NITRATE and NITRITE

Maximum Acceptable Value for Nitrate (Short-term)
Based on health considerations, the concentration of nitrate (as NO_3) in drinking-water should not exceed 50 mg/L.

Maximum Acceptable Value for Nitrite (Short-term)
Based on health considerations, the short-term concentration of nitrite (as NO_2) in drinking-water should not exceed 3 mg/L.

Maximum Acceptable Value for Nitrate plus Nitrite (Short-term)
The sum of the ratios of the concentrations of each to its Maximum Acceptable Value (short-term) should not exceed 1.

DWSNZ (2008): Maximum Acceptable Value for Nitrite (Long-term and Provisional)
Based on health considerations, the long-term concentration of nitrite (as NO_2) in drinking-water should not exceed 0.2 mg/L. The WHO guideline value for chronic (long-term) effects of nitrite is considered provisional owing to uncertainty surrounding the relevance of the observed adverse health effects for humans and the susceptibility of humans compared with animals.

Note that WHO (2011) no longer includes a long-term (chronic) guideline value for nitrite.

Nitrate and nitrite are included in the plan of work of the rolling revision of the WHO Guidelines for Drinking-water Quality.

The Prescribed Concentration or Value (PCV) for nitrate in England and Wales is 50 mg/L as nitrate. The Prescribed Concentration or Value (PCV) for nitrite in England and Wales is 0.5 mg/L as nitrite at the consumers' taps and 0.1 mg/L at the WTP. See Notes.

The maximum contaminant level or MCL for nitrate (USEPA 2009/2011) is 10 mg/L as N, and 1 mg/L for nitrite as N, or a total of 10 mg/L. The maximum acceptable concentration in Canada is 10 mg/L for nitrate as N, and 1 mg/L for nitrite as N. In cases where nitrite is measured separately from nitrate, the concentration of nitrite should not exceed 3.2 mg/L as NO_2^-.

The Australian Drinking Water Guidelines (NHMRC, NRMMC 2011) state that based on health considerations, the guideline value of 50 mg-NO_3/L (as nitrate) has been set to protect bottle-fed infants under 3 months of age. Up to 100 mg-NO_3/L can be safely consumed by adults, and by children over 3 months of age. Where a water supply has between 50 and 100 mg-NO_3/L nitrate, active measures are required to ensure that those caring for infants are aware of the need to use alternative water sources in making up bottle feeds for babies under 3 months of age. Based on health considerations, the concentration of nitrite in drinking water should not exceed 3 mg-NO_2/L (as nitrite).

Note that 50 mg/L nitrate as NO_3^- is equivalent to 11.3 mg/L as N, 3 mg/L nitrite as NO_2^- is equivalent to 0.9 mg/L as N, and 0.2 mg/L nitrite as NO_2^- is equivalent to 0.06 mg/L as N.

Sources to Drinking-water
1. To Source Waters
Nitrate and nitrite can enter the aquatic environment from the oxidation of vegetable and animal debris and animal excrement.

Nitrate and nitrite can also enter water from agricultural, domestic and industrial discharges. Nitrate is used in chemical fertilisers, oxidising agents in the chemical industry, in the manufacture of glass, enamels for pottery, matches, pickling meat and in the production of explosives. A major source of nitrate is from municipal wastewaters and septic tanks. Nitrite is also used as a corrosion inhibitor in industry, and as a food preservative, especially for curing meats.

2. From the Treatment Processes

The chlorination of raw waters containing significant amounts of ammonia or nitrite may lead to increases in nitrate through their oxidation. As 70% or more of the chlorine consumed during the oxidation of ammonia leads to nitrogen (the gas) production, the increase in nitrate concentrations is likely to be small unless ammonia concentrations are high.

3. From the Distribution System

Nitrite can be formed chemically in distribution pipes by *Nitrosomonas* bacteria during stagnation of nitrate-containing and oxygen-poor drinking-water in galvanised steel pipes, or if chloramination is used to provide a residual disinfectant but its occurrence is almost invariably sporadic. Nitrification in distribution systems can increase nitrite levels, usually by 0.2 – 1.5 mg/L.

Forms and Fate in the Environment

Nitrate and nitrite are naturally occurring ions which make up part of the nitrogen cycle. Nitrate is the oxidised form of combined nitrogen found in natural waters and in dilute aqueous solutions is chemically unreactive. Under anaerobic conditions nitrate may be reduced to nitrite and ammonia. Nitrite is seldom present in surface waters at significant concentrations but may be present in groundwaters. High nitrite concentrations are generally indicative of contamination. Incomplete nitrification of ammonia and denitrification of nitrate result in the biochemical production of nitrite which is generally present only under anaerobic conditions.

Typical Concentrations in Drinking-water

Nitrate

Nitrate was routinely measured in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme. Of 1908 samples analysed between 1983 and 1989, 14 samples (0.7%) contained concentrations equal to or exceeding the 1984 MAV of 10 mg/L (N).

The P2 Chemical Determinand Identification Programme, sampled from 673 zones, found nitrate concentrations to range from “not detectable” (nd) to 30 mg/L as NO_3-N, with the median concentration being 0.2 mg/L (Limit of detection = 0.1 mg NO_3-N/L). The Priority 2 Identification Programme found 6 distribution zones supplying drinking-water to a total of 1017 people with nitrate at greater than the MAV (ESR 2001).

In 2012 the Canterbury District Health Board stated that 33 of 289 wells tested in Canterbury exceeded the MAV for nitrate, the majority being around Ashburton.

26,177 water utilities in the US reported detecting nitrate in tap water since 2004, according to EWG's analysis of water quality data supplied by state water agencies, with the highest concentration being 30 mg/L as N.

Nitrite

Nitrite was not measured routinely in New Zealand drinking-water supplies as part of the Department of Health three yearly surveillance programme.

The P2 Chemical Determinand Identification Programme, sampled from 227 zones, found nitrite concentrations to range from "not detectable" (nd) to 0.088 mg/L, with the median concentration being "nd" (Limit of detection = 0.005 NO_2^- -N mg/L). The Priority 2 Identification Programme found no distribution zones supplying drinking-water with nitrite at greater than the MAV (ESR 2001).

2,719 water utilities in the US reported detecting nitrite in tap water since 2004, according to EWG's analysis of water quality data supplied by state water agencies, with the highest concentration being 2.78 mg/L as N.

Removal Methods

Nitrate

Nitrate is not removed from water by classical methods of treatment. Ion exchange systems have been developed for removing nitrate, but dilution with water of lower nitrate concentration from another source, where one is available, is commonly used.

Nitrite

Treatment of the water with an oxidising agent such as chlorine will convert the nitrite to nitrate. The nitrate can then be treated as explained for nitