and 2 mg NH<sub>4</sub><sup>+</sup>/L (1.6 mg NH<sub>4</sub><sup>+</sup>-N/L) were reduced to below a predefined breakthrough concentration of 0.5 mg/L. Adsorption capacities of 0.156 mol/L (2.2 mg NH<sub>4</sub><sup>+</sup>-N/ml resin), 0.085 mol/L (1.2 mg NH<sub>4</sub><sup>+</sup>-N/ml resin) and 0.0317 mol/L (0.4 mg NH<sub>4</sub><sup>+</sup>-N/ml resin) and BVs of 295, 340 and 380 were reported for the above three initial concentrations, respectively.

The major considerations when using ion exchange treatment include chromatographic peaking, disposal of the resin regenerant (Clifford, 1999) and the possible increased corrosivity of the treated water (Schock and Lytle, 2010). Regeneration results in a brine waste stream that contains high ammonium concentrations and must be disposed of appropriately, thus increasing the cost of this process. The exchange of ions can cause mineral imbalances that could increase the corrosive nature of the treated water (Schock and Lytle, 2010). In some cases, post-treatment corrosion control measures may need to be taken, to ensure that corrosion problems do not occur following treatment.

#### 7.1.4 Membrane filtration

The available scientific information on the removal of ammonia from water supplies by membrane technologies is limited. These processes are based on forcing water across a



membrane under pressure while the ionic species, such as ammonium, are retained in the waste stream. Reverse osmosis (RO) treatment systems typically require pre-filtration for particle removal and often include other pretreatment steps, such as the addition of anti-scaling agents, prechlorination/dechlorination and softening. Post-treatment steps typically include pH adjustment, corrosion inhibitor addition and disinfection (Cevaal et al., 1995).

RO and, to a lesser extent, nanofiltration (NF), can be effective technologies for reducing ammonia concentrations in drinking water (Koyuncu et al., 2001; Koyuncu, 2002; Kurama et al. 2002; Quail, 2008).

Koyuncu (2002) conducted a pilot-scale study to evaluate the effectiveness of nanofiltration and low-pressure reverse osmosis (LPRO) membranes for ammonia removal under different operating parameters. A spiral wound module was operated at feed influent ammonia concentrations in the range of 10–15 mg/L. The LPRO membrane showed a higher performance than the nanofiltration membrane under the tested conditions. The rejection of ammonia was increased with an increase in the pressure for both membranes. The LPRO membrane was capable of rejecting from 90% to 95% of ammonia concentrations using a feed pressure in the range of 3 to 6 bar (43.5–87.0 pounds per square inch [psi]) and temperature in the range of 15–25°C. The nanofiltration membrane achieved up to 90% rejection at the same tested conditions. At temperatures above 25°C, the LPRO membrane showed a decrease of the rejection rate, whereas the rejection rate of the nanofiltration membrane was slightly affected (Koyuncu, 2002). Both membranes had a negative charge at neutral and high pH and neutral or slightly positive charge at low pH. The study reported that a neutral pH was optimal for ammonia rejection by both membranes.

An earlier pilot-scale study by Koyuncu et al. (2001) evaluated the efficiency of brackish water reverse osmosis (BWRO) and saline water reverse osmosis (SWRO) membranes for the removal of ferroammonium sulphate ( $\text{Fe} \cdot (\text{NH}_4\text{OH}) \cdot (\text{SO}_4)_2 \cdot \text{H}_2\text{O}$ ) and ammonium hydroxide ( $\text{NH}_4\text{OH}$ ) in aqueous solution. Both membranes demonstrated a higher rejection rate for the ammonium complex than for ammonium hydroxide. The SWRO membrane was capable of rejecting 99% and 90% of the ammonium complex from influent concentrations of 8.5 mg/L and 100 mg/L, respectively. The BWRO membrane achieved a rejection rate of 96% and 83% of the ammonium complex from influent concentrations of 10 mg/L and 30 mg/L, respectively. The SWRO membrane was capable of rejecting 30–40% of ammonium hydroxide from influent concentrations of 2–90 mg/L. The BWRO membrane achieved rejection rates in the range of 10–20% of ammonium hydroxide from influent concentrations ranging from 1 to 186 mg/L. The study also reported rejection rates of 95% and 60% of ammonia in surface water by BWRO and SWRO membranes, respectively. The feed water had influent concentrations in the range of 3–4 mg/L and neutral pH. No fouling and no reduction in flux were observed during the 5-hour experimental runs (Koyuncu et al., 2001).

Laboratory testing of RO membranes found that two membranes (Desal-3LP and Desal-3b SE, Osmonics) were capable of effectively rejecting  $\text{NH}_4^+$ . An average rejection of 95% was achieved from 6.5 mg  $\text{NH}_4^+$ /L (5.05 mg  $\text{NH}_4^+$ -N/L) as a feed concentration. The study has found that tested nanofiltration membranes achieved up to 26% removal (Kurama et al. 2002).

Considerations when using RO treatment include disposal of the reject water and the possible increased corrosivity of the treated water (Schock and Lytle, 2011). RO rejects a significant portion of the influent water as contaminant-rich brine (Taylor and Wiesner, 1999). The concentrate discharge must be considered and disposed of appropriately. The removal of contaminants can cause mineral imbalances that could increase the corrosive nature of the treated

water (Schock and Lytle, 2011). In some cases, post-treatment corrosion control measures may need to be taken.

#### 7.1.5 *Combination of reverse osmosis and biological treatment*

Nagy and Granlund (2008) Quail (2008) and McGovern and Nagy (2010) presented a combined process of an RO (75% water treated) system and biological treatment (25% water treated) to remove inorganic contaminants found in groundwater simultaneously and to address copper corrosion control. The maximum design capacity of the water treatment plant was 6.5 MGD (24 605 m<sup>3</sup>/day). A spiral wound polyamide thin film composite RO membrane was capable of reducing of an ammonia concentration of 2.0 mg NH<sub>4</sub><sup>+</sup>-N/L to 0.08 mg NH<sub>4</sub><sup>+</sup>-N/L, achieving greater than 96% reduction at a system recovery of 82% and a feed pressure of 130 psi. Pretreatment included pH adjustment and addition of anti-scalant. While maintaining a dissolved oxygen concentration of 3 mg/L, the ammonia biological filters were seeded with backwash water from the existing wastewater plant. The nitrification process, established within 2 months, was capable of reducing an average influent ammonia concentration of 2.0 mg NH<sub>4</sub><sup>+</sup>-N/L to an average effluent concentration of less than 0.16 mg NH<sub>4</sub><sup>+</sup>-N/L, achieving reduction greater than 92% (McGovern and Nagy, 2010). The filters operated at a hydraulic loading rate of 4 gpm/ft<sup>2</sup> (9.8 m/h) and had a media depth of 1.5 m. Blended water had an ammonia concentration of less than 0.16 mg NH<sub>4</sub><sup>+</sup>-N/L.

#### 7.1.6 *Air stripping*

Although air stripping is a common practice for removing ammonia from wastewater, its treatment efficiency for drinking water is expected to be marginal due to the low Henry's Law constant (0.0006 at 20°C) in combination with relatively low concentrations of ammonia encountered in source water (Crittenden et al., 2005).

Separation of non-ionized ammonia from water can be achieved with air stripping in a packed tower by raising the pH of the water above 10 and increasing the temperature. As ammonia is soluble in water, a high air to water ratio is required; pH adjustment after the aeration is required for subsequent processes (U.S. EPA, 2000).

#### 7.1.7 *Emerging technologies*

Several drinking water treatment technologies for ammonia are being developed but are still primarily in the experimental stage or do not have peer-reviewed information on the effectiveness of pilot-scale or large-scale application. Some of the emerging technologies include the following:

- *Trickling filters:* A pilot-scale study evaluated trickling filters for simultaneous biological removal of ammonia, iron and manganese from potable water. Influent ammonia concentrations in the range of 0.5–3.0 mg/L were reduced up to 82% in the finished water under a variety of operating conditions (Tekerekopoulou and Vayenas, 2007, 2008).
- *Electrochemical removal:* A pilot-scale charge barrier capacitive deionization process is reported as effective in removing total dissolved solids, nitrate and ammonia from water. The process employs an adsorption of ions on the surface of two oppositely charged electrodes. The process achieved ammonia removal up to 88.1% at 1000 mg/L as feed concentration (Broseus et al., 2009).
- *Submerged membrane bioreactors:* Although the use of membrane bioreactors has been applied primarily in wastewater treatment, this technique has recently been considered as a new technology in drinking water treatment. Laboratory studies examined the

effectiveness of hollow fibre membrane modules directly immersed inside the activated sludge reactors for ammonia removal. Removal efficiencies in the range of 89–98% were achieved by the submerged membrane bioreactors through biological nitrification. The influent  $\text{NH}_3\text{-N}$  concentrations have been reported in the range of 2.00–4.24 mg/L (Li and Chu, 2003; Tian et al., 2009).

#### 7.1.8 *Nitrification in the distribution system*

One of the main concerns related to the presence of ammonia in drinking water is the potential for the formation of nitrite and nitrate, parameters with health risks and drinking water quality guidelines. Nitrite and nitrate are the products of nitrification, a two-step process that oxidizes ammonia either in natural water or in water that has been disinfected by chloramine. The occurrence of nitrification in chloraminated distribution systems has been well documented (Skadsen, 1993; Odell et al., 1996; Wilczak et al. 1996). According to Kirmeyer et al. (1995) and Wilczak et al. (1996), nitrification may occur at 63% of utilities that use chloramine as a secondary disinfectant. In a survey of 56 utilities using monochloramine, 48% of them reported that they had experienced nitrification (Kirmeyer et al., 2004).

Nitrification can occur irrespective of pipe material—plastic, polyvinyl chloride, asbestos-cement, ductile iron and cast iron. Certain pipe materials, such as unlined cast iron pipes or old mortar-lined iron pipes, may provide more favourable conditions for nitrification to occur (Cohen et al., 2001). Accumulated sediment and biofilm can protect the ammonia-oxidizing bacteria from chloramine residual. Higher concentrations of ammonia-oxidizing bacteria were detected in reservoir and pipe sediment materials than in pipe biofilm samples (Wolfe et al., 1990).

Nitrification in the distribution systems can have adverse impacts on water quality. These impacts include increased nitrite and nitrate levels, reduced chloramine residuals, increased bacterial regrowth (i.e., increased heterotrophic plate count [HPC], with a possible detection of *Escherichia coli*), as well as a reduction of pH and dissolved oxygen (Kirmeyer et al., 1995, 2004; Odell et al., 1996; Wilczak et al., 1996; Bremer et al., 2001; U.S. EPA, 2002; Lytle et al., 2007; Muylwyk, 2009; Zhang et al., 2009). Studies have also reported a link between corrosion problems and nitrification (Edwards and Dudi, 2004; Douglas et al., 2004; Zhang et al., 2008, 2010).

The potential increase of nitrite in the distribution system due to nitrification is significant, as it may exceed 1 mg/L  $\text{NO}_2\text{-N}$ . However, when nitrite concentrations increase as a result of nitrification, the primary concern for utilities is that nitrite consumes chlorine and decomposes chloramines, which results in an increase in microbial counts, including an increase in the potential presence of coliform bacteria in the distribution system (Baribeau, 2006; Smith, 2006). Harrington et al. (2002) and the U.S. EPA (2002) noted that increases in nitrite up to 1 mg  $\text{NO}_2\text{-N/L}$  due to nitrification could theoretically occur in any system in which the total ammonia concentration entering the distribution system is greater than 1 mg-N/L.

Factors contributing to nitrification in the distribution system include warm water temperatures, pH, a low  $\text{Cl}_2\text{:NH}_3\text{-N}$  ratio and the concurrent increase of free ammonia concentrations and chloramine residual. A number of distribution system parameters, such as detention time, reservoir design and operation, sediment and tuberculation in piping, biofilm and the absence of sunlight, can affect the nitrification (Skadsen, 1993; Kirmeyer et al., 1995, 2004; U.S. EPA, 1999; Lytle et al., 2007; Fleming et al., 2008; Baribeau, 2010).

The optimum temperature for nitrifiers to grow ranges between 20°C and 30°C (Baribeau, 2006); however, regrowth and nitrification can occur at temperatures as low as 5°C or



even less in systems with long detention times (Pintar et al., 2000). Kors et al. (1998) discussed a case of nitrification under extreme cold-water conditions (below 4°C). The increase in temperature will increase the chloramine decomposition rate, which will promote nitrification, as more free ammonia will be released (Baribeau, 2006).

Although the optimum pH range for nitrifiers to grow is 7.5–8.0, nitrification can occur at pH 6.6–9.8 (Kirmeyer et al., 1995; Odell et al., 1996; Wilczak et al., 1996; Baribeau, 2006; Wilczak, 2006b). The pH may decrease during nitrification in low-alkalinity water. If the pH decreases below 8.0, chloramine decomposition may be accelerated. The pH data should be evaluated carefully, because pH may vary throughout the system depending on factors other than nitrification such as corrosion. Theoretical oxygen concentration (O<sub>2</sub>) required for biological oxidation of 1 g NH<sub>4</sub><sup>+</sup>-N to NO<sub>2</sub><sup>-</sup>-N is 3.22 g O<sub>2</sub>, and 1.11 g O<sub>2</sub> to oxidize 1 g NO<sub>2</sub><sup>-</sup>-N to NO<sub>3</sub><sup>-</sup>-N. Thus the total theoretical O<sub>2</sub> requirement is 4.33 g O<sub>2</sub> to oxidize 1 g NH<sub>4</sub><sup>+</sup>-N to NO<sub>3</sub><sup>-</sup>-N (Baribeau, 2006).

The initial Cl<sub>2</sub>:NH<sub>3</sub>-N weight ratio used to form monochloramine (the preferred chloramine species) affects the level of the free ammonia available in the distribution system (Fleming et al., 2005, 2008). Free ammonia may enter the distribution system from the treatment plant due to the overdosing of ammonia or incomplete reaction with free chlorine. The measurement of free chlorine immediately upstream of the point of ammonia addition is critical to the proper dosing of ammonia at the treatment plant. Minimizing free ammonia entering the distribution system is extremely important (Cohen and Friedman, 2006; Wilczak, 2006b). A weight ratio of Cl<sub>2</sub>:NH<sub>3</sub>-N should generally be maintained between 4.5:1 and 5:1 in the plant effluent to enhance the formation of monochloramine and reduce the concentration of free ammonia entering into the distribution system (Harrington, 2003; Kirmeyer, 2004; Skadsen and Cohen, 2006). However, the water quality parameters and utility-specific chlorine demand must be considered when selecting the target ratio (Skadsen and Cohen, 2006). Kirmeyer et al. (2004) and Skadsen and Cohen (2006) suggested that minimizing free ammonia entering the distribution system to a concentration below 0.1 mg NH<sub>3</sub>-N/L and preferably below 0.05 mg NH<sub>3</sub>-N/L is an important optimization goal to reduce the potential for nitrification.

When free chlorine is the desired residual disinfectant in the distribution system, the removal of naturally occurring ammonia in the source water is beneficial to reduce chlorine demand and avoid chloramine formation. It is important to be aware that monochloramine may interfere with the N,N-diethyl-p-phenylendiamine (DPD) method used to monitor free chlorine and can create a false positive reading (Smith, 2006; Pon, 2008). For utilities practicing chloramination, it is important to take into consideration the ammonia concentration in the source water when establishing the ammonia dosage for chloramine formation (Skadsen and Cohen, 2006; Muylwyk, 2009; Shorney-Darby and Harms, 2010). Wolfe et al. (1990) reported that using Cl<sub>2</sub>:NH<sub>3</sub>-N ratio of 3:1 results in approximately 0.2 mg/L free ammonia when maintaining a total chlorine concentration of 1.5 mg/L in the distribution system. Bower and Crowe (1988) demonstrated that an ammonia-nitrogen concentration of 0.25 mg/L would promote the growth rate of nitrifying organisms in both the treatment plant and the distribution system. An optimization of Cl<sub>2</sub>:NH<sub>3</sub>-N ratio should ensure that Health Canada's guideline for chloramines is not exceeded (Health Canada, 1995).

Although chloramine is more stable than free chlorine, it decomposes and releases free ammonia. An understanding of chloramine chemistry is critical in order to maintain chloramine residual, prevent the release of free ammonia in the distribution system and prevent or control nitrification. The rate of chloramine residual loss in the distribution system is affected by reactions with natural organic matter (NOM) and inorganic constituents (chloramine demand)

and a combination of hydrolysis and acid-catalysed disproportionation reactions (chloramine decay). Chloramine demand and decay in the distribution system release free ammonia, which, along with the ammonia entering the system, provides substrate for ammonia-oxidizing bacterial growth and promotes nitrification (Skadsen, 1993; Vikesland et al., 2001, 2006; Kirmeyer et al., 2004; Chowdhury et al., 2006; Wilczak, 2006b). Chlorine/chloramine demand should be satisfied as much as possible within the treatment plant, and chloramine decay should be minimized in the distribution system, as these reactions increase the free ammonia concentration in the distribution system and trigger nitrification (Baribeau, 2006; Wilczak, 2006b). It is important to note that even the stringent control of excess free ammonia and the maintenance of a proper  $\text{Cl}_2:\text{NH}_3\text{-N}$  ratio may not always be effective in preventing nitrification. This is due to the fact that chloramine in the distribution system will start to decay based on water quality conditions and water age, releasing free ammonia into the water (Cohen and Friedman, 2006).

The presence of bromide in chloraminated water complicates system chemistry by reacting with chlorine and chloramine species to form bromamines. The bromamines are capable of accelerating chloramine decay and may also be able to combine with organic contaminants to form halogenated organics, which remain poorly understood to date (Vikesland et al., 2001; Kirmeyer et al., 2004).

*N*-Nitrosodimethylamine (NDMA) is a nitrogen-containing disinfection by-product that may be formed during the treatment of drinking water, particularly during chloramination and, to a lesser extent, chlorination (Richardson, 2005; Charrois and Hrudehy, 2007; Nawrocki and Andrzejewski, 2011). The key to controlling the formation of NDMA lies in limiting its precursors, including dichloramine. As such, optimization and control of free ammonia are important elements in preventing NDMA formation. Additional information on NDMA is available in the Guideline Technical Document on NDMA (Health Canada, 2011).

A research study (Kirmeyer et al., 1995) based on literature reports, case studies, an analytical survey and a phone survey of large chloraminated systems obtained conflicting results regarding the water quality and the treatment factors that affect nitrification episodes. In combination with the distribution system hydraulics, the importance of one factor over another factor causing nitrification was specific to each system. In general, free ammonia promotes nitrification in the distribution system and is available either through ammonia feed overdose or through release of free ammonia from chloramine demand and decay (Kirmeyer et al., 1995).

The treatment plant, the distribution systems and storage facilities all require monitoring for specific parameters. Parameters that can be monitored for potential causes of nitrification include chloramine residual,  $\text{Cl}_2:\text{NH}_3\text{-N}$  ratio, free ammonia concentration entering the distribution system, pH and temperature. Products of nitrification that can be monitored include nitrite/nitrate and HPC at the entry point of the distribution system and throughout the system (Odell et al., 1996; Wilczak et al., 1996).

The concentration of free ammonia entering the distribution system and at key locations in the system, such as storage facilities and areas with long water detention times (e.g., dead ends), in addition to parameters such as total chlorine residual and nitrite, is a very useful parameter to monitor for nitrification control. In particular, Smith (2006) suggested that a free ammonia concentration greater than 0.1 mg  $\text{NH}_4^+\text{-N/L}$  at storage facilities can be used as an indicator of nitrification requiring further investigation (i.e., alert level).

A site-specific evaluation is necessary to establish a nitrification monitoring program. The program should identify system-specific alert and action levels, which can be used to determine the appropriate level of nitrification response. The monitoring frequency of the parameters depends on the location and the purpose of the data. Distribution system nitrification

parameters considered to be of higher priority are total chlorine residual, nitrite and nitrate. Changes in the trend of these nitrification parameters should trigger more frequent monitoring of other parameters, such as free ammonia.

There are several preventive and corrective measures that can be taken to address nitrification (AWWA, 2006). Preventive measures include:

- Control of water quality parameters (pH, free ammonia entering the distribution system, organic matter) and operating parameters ( $\text{Cl}_2:\text{NH}_3\text{-N}$  weight ratio and chloramine residual):
  - Establishing the proper pH level is essential for maintaining chloramine residual in the distribution system and limiting nitrification (Wilczak, 2006b).
  - A minimization of free ammonia entering the distribution system to concentrations below 0.1 mg  $\text{NH}_3\text{-N}$  /L and preferably below 0.05 mg  $\text{NH}_3\text{-N}$ /L is an important optimization goal to reduce the potential for nitrification (Kirmeyer et al., 2004).
  - In general, chloramine residuals, greater than 2.0 mg/L (leaving the treatment plant) appear to be effective in preventing nitrification by limiting the growth of ammonia-oxidizing bacteria (Kirmeyer et al., 1995; Odell et al., 1996; U.S. EPA, 1999; Harrington et al., 2003). The chloramine residual concentration leaving the treatment plant will vary depending on the size of the distribution system and the water quality characteristics (U.S. EPA, 1999; Skadsen and Cohen, 2006). However, once nitrification is under way, the high chloramine residual (up to 8 mg/L) may not control nitrification (Skadsen, 1993). Increasing the chloramine concentration during a nitrification event may exacerbate the process, because it leads to an increase in the level of free ammonia as a result of chloramine decay (Woolschlager et al., 2001; Harrington et al., 2003; Hill and Arweiler, 2006).
- *Corrosion control programs*: These may help minimize pipe biofilms and sediment, limit attachment of microorganisms, reduce the reaction between chloramine and corrosion products and thus reduce chloramine demand (Wilczak, 2006b).
- *Distribution system pipe flushing*: Sediment flushing in the pipe network, reservoir turnover and cleaning will prevent or delay the onset of nitrification (Hill and Arweiler, 2006; Wilczak, 2006b). However, once nitrification occurs, flushing alone may be limited in effectiveness (Skadsen and Cohen, 2006).
- *Booster chlorination or chloramination stations*: Attention is given to recombining the released (increased) free ammonia in the distribution system by booster chlorination to maintain the ratio near 5:1 throughout the system (Wilczak, 2006b). Free ammonia residual needs to be measured before chemical addition. If sufficient free ammonia is still present, only chlorine needs to be added.
- *Temporary/seasonal free chlorination (breakpoint chlorination)*: Periodic switching to free chlorine is a preventive and/or effective control method practised by water utilities. However, a temporary switch to free chlorination in the distribution system has been associated with numerous problems, including a temporary increase in HPC, coliform-positive samples (related to the sloughing of existing biofilm layers) (Odell et al., 1996), potential taste and odour problems, and potential disinfection by-product problems (Skadsen, 1993; Hill and Arweiler, 2006). Studies by Kirmeyer et al. (1995) and Odell et al. (1996) suggested that a return to chloramination following a free chlorination period led to subsequent nitrification within a short period.

- *Chlorite addition*: It appears that chlorite is effective for nitrification prevention (McGuire et al., 1999; Baribeau, 2006; Wilczak, 2006b). The latest research demonstrates that chlorite addition is less effective in areas where nitrification has been substantially developed before the chlorite application. Chlorite application prior to nitrification development is a strategy for nitrification prevention for utilities with significant seasonal changes in their finished water temperature (McGuire et al., 2009; Zhu et al., 2010). However, chlorite addition is considered to be controversial, as chlorite is a regulated contaminant, and its presence can also lead to the formation of chlorate (Skadsen and Cohen, 2006). Utilities wishing to use chlorite addition as a control strategy should ensure that the *Guidelines for Canadian Drinking Water Quality* for chlorite and chlorate (Health Canada, 2008b) are not exceeded.

Corrective measures are similar to the preventive measures and include:

- distribution system pipe flushing;
- temporary/seasonal free chlorination (breakpoint chlorination);
- reservoir cycling to limit water age. During severe nitrification episodes, reservoir cleaning, as well as drainage and disinfection, may be needed; and
- chlorite addition.

The different measures used to control the nitrification episodes vary in their effectiveness and their ability to provide long-term improvements in nitrification problems. For these reasons, comprehensive strategies aimed at the prevention of nitrification episodes are recommended over strategies aimed at controlling nitrification as it occurs. Any strategy should also ensure that the relevant *Guidelines for Canadian Drinking Water Quality* (e.g., chloramines) are not exceeded. Detailed information on nitrification control and prevention measures is available in reports and reviews by Kirmeyer et al. (1995), AWWA (2006) and Zhang et al. (2009).

## 7.2 Residential scale

Generally, it is not recommended that drinking water treatment devices be used to provide additional treatment to municipally treated water. In cases where an individual household obtains its drinking water from a private well, a private residential drinking water treatment device may be an option for reducing ammonia concentrations in drinking water. Although no certified residential treatment devices are currently available for the reduction of ammonia levels in drinking water, treatment devices using reverse osmosis or ion exchange may be effective for the reduction of ammonia concentrations in drinking water.

Before a treatment device is installed, the water should be tested to determine general water chemistry and verify the presence and concentration of ammonia. Periodic testing by an accredited laboratory should be conducted on both the water entering the treatment device and the finished water to verify that the treatment device is effective. Devices can lose removal capacity through use and time and need to be maintained and/or replaced. Consumers should verify the expected longevity of the components in their treatment device as per the manufacturer's recommendations.

Health Canada does not recommend specific brands of drinking water treatment devices, but strongly recommends that consumers use devices that have been certified by an accredited certification body as meeting the appropriate NSF/ANSI drinking water treatment unit standards. These standards have been designed to safeguard drinking water by helping to ensure the

material safety and performance of products that come into contact with drinking water. Certification organizations provide assurance that a product conforms to applicable standards and must be accredited by the Standards Council of Canada (SCC). In Canada, the following organizations have been accredited by the SCC to certify drinking water devices and materials as meeting NSF/ANSI standards (SCC, 2011):

- Canadian Standards Association International ([www.csa-international.org](http://www.csa-international.org));
- NSF International ([www.nsf.org](http://www.nsf.org));
- Water Quality Association ([www.wqa.org](http://www.wqa.org));
- Underwriters Laboratories, Inc. ([www.ul.com](http://www.ul.com));
- Quality Auditing Institute ([www.qai.org](http://www.qai.org)); and
- International Association of Plumbing & Mechanical Officials ([www.iapmo.org](http://www.iapmo.org)).

An up-to-date list of accredited certification organizations can be obtained from the SCC ([www.scc.ca](http://www.scc.ca)).

## 8.0 Kinetics and metabolism

There is evidence of absorption of ammonia by the inhalation and oral routes of exposure. Most ammonia, either ingested from exogenous sources or produced endogenously in the intestinal tract, is absorbed. Very little is known about the distribution of ammonia through inhalation and dermal exposures. A substantial part of ammonia is metabolized in the liver, where it is transformed into urea and glutamine. Ammonia or ammonium ion reaching the tissues is taken up by glutamic acid, which is an intermediate in other reactions. The principal means of excretion of ammonia that reaches the circulation in mammals is as urinary urea; minimal amounts are excreted in the faeces and in exhaled air. Although some data exist on distribution of ammonia from the oral exposure, there are no quantitative data published on distribution of ammonia from the inhalation and dermal exposure (U.S. EPA, 1989; ATSDR, 2004).

### 8.1 Absorption

Most of the ammonium ion in humans is endogenously produced in the digestive tract (4200 mg/day on average), much of it arising from the bacterial degradation of nitrogenous compounds from ingested food. More than 70% is synthesized or liberated within the colon and its faecal contents (Summerskill and Wolpert, 1970). About 99% of the ammonium ion endogenously produced is absorbed. Although the ammonium ion is a normal constituent of plasma at low levels, human and animal data show that little of the ammonium ion absorbed from the gastrointestinal tract reaches the systemic circulation as ammonia or ammonium compounds. Rather, it is absorbed via the hepatic portal vein directly to the liver, where most of it is converted to urea and glutamine in healthy individuals (Brown et al., 1957; Salvatore et al., 1963; Summerskill and Wolpert, 1970; Pitts, 1971). Analysis of plasma drawn from 10 healthy young male subjects yielded a mean endogenously derived ammonium ion concentration of 39 µg/100 mL (Brown et al., 1957). Analysis of urinary urea from subjects following oral administration of <sup>15</sup>N-labelled ammonium chloride solution indicated that 30–65% of the radioactive nitrogen administered was absorbed and metabolized (Richards et al., 1975; Metges et al., 1999).

Information on exposure to exogenous ammonia by the oral route predominantly involves case reports of people who have swallowed household ammonia. Studies provide evidence of ammonia absorption by the oral route, few provide quantitative data. For example, ammonium



ion concentrations of 153 and 33 ppm were detected in the stomach and blood, respectively, of a man who died following the ingestion of an unknown amount of ammonium hydroxide solution (Klendshoj and Rejent, 1966). In a study conducted in volunteers who ingested approximately 15 mg  $\text{NH}_4^+$ /kg bw/day as a single dose, 11 out of 20 subjects showed a 33% increase in arterial blood concentrations of ammonium above fasting levels, whereas cirrhotic patients showed increases of about 150% of arterial blood concentrations of ammonium, followed by a slow return to normal levels (Conn, 1972).

Inhalation studies conducted in humans (Silverman et al., 1949; Landahl and Hermann, 1950) and animals (Boyd et al., 1944; Dalhamn, 1963; Egle, 1973) suggest that ammonia is mainly retained in the upper respiratory tract and that only a small proportion is absorbed into the blood. Animal studies also suggested that an adaptive response mechanism may be activated with long-term exposure (Schaerdel et al., 1983).

No relevant quantitative data on the dermal absorption of ammonia were found in available literature.

## 8.2 Distribution and metabolism

No quantitative data on the distribution and metabolism of exogenously introduced ammonia in humans were located in the available literature. Ammonia plays a part in maintaining the acid-base balance in tissues of mammals. The dynamic equilibrium of the ammonium ion with ammonia in an aqueous solution is dependent on pH. For example, at physiological pH (~7.4), ammonium hydroxide is 99% in the ammonium ion form, but only 50% would be ionized at pH 9.25 (ATSDR, 2004). It is known that non-ionized ammonia ( $\text{NH}_3$ ) is freely diffusible, whereas the ammonium ion is less so, and it is mostly confined to the extracellular compartment (Stabenau et al., 1958).

Human oral exposure data clearly indicate that ammonia readily enters the portal circulation and is delivered to the liver. The most substantial first-pass metabolites of ammonia are urea and glutamine (Fürst et al., 1969; Pitts, 1971; Conn, 1972), as has been demonstrated for endogenously produced ammonia (Summerskill and Wolpert, 1970; Pitts, 1971). Ammonia and the ammonium ion can be rapidly converted to glutamine in the brain and other tissues (Takagaki et al., 1961; Warren and Schenker, 1964). The nitrogen is released from glutamine within tissues and used for protein synthesis as needed (Duda and Handler, 1958; Vitti et al., 1964; Fürst et al., 1969; Richards et al., 1975). In hypophysectomized rats that were administered [ $^{15}\text{N}$ ]ammonium citrate orally by gavage, labelled protein was found in liver, kidney, spleen, heart and skeletal muscle 6–72 hours after administration (Vitti et al., 1964). The administration of ammonium salt to humans by gavage resulted in a corresponding increase in blood urea concentration transported out of the liver (Fürst et al., 1969). Information on the distribution of endogenously produced ammonia suggests that the proportion of ammonia absorbed through inhalation would be distributed to all body compartments via the blood, where it would be used in protein synthesis or as a buffer, and that excess levels would be reduced to normal levels by urinary excretion or converted by the liver to glutamine and urea. If present in quantities that overtax these organs, ammonia is distributed to other tissues and is known to be detoxified in the brain (Takagaki et al., 1961; Warren and Schenker, 1964).

No quantitative data on the distribution of ammonia following dermal exposure were found in the available literature.

There is no published information on the transfer of ammonia from pregnant women to the fetus through the placenta or from nursing women to their offspring through breast milk.

However, umbilical uptake of ammonia has been demonstrated in the lamb (Marconi et al., 1989).

### **8.3 Excretion**

In healthy people, ammonia is efficiently metabolized via the urea cycle, primarily in the liver, and eliminated in the urine and faeces (Fürst et al., 1969; Richards et al., 1975). A limited study on human exposure to <sup>15</sup>N-labelled ammonia in drinking water showed that 25% of the initial dose was excreted in the urine within the first 6 hours after exposure, and approximately 72% within 3 days (Fürst et al., 1969). Small amounts of labelled nitrogen were also excreted as urea in faeces (Richards et al., 1975). These data are in agreement with data on the excretion of endogenously produced ammonia (Summerskill and Wolpert, 1970).

## **9.0 Health effects**

### **9.1 Effects in humans**

Information regarding the health effects of ammonia in humans consists largely of case reports of fatalities or illnesses following massive inhalation and/or dermal exposures resulting from accidental explosions or leakages. Controlled studies on the effects of oral exposure are limited. In general, more data are available on inhalation exposure than on oral or dermal exposure.

#### *9.1.1 Acute toxicity*

Ingestion of concentrated ammonia causes irritation and damage to the mouth, throat and gastrointestinal tract. However, such an exposure scenario is unlikely at the levels of ammonia encountered in the environment. The few case reports of acute oral exposures to ammonia were not conclusive, as no dose information was provided. Poisoning events in humans and related deaths have been reported following accidental or intentional ingestion of household ammonium salts (Klendshoj and Rejent, 1966; Klein et al., 1985), but no quantitative data are available, although levels found in household ammonium salts are expected to be significantly higher than those in drinking water. Qualitative observations reported include oesophageal lesions and oedema, as reported in five persons who ingested household ammonia as ammonium hydroxide, one of whom experienced acute respiratory obstruction (Klein et al., 1985; Christesen, 1995). A 69-year-old woman who ingested an unknown quantity of lemon ammonia (3% ammonium ion) was found semi-conscious and making gurgling respiratory sounds (Klein et al., 1985). Radiographic results were consistent with aspiration pneumonia. The main alterations determined by endoscopic examinations were laryngeal and epiglottic oedema and a friable, erythematous oesophagus with severe corrosive injury. Death occurred several days later following acute respiratory distress syndrome and renal failure (Klein et al., 1985). Klendshoj and Rejent (1966) also reported acute toxicity causing the death of a 57-year-old man who ingested an unknown amount of ammonium hydroxide; autopsy showed haemorrhagic oesophagus, stomach, and duodenum.

Several cases of gastrointestinal disorders have been described among young children (2–3 years old) who bit into ammonia pellets or capsules (Lopez et al., 1988; Rosenbaum et al., 1998). All of the children experienced one or more of the following symptoms: vomiting, drooling, dysphagia, cough, and oral or pharyngeal burns. In the reported cases, none of the children had oesophageal or respiratory burns, and all healed within a few days. In another study, oesophageal lesions, acute respiratory obstruction and oedema were reported following ingestion

of household ammonium hydroxide (Klein et al., 1985; Christesen, 1995). These observations were not quantified. Overall, several cases of accidental acute exposure to ammonia gas in humans have resulted in death (Price et al., 1983; Arwood et al., 1985; Burns et al., 1985) or respiratory tract irritation (de la Hoz et al., 1996). The concentrations of ammonia were not clearly quantified in these studies, although the levels of ammonia were higher than those found in drinking water or environmental exposure levels.

#### *9.1.2 Subchronic and chronic toxicity and carcinogenicity*

No information was available regarding systemic effects (including respiratory, cardiovascular, haematological, hepatic and endocrine effects) of ammonia or ammonium compounds in humans following chronic oral exposure.

The available chronic exposure data are primarily related to inhalation. Several studies of farmers working in enclosed livestock facilities indicate that ammonia may contribute to transient respiratory distress (Vogelzang et al., 1997, 2000; Cormier et al., 2000; Donham et al., 2000; Melbostad and Eduard, 2001). However, it is not clear from these studies what the contribution of ammonia is to the respiratory changes; other factors, including co-exposure to dust, carbon dioxide, endotoxins, fungi, bacteria and/or moulds, complicate the interpretation of these studies.

There are no validated data available regarding carcinogenic effects of ammonia or ammonium compounds in humans following oral exposure. Ammonia has not been classified by the International Agency for Research on Cancer (IARC) according to carcinogenicity.

#### *9.1.3 Neurotoxicity*

Neurological symptoms of acute exposure to highly concentrated anhydrous ammonia aerosols include blurred vision, diffuse non-specific encephalopathy, loss of consciousness, muscle weakness and decreased deep tendon reflexes (George et al., 2000).

Ammonia has potentially deleterious effects on the central nervous system. Depending upon the severity and duration of exposure, these effects may include seizures and cerebral palsy (Felipo and Butterworth, 2002).

No information was found regarding neurological effects of ammonia or ammonium compounds in humans following oral exposure.

An increased concentration of ammonia in the blood and brain can occur as a result of hepatic encephalopathy, where liver function is impaired and the organ cannot metabolize ammonia (Felipo and Butterworth, 2002).

#### *9.1.4 Genotoxicity*

Data on the genotoxicity of ammonia in humans are limited to a study of 22 workers exposed to unknown concentrations of ammonia in air at a fertilizer factory compared with 42 control workers (Yadav and Kaushik, 1997). The results of blood sample analyses to detect genotoxic impacts showed a significant increase in the frequency of chromosomal aberrations, sister chromatid exchange and micronuclei induction in exposed workers compared with controls. These results reveal the genotoxic potential of ammonia. The authors clearly demonstrated dose-response correlations, although it is important to be mindful of the possible confounding factors associated with such a study.



## 9.2 Effects on experimental animals

### 9.2.1 Acute toxicity

Acute studies in animals support the fact that the respiratory tract is a sensitive target of ammonia toxicity (Richard et al., 1978; Kapeghian et al., 1982; Schaerdel et al., 1983). Acute exposures (1 hour to 1 week) to low concentrations of ammonia in air ( $\leq 1000$  ppm) irritate the upper respiratory tract, whereas exposures (3 hours to 2 weeks) to high concentrations ( $\geq 4000$  ppm) result in severe damage to the upper and lower respiratory tract and alveolar capillaries (Coon et al., 1970; Richard et al., 1978; Kapeghian et al., 1982; Schaerdel et al., 1983). Other effects on remote organs (renal, cardiovascular) observed following inhalation exposure were not consistent and may be secondary to the respiratory tract damage.

The syndrome of ammonia intoxication in rats, guinea pigs and cats consists mainly of dyspnoea, muscle fasciculation and convulsions, terminating in an early acute pulmonary oedema (Koenig and Koenig, 1949). However, the results are not consistent through all the studies. A single gavage dose study (Koenig and Koenig, 1949) showed that an ammonium dose of 303 mg/kg bw as ammonium chloride was lethal to guinea pigs (30/40 died) as a result of pulmonary oedema. In contrast, Boyd and Seymour (1946) reported no deaths in cats, rabbits, guinea pigs or rats after administration of a similar dose of ammonium (337 mg/kg bw as ammonium chloride). Other dose–response studies in rats exposed to ammonia for 15, 30 and 60 minutes have been used to establish median lethal concentration ( $LC_{50}$ ) values of 112, 71.9 and 48.4 mg/L, respectively (ATSDR, 2004). However, the consistency of the database is limited for various reasons, including the use of single exposure data only (Koenig and Koenig, 1949) or too high dosages (Barzel, 1975). In addition, the associated anion in the ammonium salt administered plays an important role. In fact, ammonium chloride is widely used to induce metabolic acidosis in animal studies; it is now known that the metabolic acidosis that can affect the lungs, kidney, nervous system, liver and bone is actually due to the formation of hydrogen chloride. For example, De Sousa et al. (1974) showed that the decrease in plasma bicarbonate induced by the administration of hydrochloric acid to dogs was significantly greater than that induced by the administration of equivalent quantities of hydrogen ion as nitric or sulfuric acid. It is therefore inappropriate to extrapolate findings obtained with ammonium chloride (or any ammonium salt) to equivalent amounts of ammonium derived from a different salt. This is one reason why caution should be exercised in deriving an oral minimal risk level for ammonia.

### 9.2.2 Short-term exposure

As with acute exposure, the animal studies that examined the toxicity of short-term intermittent or continuous exposure to ammonia suggest that the respiratory tract is the most sensitive target of toxicity. Symptoms of irritation, nasal lesions, dyspnoea and pulmonary inflammation have been observed in several animal species (Coon et al., 1970; Broderson et al., 1976; Gaafar et al., 1992).

Administration of ammonia in drinking water to rats at a dose of approximately 42 mg/kg bw/day for 8 weeks resulted in accelerated cell migration leading to mucosal atrophy in the stomach antrum and enlargement of the proliferative zone in the atrum (Tsujii et al., 1993).

However, continuous inhalation exposure of groups of rats to ammonia concentrations ranging from 40 to 470 mg/m<sup>3</sup> showed no evidence of toxicity in 15 rats exposed to 40 mg/m<sup>3</sup> for 114 days or 48 rats exposed to 127 mg/m<sup>3</sup> for 90 days (Coon et al., 1970). In the same study, of 49 rats exposed continuously to ammonia at a concentration of 262 mg/m<sup>3</sup> in air for 90 days, 25% had mild nasal discharge; 50 of 51 rats died at day 65 of continuous exposure to ammonia at a concentration of 455 mg/m<sup>3</sup>, whereas 13 of 15 rats exposed to ammonia at a concentration of

470 mg/m<sup>3</sup> died before the end of the study. There were no significant haematological differences between experimental and control animals examined following a continuous 90-day exposure of rats to an ammonia concentration of 127 mg/m<sup>3</sup>.

Although no short-term dermal exposure studies were identified, based on the irritant properties of ammonia, it is reasonable to assume that direct contact of the skin with ammonia for a prolonged time will produce irritation.

### 9.2.3 Long-term exposure and carcinogenicity

The available information does not suggest that ammonia is carcinogenic. However, well-designed studies in animals have not been conducted, and the relevance of the available data to assess the cancer risk of oral exposure to ammonia is uncertain (ATSDR, 2004).

Exposure of 50 randomly bred 5-week-old Swiss C3H mice to ammonium at a dose of 193 mg/kg bw/day as ammonium hydroxide in drinking water for 2 years did not produce carcinogenic effects, nor did it affect spontaneous development of breast adenocarcinomas, which are characteristic of these animals (Toth, 1972). In another study, mice treated by gavage with ammonia dissolved in water at a dose of 42 mg/kg bw/day as ammonium ion for 4 weeks did not show any evidence of a carcinogenic effect (Uzvölgyi and Bojan, 1980). However, the authors demonstrated that, in the presence of ammonia, a non-carcinogenic precursor can initiate the development of lung tumours. For example, in the study above, when mice were treated with diethyl pyrocarbonate (a widely used antimicrobial agent for the preservation of beverages and food) prior to the administration of ammonia, lung tumours were observed in 9 of 16 mice; the reaction of diethyl pyrocarbonate or its by-products with ammonia may have formed urethane, a known carcinogen. In addition, Tsujii et al. (1995) demonstrated that gastric cancer metastasis significantly increased in rats pretreated with the initiator *N*-methyl-*N*-nitro-*N*-nitrosoguanidine in drinking water 24 weeks before receiving ammonia solution (estimated dose 200 mg/day), compared with control rats receiving ammonia only.

### 9.2.4 Genotoxicity

Very limited *in vivo* and *in vitro* studies pertaining to the genotoxicity of ammonia are available.

#### 9.2.4.1 In vitro findings

Early studies suggesting that ammonia may be mutagenic have been reviewed in other work (U.S. EPA, 1989). *In vitro* studies demonstrated that ammonia was able to induce back-mutations from dependence on streptomycin in *E. coli* (Demerec et al., 1951). The authors suggested that the mutagenic effect observed may not be specific, but that treatment with ammonia may increase the mutation rate of the whole genome. The addition of ammonia solution to mouse 3T3 cells resulted in a dramatic decrease in cellular multiplication ( $p < 0.001$ ) and changes in morphology (Visek et al., 1972).

#### 9.2.4.2 In vivo findings

The effect of exposure of larvae of *Drosophila melanogaster* to ammonia was examined by Lobashev and Smirnov (1934). A 95% mortality rate was reported when the flies were exposed to fumes of 10 000 ppm ammonia hydroxide solution. The offspring of the survivors displayed a mutation rate of 0.54%, which was statistically significant in comparison with controls, which showed a rate of 0.05%.

Several *in vivo* studies in *D. melanogaster* resulted in a positive response for mutagenic lethality, but negative responses for sex-linked recessive lethal mutations and dominant lethality (Auerbach and Robson, 1947).

#### 9.2.5 Reproductive and developmental toxicity

There are no adequate studies for assessing the potential reproductive toxicity of ammonia. Very limited data were found regarding developmental effects of ammonia in animals. The most relevant study available was conducted on female Wistar rats (Miñana et al., 1995). Rats exposed to ammonium ion through their mother's diet (estimated dose 4293 mg/kg bw/day for the mothers) both *in utero* from gestational day 1 and through lactation, followed by a normal diet after lactation, had offspring with a marked decrease in growth rate. The authors suggested that the reduced growth of ammonia-exposed rats could be a consequence of the impaired function of *N*-methyl-D-aspartate receptors.

#### 9.2.6 Neurotoxicity

Neurological effects of acute exposure to low levels of ammonia (100 ppm) via inhalation include depression of free-access wheel running behaviour in rodents (Tepper et al., 1985). Data concerning the oral route of exposure were not available.

### 9.3 Mode of action

The mode of action for ammonia varies with the route of exposure. Many reported effects of ammonia are due to its alkalinity, which results in tissue damage (ATSDR, 2004). Toxicological information considered appropriate for the inhalation route of exposure appears different from the oral route of exposure; however, little information is available for oral exposure. There are no overt data on the pharmacokinetics of ammonia in the available literature, and no health-based endpoint that occurs from ingestion of ammonia at current exposure levels has been identified.

In general, ammonia is thought to alter the acid–base balance in the body, which in turn can result in physiological effects such as an alteration in glucose tolerance and a decreased sensitivity to insulin (U.S. EPA, 1989; WHO, 2003).

## 10.0 Classification and assessment

Ammonia is formed endogenously and is present in the human body at concentrations significantly higher than those typically found in drinking water. Ammonia is also efficiently metabolized by the body, and little information concerning the toxicity of ammonia through the ingestion route of exposure was found in the literature.

There are no validated data available regarding carcinogenic effects of ammonia or ammonium compounds in humans following oral exposure. Ammonia has not been classified by IARC according to its carcinogenicity.

In humans, most health effects reported as a result of ammonia exposure are associated with exposure through inhalation, which is not a relevant mode of action in the consideration of toxicity associated with drinking water. Although ingestion of concentrated ammonia causes irritation and damage to the mouth, throat and gastrointestinal tract, these effects are unlikely to occur at the concentrations of ammonia found in drinking water (Klendshoj and Rejent, 1966; Klein et al., 1985; Lopez et al., 1988).

In animals, acute and short-term exposure studies suggest that the respiratory tract is the most sensitive target of toxicity. Available scientific evidence does not suggest that ammonia is carcinogenic in animals. However, well-designed studies have not been conducted, and the relevance of the available data to assess the cancer risk of oral exposure to ammonia is uncertain.

Based on the lack of an appropriate endpoint from the ingestion of ammonia, the lack of sufficient evidence of systemic effects in humans, as well as limited relevant studies in experimental animals, no health-based guideline can be derived for ammonia in drinking water.

### **10.1 International considerations**

The World Health Organization has assessed the toxicity of ammonia in drinking water and concluded that a guideline value need not be established, based on the absence of health effects at the concentrations to be expected in drinking water (WHO, 2003).

The U.S. EPA has not established a regulatory limit for the presence of ammonia in drinking water. In Australia, an aesthetic limit (based on the corrosion of copper pipes and fittings) has been established at 0.5 mg/L, measured as ammonia. No health-based guideline value has been established for ammonia (NHMRC, 2004).

## **11.0 Rationale**

The production of ammonia is a normal part of metabolism, and levels occurring in the human body from metabolic processes generally exceed concentrations found in drinking water. Furthermore, health effects associated with ammonia exposure have primarily been reported for the inhalation route of exposure, and little information is available concerning the toxicological effects resulting from ingestion of ammonia.

Although no health-based guideline value can be derived for ammonia in drinking water, it is still important to maintain minimal levels of ammonia in drinking water. High levels of ammonia in water are associated with a number of potential treatment issues, including nitrification. Once nitrification occurs, it may cause significant degradation of the water quality, including increases in nitrite/nitrate concentrations, decreases in chloramine residual and increases in bacterial counts. Additional effects that may be observed include decreases in pH, decreases in oxygen concentration and corrosion issues in the distribution system. The end result of nitrification has significant health concerns, as outlined in the Guideline Technical Document on nitrate/nitrite. Limiting the excess of free ammonia entering the distribution system to concentrations below 0.1 mg/L, and preferably below 0.05 mg/L, as ammonia-nitrogen, will help prevent nitrification.

## **12.0 References**

Abd El-Hady, H.M., Grunwald, A., Vlekova, K. and Zeithammerova, J. (2001). Lewatit S100 in drinking water treatment for ammonia removal. *Acta Polytech.*, 41(1): 31–33.

Alberta Environment (2010). Personal communication from D. Reid, Drinking Water and Wastewater Section, Water Policy Branch, Edmonton, Alberta, March.

Andersson, A., Laurent, P., Kihn, A., Prévost, M. and Servais, P. (2001). Impact of temperature on nitrification in biological activated carbon (BAC) filters used for drinking water treatment. *Water Res.*, 35(12): 2923–2934.

- APHA, American Water Works Association and Water Environment Federation (2005). Standard methods for the examination of water and wastewater. 21<sup>st</sup> edition. American Public Health Association, Washington, DC.
- Arwood, R., Hammond, J. and Ward, G.G. (1985). Ammonia inhalation. *J. Trauma*, 25(5): 444–447.
- Asman, W.A.H., Sutton, M.A. and Schjorring, J.K. (1998). Ammonia: emission, atmospheric transport and deposition. *New Phytol.*, 139(1): 27–48.
- ASTM (2003). Standard test methods for ammonia nitrogen in water. American Society for Testing and Materials. West Conshohocken, PA, 2003. DOI: 10.1520/D1426-03. Available at: [www.astm.org](http://www.astm.org)
- Atlas, R.M. and Bartha, R. (1998). Biochemical cycling: nitrogen, sulfur, phosphorus, iron, and other elements. In: *Microbial ecology. Fundamentals and applications*. 4<sup>th</sup> edition. L. Fogel and G. Wong (eds.). Benjamin/Cummings Science Publishing, Menlo Park, California. pp. 414–425.
- ATSDR (2004). Toxicological profile for ammonia. Agency for Toxic Substances and Disease Registry, Atlanta, Georgia Available at: [www.atsdr.cdc.gov/toxprofiles/tp.asp?id=11&tid=2](http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=11&tid=2)
- Auerbach, C. and Robson, J.M. (1947). XXXIII. Test of chemical substances for mutagenic action. *Proc. R. Soc. Edinb. (Nat. Environ.)*, 62: 284–291.
- AWWA (2006). Water chlorination/chloramination practices and principles: manual of water supply practices. 2nd edition. American Water Works Association, Denver, Colorado.
- Bablon, G.P., Ventresque, C. and Ben Aim, R. (1988). Developing a sand–GAC filter to achieve high-rate biological filtration. *J. Am. Water Works Assoc.*, 80(12): 47–53.
- Baribeau, H. (2006). Chapter 6: Growth and inactivation of nitrifying bacteria. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems*. Manual of Water Supply Practices M56. 1st edition. American Water Works Association, Denver, Colorado.
- Baribeau, H. (2010). Chloramination issues: overview of nitrification. In: *Proceedings of the American Water Works Association Inorganic Contaminants Workshop*, Denver, Colorado. March 2, 2010. American Water Works Association, Denver, Colorado.
- Barzel, U.S. (1975). The effect of chronic ammonium chloride ingestion on parathyroid hormone function. *Nephron*, 14(5): 339–346.
- Bouwer, E.J. and Crowe, P.B. (1988). Biological processes in drinking water treatment. *J. Am. Water Works Assoc.*, 80(9): 82–93.
- Bouwman, A.F., Lee, D.S., Asman, W.A.H., Dentener, F.J., Van Der Hoek, K.W. and Olivier, J.G.J. (1997). A global high-resolution emission inventory for ammonia. *Global Biogeochem. Cycles*, 11(4): 561–587.
- Boyd, E.M. and Seymour, K.G.W. (1946). Ethylenediamine dihydrochloride or chlor-ethamine. II. Untoward and toxic reactions. *Exp. Med. Surg.*, 4: 223–227.
- Boyd, E.M., MacLachlan, M.L. and Perry, W.F. (1944). Experimental ammonia gas poisoning in rabbits and cats. *J. Ind. Hyg. Toxicol.*, 26(1): 29–34.
- Bremer, P.J., Webster, B.J. and Wells, D.B. (2001). Biocorrosion of copper in potable water. *J. Am. Water Works Assoc.*, 93(8): 82–91.
- Broderson, J.R., Lindsey, J.R. and Crawford, J.E. (1976). The role of environmental ammonia in respiratory mycoplasmosis of rats. *Am. J. Pathol.*, 85(1): 115–130.

Broseus, R., Cigana, J., Barbeau, B., Daines, C. and Suty, H. (2009). Removal of total dissolved solids, nitrates and ammonium ions from drinking water using charge-barrier capacitive deionisation. *Desalination*, 249(1): 217–223.

Brown, R.H., Duda, G.D., Korke, S. and Handler, P. (1957). A colorimetric micromethod for determination of ammonia: the ammonia content of rat tissue and human plasma. *Arch. Biochem. Biophys.*, 66: 301–309.

Burns, T.R., Greenberg, S.D., Mace, M.L. and Jachimczyk, J.A. (1985). Ultrastructure of acute ammonia toxicity in the human lung. *Am. J. Forensic Med. Pathol.*, 6(3): 204–210.

Camford Information Services (2003). CPI product profile for ammonia. Scarborough, Ontario. Available at: [www.camfordinfo.com/pdfs/cpipp\\_order\\_form.pdf](http://www.camfordinfo.com/pdfs/cpipp_order_form.pdf)

Cevaal, J.N., Suratt, W.B. and Burke, J.E. (1995). Nitrate removal and water quality improvements with reverse osmosis for Brighton, Colorado. *Desalination*, 103(1-2): 101–111.

Charrois, J.W.A. and Hrudey, S.E. (2007). Breakpoint chlorination and free-chlorine contact time: implications for drinking water N-nitrosodimethylamine concentration. *Water Research* . 41, 674-682

Chowdhury, Z.K., Passantino, L., Summers, S., Work, L., Rossman, L. and Uber, J. (2006). Assessment of chloramine and chlorine residual decay in the distribution system. American Water Works Association Research Foundation and American Water Works Association, Denver, Colorado.

Christesen, H.B.T. (1995). Prediction of complications following caustic ingestion in adults. *Clin. Otolaryngol.*, 20(3): 272–278.

Clifford, D.A. (1999). Chapter 9. Ion exchange and inorganic adsorption. In: *Water quality and treatment: a handbook of community water supplies*. 5th edition. R.D. Letterman (ed.). McGraw-Hill, New York, New York.

Cohen, K. and Friedman, M. (2006). Chapter 1: Introduction to nitrification in drinking water and impact on regulatory compliance. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems*. Manual of Water Supply Practices M56. 1st edition. American Water Works Association, Denver, Colorado.

Cohen, Y., Smith, S. and Baribeau, H. (2001). Nitrification: causes, prevention and control. *Opflow*, 27(12): 6–12.

Conn, H.O. (1972). Studies of the source and significance of blood ammonia. IV. Early ammonia peaks after ingestion of ammonia salts. *Yale J. Biol. Med.*, 45(5): 543–549.

Coon, R.A., Jones, R.A., Jenkins, L.J., Jr. and Siegel, J. (1970). Animal inhalation studies on ammonia, ethylene, glycol, formaldehyde, dimethylamine and ethanol. *Toxicol. Appl. Pharmacol.*, 16(3): 646–655.

Cormier, Y., Israel-Assayag, E., Racine, G. and Duchaine, C. (2000). Farming practices and the respiratory health risks of swine confinement buildings. *Eur. Respir. J.*, 15(3): 560–565.

Crittenden, J.C., Trussell, R.R., Hand, D.W., Howe, K.J. and Tchobanoglous, G. (2005). *Water treatment: principles and design*. 2nd edition. John Wiley & Sons, Hoboken, New Jersey.

Dalhamn, T. (1963). Effect of ammonia alone and combined with carbon particles on ciliary activity in the rabbit trachea *in vivo*, with studies of the absorption capacity of the nasal cavity. *Int. J. Air Water Pollut.*, 7: 531–539.

de la Hoz, R.E., Schlueter, D.P. and Rom, W.N. (1996). Chronic lung disease secondary to ammonia inhalation injury: a report on three cases. *Am. J. Med.*, 29(2): 209–214.

Demerec, M., Bertani, G. and Flint, J. (1951). A survey of chemicals for mutagenic action on *E. coli*. *Am. Nat.*, 85(821): 119–136.



- Demir, A., Günay, A. and Debik, E. (2002). Ammonium removal from aqueous solution by ion-exchange using packed bed natural zeolite. *Water SA*, 28(3): 329–335.
- Denmead, O.T., Freney, J.R. and Simpson, J.R. (1982). Atmospheric dispersion of ammonia during application of anhydrous ammonia fertilizer. *J. Environ. Qual.*, 11: 568–572.
- Department of National Health and Welfare (1993). *Water treatment principles and applications. A manual for the production of drinking water.* Published by the Canadian Water and Wastewater Association in cooperation with the Communication Group, Ottawa, Ontario.
- De Sousa, R.C., Harrington, J.T., Ricanati, E.S., Shelkrot, J.W. and Schwartz, W.B. (1974). Renal regulation of acid–base equilibrium during chronic administration of mineral acid. *J. Clin. Invest.*, 53(2): 465–476.
- Donham, K.J., Cumro, D., Reynolds, S.J. and Merchant, J.A. (2000). Dose–response relationships between occupational aerosol exposures and cross-shift declines of lung function in poultry workers: recommendations for exposure limits. *J. Occup. Environ. Med.*, 42(3): 260–269.
- Douglas, I., Guthmann, J., Muylwyk, Q. and Snoeyink, V. (2004). Corrosion control in the City of Ottawa -- Comparison of alternatives and case study for lead reduction in drinking water. In: Robertson, W. and Brooks, T. (Ed.). 11th Canadian National Drinking Water Conference and Second Policy Forum. Calgary, Alberta, Canada. April 3 - 6.
- Duda, G.D. and Handler, P. (1958). Kinetics of ammonia metabolism *in vivo*. *J. Biol. Chem.*, 232(1): 303–314.
- Edwards, M. and Dudi, A. (2004). Role of chlorine and chloramine in corrosion of lead-bearing plumbing materials. *J. Am. Water Works Assoc.*, 96(10): 69–81.
- Egle, J.L. (1973). Retention of inhaled acetone and ammonia in the dog. *Am. Ind. Hyg. Assoc. J.*, 34(12): 533–539.
- Environment Canada and Health Canada (2001). Canadian Environmental Protection Act, 1999. Priority Substances List Assessment Report. Ammonia in the aquatic environment. Ottawa, Ontario. Available at: [www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl2-lsp2/ammonia/index-eng.php](http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl2-lsp2/ammonia/index-eng.php)
- Felipo, V. and Butterworth, R.F. (2002). Neurobiology of ammonia. *Prog. Neurobiol.*, 67(4): 259–279.
- Fleming, K., Harrington, G.W. and Noguera, D.R. (2005). Nitrification potential curve: a new strategy for nitrification prevention. *J. Am. Water Works Assoc.*, 97(8): 90–99.
- Fleming, K., Harrington, G.W. and Noguera, D.R. (2008). Using nitrification potential curve to evaluate full scale drinking water distribution system. *J. Am. Water Works Assoc.*, 100(10): 92–103.
- Fürst, P., Josephson, B., Maschio, G. and Vinnars, E. (1969). Nitrogen balance after intravenous and oral administration of ammonium salts to man. *J. Appl. Phys.*, 26(1): 13–22.
- Gaafar, H., Girgis, R., Hussein, M. and el-Nemr, F. (1992). The effect of ammonia on the respiratory nasal mucosa of mice. A histological and histochemical study. *Acta Otolaryngol.*, 112(2): 339–342.
- Gaspard, M., Neveu, A. and Martin, G. (1983). Clinoptilolite in drinking water treatment for NH<sub>4</sub><sup>+</sup> removal. *Water Res.*, 17(3): 279–288.
- Gauntlett, R. (1981). Removal of ammonia and nitrate in the treatment of potable water. In: Biological fluidized bed treatment of water and wastewater. Cooper P. and Atkinson, B. (eds.). Published for the Water Research Centre, Stevenage Laboratory, by Horwood. p. 48.
- George, A., Bang, R.L., Lari, A.R., Gang, R.K. and Kanjoor, J.R. (2000). Liquid ammonia injury. *Burns*, 26(4): 409–413.

- Goodall, J.B. (1979). Biological removal of ammonia. U.S. Environmental Protection Agency, Washington, DC (EPA 570/19-79/020).
- Goulding, K.W.T., Bailey, N.J., Bradbury, N.J., Hargreaves, P., Howe, M., Murphy, D.V., Poultron, P.R. and Willison, T.W. (1998). Nitrogen deposition and its contribution to nitrogen cycling and associated soil processes. *New Phytol.*, 139(1): 49–58.
- Groeneweg, J., Sellner, B. and Tappe, W. (1994). Ammonia oxidation in *Nitrosomonas* at  $\text{NH}_3$  concentration near  $K_m$ : effects of pH and temperature. *Water Res.*, 28(12): 25–61.
- Haralambous, A., Maliou, E. and Malamis, M. (1992). The use of zeolite for ammonia uptake. *Water Sci. Technol.*, 25(1): 139–145.
- Harrington, G.W., Noguera, D.R., Kandou, A.I. and Vanhoven, D.J. (2002). Pilot-scale evaluation of nitrification control strategies. *J. Am. Water Works Assoc.*, 94(11): 78–89.
- Harrington, G., Noguera, D., Bone, C., Kandou, A., Oldenburg, P., Regan, J. and van Hovem, D. (2003). Ammonia from chloramine decay: effects on distribution system nitrification. American Water Works Association Research Foundation and American Water Works Association, Denver, Colorado.
- Health Canada (1995). Guidelines for Canadian drinking water quality: Supporting document—chloramines. Bureau of Chemical Hazards, Health Protection Branch, Ottawa, Ontario. Available at: [www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/chloramines/index-eng.php](http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/chloramines/index-eng.php)
- Health Canada (2008a). Personal communication from K. Taracha, Public Health Engineering Section, First Nations and Inuit Health Branch, Ottawa, Ontario, July.
- Health Canada (2008b). Guidelines for Canadian drinking water quality: Guideline technical document—Chlorite and chlorate. Water Quality and Health Bureau, Healthy Environments and Consumer Safety Branch, Ottawa, Ontario. Available at: [www.hc-sc.gc.ca/ewh-semt/alt\\_formats/hecs-sesc/pdf/pubs/water-eau/chlorite-chlorate/chlorite-chlorate-eng.pdf](http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/chlorite-chlorate/chlorite-chlorate-eng.pdf)
- Health Canada (2011). Guidelines for Canadian drinking water quality: Guideline technical document—N-Nitrosodimethylamine. Water, Air and Climate Change Bureau, Healthy Environments and Consumer Safety Branch, Ottawa, Ontario. Available at: [www.hc-sc.gc.ca/ewh-semt/alt\\_formats/hecs-sesc/pdf/pubs/water-eau/ndma/ndma-eng.pdf](http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/ndma/ndma-eng.pdf)
- Health Canada (2012). Personal communication from A.M. Tugulea, Environmental and Radiation Health Sciences Directorate, Healthy Environments and Consumer Safety Branch, Ottawa, Ontario.
- Hill, C.P. and Arweiler, S. (2006). Chapter 9: Assessment and operational responses to nitrification episodes. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems. Manual of Water Supply Practices M56*. 1st edition. American Water Works Association, Denver, Colorado.
- Hodi, M., Polyak, K. and Hlavey, J. (1995). Removal of pollutants from drinking water by combined ion exchange and adsorption methods. *Environ. Int.*, 21(3): 325–331.
- Hossain, M., Fakhruddin, A.N.M. and Khan, S. (2007). Impact of raw water ammonia on the surface water treatment processes and its removal by nitrification. *Bangladesh J. Microbiol.*, 24(2): 85–89.
- HSDB (2005). Ammonia. Hazardous Substances Data Bank, National Library of Medicine, Bethesda, Maryland. Available at: <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/r?dbs+hsdb:@term+@DOCNO+162>



- Huang, Y.H. and Zhang, T.C. (2005). Effects of dissolved oxygen on formation of corrosion products and concomitant oxygen and nitrate reduction in zero-valent iron system with or without aqueous  $\text{Fe}^{2+}$ . *Water Res.*, 39(9): 1751–1760.
- Hutchinson, G.L., Mosier, A.R. and Andre, C.E. (1982). Ammonia and amine emissions from a large cattle feedlot. *J. Environ. Qual.*, 11(2): 288–293.
- IPCS (1986). Ammonia. International Programme on Chemical Safety, World Health Organization, Geneva (Environmental Health Criteria 54).
- Janda, V. and Rudovský, J. (1994). Removal of ammonia from drinking water by biological nitrification. *J. Water SRT – Aqua*, 43(3): 120–125.
- Kapeghian, J.C., Mincer, H.H., Jones, A.B., Verlagieri, A.J. and Waters, W. (1982). Acute inhalation toxicity of ammonia in mice. *Bull. Environ. Contam. Toxicol.*, 29(3): 371–378.
- Kihn, A., Andersson, A., Laurent, P., Servais, P. and Prévost, M. (2002). Impact of filtration material on nitrification in biological filters used in drinking water production. *J. Water SRT – Aqua*, 51(1): 35–46.
- Kirmeyer, G., Odell, L., Jacangelo, J., Wilczak, A. and Wolfe, R. (1995). Nitrification occurrence and control in chloraminated water systems. American Water Works Association Research Foundation and American Water Works Association, Denver, Colorado.
- Kirmeyer, G.J., LeChevallier, M., Baribeau, H., Martel, K., Thompson, G., Radder, L., Klement, W. and Flores, A. (2004). Optimizing chloramine treatment. 2nd edition. American Water Works Research Foundation and American Water Works Association, Denver, Colorado (AwwaRF Report 90993).
- Klein, J., Olson, K.R. and McKinney, H.E. (1985). Caustic injury from household ammonia. *Am. J. Emerg. Med.*, 3(4): 320–322.
- Klendshoj, N.C. and Rejent, T.A. (1966). Tissue levels of some poisoning agents less frequently encountered. *J. Forensic Sci.*, 11(1): 75–80.
- Koenig, H. and Koenig, R. (1949). Production of acute pulmonary edema by ammonium salts. *Proc. Soc. Exp. Biol.*, 70(3): 375–380.
- Kors, L.J., Moorman, J.H.N., Wind, A.P.M. and van der Hoek, J.P. (1998). Nitrification and low temperature in a raw water reservoir and rapid sand filters. *Water Sci. Technol.*, 37(2): 169–176.
- Koyuncu, I. (2002). Effect of operating conditions on the separation of ammonium and nitrate ions with nanofiltration and reverse osmosis membranes. *J. Environ. Sci. Health A Tox. Hazard. Subst. Environ. Eng.*, 37(7): 1347–1359.
- Koyuncu, I., Topacikk, D., Turan, M., Celik, M.S. and Sarikaya, H.Z. (2001). Application of membrane technology to control ammonia in surface water. *Water Sci. Technol. Water Supply*, 1(1): 117–124.
- Kurama, H., Poetzschke, J. and Haseneder, R. (2002). The application of membrane filtration for the removal of ammonium ions from potable water. *Water Res.*, 36(11): 2905–2909.
- Landahl, H.D. and Herrmann, R.G. (1950). Retention of vapors and gases in the human nose and lung. *Arch. Ind. Hyg. Occup. Med.*, 1(1): 36–45.
- Li, X. and Chu, H.P. (2003). Membrane bioreactor for the drinking water treatment of polluted surface water supply. *Water Res.*, 37(19): 4781–4791.

- Lin, S.H. and Wu, C.L. (1996). Ammonia removal from aqueous solution by ion exchange. *Ind. Eng. Chem. Res.*, 35(2): 553–558.
- Lobashev, M.E. and Smirnov, F.A. (1934). Nature of the action of chemical agents on mutational process in *Drosophila melanogaster*. II. The effect of ammonia on the occurrence of lethal transgenations. *Acad. Sci. C. R. Biol.*, 3: 174–176.
- Lopez, G., Dean, B.S. and Krenzelo, E.P. (1988). Oral exposure to ammonia inhalants: a report of 8 cases. *Vet. Hum. Toxicol.*, 30: 350.
- Lytle, D., Sorg, T., Wang, L., Muhlen, C., Rahrig, M. and French, K. (2007). Biological nitrification in a full scale and pilot scale iron removal drinking water treatment plant. *J. Water SRT – Aqua*, 56(2): 125–136.
- Marconi, A.M., Battaglia, F.C., Meschia, G. and Sparks, J.W. (1989). A comparison of amino acid arteriovenous differences across the liver and placenta of the fetal lamb. *Am. J. Physiol.*, 257(6 Pt. 1): E909–915.
- McGovern, B. and Nagy, R. (2010). Biological removal of iron, manganese and ammonia simultaneously utilizing pressurized downflow filtration. In: *Proceedings of the American Water Works Association Water Quality Technology Conference*. American Water Works Association, Denver, Colorado.
- McGuire, M., Lieu, N. and Pearthree, M. (1999). Using chlorite ion to control nitrification. *J. Am. Water Works Assoc.*, 91(10): 52–61.
- McGuire, M.J., Wu, X., Blute, N.K., Askenaizer, D. and Qin, G. (2009). Prevention of nitrification using chlorite ion: results of a demonstration project in Glendale, Calif. *J. Am. Water Works Assoc.*, 101(10): 47–58
- Melbostad, E. and Eduard, W. (2001). Organic dust-related respiratory and eye irritation in Norwegian farmers. *Am. J. Ind. Med.*, 39(2): 209–217.
- Metges, C.C., Petzke, K.H., El-Khoury, A.E., Henneman, L., Grant, I., Bedri, S., Regan, M.M., Fuller, M.F. and Young, V.R. (1999). Incorporation of urea and ammonia nitrogen into ileal and fecal microbial proteins and plasma free amino acids in normal men and ileostomates. *Am. J. Clin. Nutr.*, 70(6): 1046–1058.
- Miñana, M.D., Marcaidam, G., Grisolíam, S. and Felipo, V. (1995). Prenatal exposure of rats to ammonia impairs NMDA receptor function and affords delayed protection against ammonia toxicity and glutamate neurotoxicity. *J. Neuropathol. Exp. Neurol.*, 54(5): 644–650.
- Mori, S., Kaneko, H., Mitsuma, T., Hayakawa, T., Yamaguchi, C. and Urama, M. (1998). Implications of gastric tropical bioactive peptides in ammonia-induced acute gastric mucosal lesions in rats. *Scand. J. Gastroenterol.*, 33(4): 386-393.
- Muramoto, S., Udagawa, T. and Okamura, T. (1995). Effective removal of musty odor in the Kanamachi purification plant. *Water Sci. Technol.*, 31(11): 219–222.
- Muylywyk, Q. (2009). Chloramine 101: Introduction to planning, design, implementation, and operation with chloramine. A little bit of chloramines chemistry. *American Water Works Association Annual Conference and Exposition, June 14–18, 2009, San Diego, California*.
- Nagy, R. and Granlund, C. (2008). Field report—Biofiltration and RO combined to address water quality challenges. *J. Am. Water Works Assoc.*, 100(12): 52, 54–55.
- Nawrocki, J. and Andrzejewski, P. (2011). Nitrosamines and water. *J. Hazard. Mater.* 189 (1-18)
- NEMI (2010). National Environmental Methods Index. United States Geological Survey and United States Environmental Protection Agency. Available at: [www.nemi.gov](http://www.nemi.gov)

NHMRC (2004). Australian drinking water guidelines. National Health and Medical Research Council, Australian Government. Available at: [www.nhmrc.gov.au/publications/synopses/eh19syn.htm](http://www.nhmrc.gov.au/publications/synopses/eh19syn.htm)

Nova Scotia Environment (2010). Personal communication from C. Mosher, Water and Wastewater Branch, Halifax, Nova Scotia, February.

Odell, L., Kirmeyer, G., Wilzak, A., Jacangelo, J., Marchinko, J. and Wolfe, R. (1996). Controlling nitrification in chloraminated systems. *J. Am. Water Works Assoc.*, 88(7): 86–98.

Park, J.B., Lee, S.H., Lee, J.W. and Lee, C.Y. (2002). Lab scale experiments for permeable reactive barriers against contaminated groundwater with ammonia and heavy metals using clinoptilolite (01-29B). *J. Hazard. Mater.*, B95(1-2): 65–79.

Pintar, K.D. and Slawson, R.M. (2003). Effect of temperature and disinfection strategies on ammonia-oxidizing bacteria in bench scale drinking water distribution system. *Water Res.*, 37(8): 1805–1817.

Pintar, K., Slawson, R. and Huck, P. (2000). Investigation of conditions and control strategies influencing nitrification in a chloraminated bench-scale distribution study. In: *Proceedings of the American Water Works Association Water Quality and Technology Conference*. American Water Works Association, Denver, Colorado.

Pitts, R.F. (1971). The role of ammonia production and excretion in regulation of acid–base balance. *N. Engl. J. Med.*, 284(1): 32–38.

Pon, R. (2008). Do you really have a free chlorine residual? *Opflow*, 34(6): 24–27.

Price, S.K., Hughes, J.E., Morrison, S.C. and Potgieter, P.D. (1983). Fatal ammonia inhalation—A case report with autopsy findings. *S. Afr. Med. J.*, 64(24): 952–955.

Quail, S. (2008). Blended RO and biological filtration: a novel treatment combination for ammonia removal and copper corrosion control. Dans : *Proceedings of the American Water Works Association Water Quality Technology Conference*, 18 novembre 2008. American Water Works Association, Denver, Colorado.

Randtke, S. (2010). Chapitre 2 : Chemistry of aqueous chlorine. Dans : *White's handbook of chlorination and alternative disinfectants*. 5e édition. John Wiley & Sons, Hoboken, New Jersey.

Rezania, L.W. (2010). Minnesota ammonia study—Phase 1 problem assessment and data collection. Dans : *American Water Works Association Inorganic Contaminants Workshop*, 28 février au 2 mars 2010. American Water Works Association, Denver, Colorado.

Richard, D., Jouany, J.M. and Boudène, C. (1978). Toxicité aiguë par voie aérienne du gaz ammoniac chez le lapin. *C. R. Acad. Hebd. Seances Acad. Sci. Ser. D*, 287(4): 375–378.

Richards, P., Brown, C.L., Houghton, B.J. and Wrong, O.M. (1975). The incorporation of ammonia nitrogen into albumin in man: the effects of diet, uremia and growth hormone. *Clin. Nephrol.*, 3(5): 172–179.

Richardson, S.D. (2005). New disinfection by products issues: Emerging DBPs and alternative routes of exposure. *Global NEST Journal*. Vol. 7, No. 1, pp 43-66.

Rittmann, B.E. and Snoeyink, V.L. (1984). Achieving biologically stable drinking water. *J. Am. Water Works Assoc.*, 76(10): 106–114.

Rogalla, F., Ravarini, P., de Larminat, G. and Couttelle, J. (1990). Large scale biological nitrate and ammonia removal. *Water Environ. J.*, 4(4): 319–328.

Rosenbaum, A.M., Walner, D.L., Dunham, M.E. and Holinger, L.D. (1998). Ammonia capsule ingestion causing upper aerodigestive tract injury. *Otolaryngol. Head Neck Surg.*, 119(6): 678–680.

Salvatore, F., Bocchini, V. and Cimino, F. (1963). Ammonia intoxication and its effects on brain and blood ammonia levels. *Biochem. Pharmacol.*, 12: 1–6.

Saskatchewan Ministry of Environment (2010). Personal communication from S. Ferris, Municipal Branch,,Regina, Saskatchewan, February.

SCC (2011). Directory of accredited product and service certification bodies. Standards Council of Canada, Ottawa, Ontario. Available at: [www.scc.ca/en/programs-services/product-certification/directory-of-accredited-clients](http://www.scc.ca/en/programs-services/product-certification/directory-of-accredited-clients)

Schaerdel, A.D., White, W.J., Lang, C.M., Dvorchik, B.H. and Bohner, K. (1983). Localized and systemic effects of environmental ammonia in rats. *Lab. Anim. Sci.*, 33: 40–45.

Schilling, K.E. (2002). Occurrence and distribution of ammonium in Iowa groundwater. *Water Environ. Res.*, 74(2): 177–186.

Schock, M.R. and Lytle, D.A. (2011). Chapter 20: Internal corrosion and deposition control. In: *Water quality and treatment: a handbook of community water supplies*. 6th edition. McGraw-Hill, New York, New York.

Schulz, D. (2010). Minnesota ammonia study—Phase 2, case studies on impacts and management strategies. In: *American Water Works Association Inorganic Contaminants Workshop*, February 28 to March 2, 2010. American Water Works Association, Denver, Colorado.

Shorney-Darby, H. and Harms, L. (2010). Chapter 4: Chlorination of potable water. In: *White's handbook of chlorination and alternative disinfectants*. 5th edition. John Wiley & Sons, Hoboken, New Jersey.

Siljeg, M., Foglar, L. and Kukucka, M. (2010). The ground water ammonium sorption onto Croatian and Serbian clinoptilolite. *J. Hazard. Mater.*, 178: 572–577.

Silverman, L., Whittenberger, J.L. and Luller, J. (1949) Physiological response of man to ammonia in low concentrations. *J. Ind. Hyg. Toxicol.*, 31(2): 74–78.

Skadsen, J. (1993). Nitrification in distribution system. *J. Am. Water Works Assoc.*, 85(7): 95–103.

Skadsen, J. and Cohen, K.Y. (2006). Chapter 8: Operational and treatment practices to prevent nitrification. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems*. Manual of Water Supply Practices M56. 1st edition. American Water Works Association, Denver, Colorado.

Smith, C. (2006). Chapter 7: Monitoring for nitrification, prevention and control. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems*. Manual of Water Supply Practices M56. 1st edition. American Water Works Association, Denver, Colorado.

Stabenau, J.R., Warren, K.S. and Rall, D.P. (1958). The role of pH gradient in the distribution of ammonia between blood and cerebrospinal fluid, brain and muscle. *J. Clin. Invest.*, 38(2): 373–383.

Stembal, T., Markie, M., Ribicic, N., Briski, F. and Sipos, L. (2005). Removal of ammonia, iron, and manganese from groundwaters of northern Croatia—pilot plant study. *Process Biochem.*, 40(1): 327–335.

Summerskill, W.H.J. and Wolpert, E. (1970). Ammonia metabolism in the gut. *Am. J. Clin. Nutr.*, 23(5): 633–639.

Takagaki, G., Berl, S., Clarke, D.D., Purpura, D.P. and Waelsch, H. (1961). Glutamic acid metabolism in brain and liver during infusion with ammonia labelled with nitrogen-15. *Nature*, 189: 326.

Taylor, J.S. and Wiesner, M. (1999). Chapter 11: Membranes. In: *Water quality and treatment: a handbook of community water supplies*. 5th edition. McGraw-Hill, New York, New York.

- Tekerlekopoulou, A.G. and Vayenas, D.V. (2007). Ammonia, iron and manganese removal from potable water using a trickling filter. *Desalination*, 210(1-3): 225–235.
- Tekerlekopoulou, A.G. and Vayenas, D.V. (2008). Simultaneous biological removal of ammonia, iron and manganese from potable water using trickling filter. *Biochem. Eng. J.*, 39: 215–220.
- Tepper, J.S., Weiss, B. and Wood, R.W. (1985). Alterations in behavior produced by inhaled ozone or ammonia. *Fundam. Appl. Toxicol.*, 5(6 Pt 1): 1110–1118.
- Tian, J., Liang, H., Nan, J., Yang, Y., You, S. and Li, G. (2009). Submerged membrane bioreactor (sMBR) for the treatment of contaminated raw water. *Chem. Eng. J.*, 148(2-3): 296–305.
- Toth, B. (1972). Hydrazine, methylhydrazine and methylhydrazine sulfate carcinogenesis in Swiss mice. Failure of ammonium hydroxide to interfere in the development of tumors. *Int. J. Cancer*, 9(1): 109–118.
- Tsuji, M., Kawano, S., Tsuji, S., Nagano, K., Sasaki, Y., Hayashi, N., Fusamoto, H. and Kamada, T. (1993). Cell kinetics of mucosal atrophy in rat stomach induced by long term administration of ammonia. *Gastroenterology*, 104(3): 796–801.
- Tsuji, M., Kawano, S., Tsuji, S., Takei, Y., Tamura, K., Fusamoto, H. and Kamada, T. (1995). Mechanism for ammonia-induced promotion of gastric carcinogenesis in rats. *Carcinogenesis*, 16(3): 563–566.
- Turan, M. and Celik, M.S. (2003). Regenerability of Turkish clinoptilolite for use in ammonia removal from drinking water. *J. Water SRT – Aqua*, 52(1): 59–66.
- U.S. EPA (1983). Methods for chemical analysis of water and wastes. U.S. Environmental Protection Agency, Washington, DC (EPA 600/4-79/020).
- U.S. EPA (1989). Summary review of health effects associated with ammonia. Health issue assessment. U.S. Environmental Protection Agency, Washington, DC (EPA 600/8-89/052F).
- U.S. EPA (1993). Methods for determination of inorganic substances in environmental samples. U.S. Environmental Protection Agency, Cincinnati, Ohio (EPA 600/R-93/100).
- U.S. EPA (1999). Alternative disinfectants and oxidants: guidance manual. Office of Water, U.S. Environmental Protection Agency, Washington, DC (EPA 815/R-99/014).
- U.S. EPA (2000). Wastewater technology fact sheet. Ammonia stripping. U.S. Environmental Protection Agency, Washington, DC (EPA 832/F-00/019).
- U.S. EPA (2002). Nitrification. Prepared for Standards and Risk Management Division, Office of Groundwater and Drinking Water, U.S. Environmental Protection Agency, Washington, DC.
- Uzövölgyi, E. and Bojan, F. (1980). Possible *in vivo* formation of a carcinogenic substance from diethyl pyrocarbonate and ammonia. *J. Cancer Res. Clin. Oncol.*, 97(2): 205–207.
- Vassileva, P. and Voikova, D. (2009). Investigation on natural and pretreated Bulgarian clinoptilolite for ammonium ions removal from aqueous solutions. *J. Hazard. Mater.*, 170(2-3): 948–953.
- Vikesland, P., Ozekin, K. and Valentine, R. (2001). Monochloramine decay in model and distribution system waters. *Water Res.*, 35(7): 1766–1776.
- Vikesland, P., Love, N., Chandram, K., Fiss, M., Rebodos, R. and Zaklikowski, A. (2006). Seasonal chlorination practices and impacts to chloraminating utilities. American Water Works Research Foundation and American Water Works Association, Denver, Colorado.

- Vissek, W.J., Kolodny, G.M. and Gross, P.R. (1972). Ammonia effects in cultures of normal and transformed 3T3 cells. *J. Cell Physiol.*, 80(3): 373–381.
- Vitti, T.G., Vukmirovich, R. and Gaebler, O.H. (1964). Utilization of ammonia nitrogen, administered by intragastric, intraperitoneal, and subcutaneous routes: effects of growth hormone. *Arch. Biochem. Biophys.*, 106: 475–482.
- Vogelzang, F.P.J., van der Gulden, J.W.J., Preller, L., Tielen, M.J.M., van Shayck, C.P. and Folgering, H. (1997). Bronchial hyperresponsiveness and exposure in pig farmers. *Int. Arch. Occup. Environ. Health*, 70(5): 327–333.
- Vogelzang, F.P.J., van der Gulden, J.W.J., Folgering, H., Heederik, D., Tielen, M.J.M. and van Shayck, C.P. (2000). Longitudinal changes in bronchial responsiveness associated with swine confinement dust exposure. *Chest*, 117(5): 1488–1495.
- Wang, Y., Kmiya, Y. and Okuhara, T. (2007). Removal of low-concentration ammonia in water by ion-exchange using Na-mordenite. *Water Res.*, 41(2): 269–276.
- Warren, K.S. and Schenker, S. (1964). Effect of an inhibitor of glutamine synthesis (methionine sulfoximine) on ammonia toxicity and metabolism. *J. Lab. Clin. Med.*, 64: 442–449.
- Weast, R.C., Astle, M.J. and Beyer, W.H. (eds.) (1988). *CRC handbook of chemistry and physics*. 69<sup>th</sup> edition. CRC Press, Boca Raton, Florida.
- Weatherley, L.R. and Miladinovic, N.D. (2004). Comparison of the ion exchange uptake of ammonium ion onto New Zealand clinoptilolite and mordenite. *Water Res.*, 38(20): 4305–4312.
- White, C., Lytle, D., DeBry, R. and Galloway, A. (2009). Design, evaluation and molecular microbial ecology of a pilot scale biologically active filter for ammonia oxidation. In: *Proceedings of the American Water Works Association Water Quality Technology Conference*, Seattle, WA, November 15–19, 2009. American Water Works Association, Denver, Colorado.
- WHO (2003). Ammonia in drinking water. Background document for development of WHO Guidelines for drinking-water quality. World Health Organization, Geneva (WHO/SDE/WSH/03.04/01).
- Wilczak, A. (2006a). Chapter 2: Nitrification in water and wastewater treatment. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems. Manual of Water Supply Practices M56*. 1st edition. American Water Works Association, Denver, Colorado.
- Wilczak, A. (2006b). Chapter 4: Overview of causes and control of nitrification in water distribution systems. In: *Fundamentals and control of nitrification in chloraminated drinking water distribution systems. Manual of Water Supply Practices M56*. 1st edition. American Water Works Association, Denver, Colorado.
- Wilczak, A., Jacangelo, J., Marcinko, J., Odell, L., Kirmeyer, G. and Wolf, R. (1996). Occurrence of nitrification in chloraminated distribution systems. *J. Am. Water Works Assoc.*, 88(7): 74–85.
- Wolfe, R., Lieu, N., Izaguirre, G. and Means, E. (1990). Ammonia-oxidizing bacteria in a chloraminated distribution system: seasonal occurrence, distribution and disinfection resistance. *Appl. Environ. Microbiol.*, 56(2): 451–462.
- Woolschlager, J., Rittmann, B., Piriou, P. and Schawartz, B. (2001). Using a comprehensive model to identify the major mechanisms of chloramine decay in distribution systems. *Water Sci. Technol. Water Supply*, 1(4): 103–110.
- Xia, W., Zhang, C., Zeng, X., Feng, Y., Weng, J., Lin, X., Zhu, J., Xiong, Z., Xu, J., Cai, Z. and Jia, Z. (2011). Autotrophic growth of nitrifying community in an agricultural soil. *ISME J.*, 5(7): 1226–1236. Available at: [www.nature.com/ismej](http://www.nature.com/ismej)



- Yadav, J.S. and Kaushik, V.K. (1997). Genotoxic effect of ammonia exposure on workers in a fertilizer factory. *Indian J. Exp. Biol.*, 35(5): 487–492.
- Zehr, J.P. and Kudela, R.M. (2011). Nitrogen cycle of the open ocean: from genes to ecosystems. *Annu. Rev. Mar. Sci.*, 3: 197–225.
- Zhang, W. and DiGiano, F.A. (2002). Comparison of bacterial regrowth in distribution systems using free chlorine and chloramine: a statistical study of causative factors. *Water Res.*, 36(6): 1469–1482.
- Zhang, Y., Groiffin, A. and Edwards, M. (2008). Nitrification in premise plumbing: role of phosphate, pH, and pipe corrosion. *Environ. Sci. Technol.*, 42(12): 4280–4284.
- Zhang, Y., Groiffin, A., Rahman, M., Camper, A. and Edwards, M. (2009). Lead contamination of potable water due to nitrification. *Environ. Sci. Technol.*, 43(6): 1890–1895.
- Zhang, Y., Edwards, M., Pinto, A., Love, N., Camper, A., Rahman, M. and Baribeau, H. (2010). Effect of nitrification on corrosion in the distribution system. American Water Works Association Research Foundation and American Water Works Association, Denver, Colorado.
- Zhu, E., Monks, V. and Song, R. (2010). Effectiveness of early application of sodium chlorite in nitrification prevention—A demonstration scale study. In: *Proceedings of the American Water Works Association Water Quality Technology Conference*, November 17, 2010. American Water Works Association, Denver, Colorado.

## Appendix A: List of acronyms

ANSI	American National Standards Institute
AOB	ammonia-oxidizing bacteria
BAC	biological activated carbon
BV	bed volume
bw	body weight
BWRO	brackish water reverse osmosis
DL	detection limit
EBCT	empty bed contact time
EPA	Environmental Protection Agency (United States)
GAC	granular activated carbon
HPC	heterotrophic plate count
IARC	International Agency for Research on Cancer
LC <sub>50</sub>	median lethal concentration
LPRO	low-pressure reverse osmosis
MDL	method detection limit
MGD	million gallons per day
MUL	maximum use limit
NF	nanofiltration
NH <sub>3</sub> -N	ammonia-nitrogen
NOB	nitrite-oxidizing bacteria
NSF	NSF International
RO	reverse osmosis
SCC	Standards Council of Canada
SWRO	saline water reverse osmosis
WHO	World Health Organization

## Appendix B: Analytical methods for the determination of ammonia-nitrogen in drinking water

Methodology	Method ID	Range of application (mg/L as N)	Detection level (mg/L as N)	Interferences / recommended action	Comments
Colorimetry	U.S. EPA 350.1	0.02–2.0	0.01	Ca <sup>2+</sup> , Mn <sup>2+</sup> / use EDTA	Linear up to 0.6 mg/L as N
	SM 4500-NH <sub>3</sub> F (phenate method)	< 0.6		Residual Cl <sub>2</sub> / use dechlorinating agent Elevated turbidity and colour / filter sample High Ca <sup>2+</sup> , Mn <sup>2+</sup> / complex with citrate Turbidity / distil or filter water	
	SM 4500-NH <sub>3</sub> G (automated phenate method)	0.02–2.0		High turbidity or colour / filter sample High Ca <sup>2+</sup> , Mn <sup>2+</sup> / add EDTA and sodium tartrate	Avoid high variability in pH of samples: intensity of colour measured is pH dependent
	SM 4500-NH <sub>3</sub> H (automated flow injection)		0.002	Large and fibrous particles / filter sample	Some interferences may be removed by distillation
	U.S. EPA 350.2 (nesslerization)	0.05–1.0	0.05	Aromatics, amines / distil water Volatile alkaline compounds / boil off at low pH Residual Cl <sub>2</sub> / use dechlorinating agent	Dropped from the Standard Methods to avoid use of mercury
Titrimetry	U.S. EPA 350.2	1.0–25	1.0	Residual Cl <sub>2</sub> / use dechlorinating agent	Sample distillation is required
	SM 4500-NH <sub>3</sub> C	5.0–100		Residual Cl <sub>2</sub> / use dechlorinating agent	Sample distillation is required
Potentiometry: ammonia ion-selective electrode	U.S. EPA 350.3	0.03–1400	0.03	Elevated concentration of dissolved ions, Hg / do not preserve sample with HgCl <sub>2</sub>	Turbidity and colour do not interfere; distillation not required; slow response below 1 mg/L as N
	SM 4500-NH <sub>3</sub> E (using known addition)	> 0.8		Elevated concentration of dissolved ions, Hg / do not preserve sample with HgCl <sub>2</sub>	Can be applied in the presence of high complexing agents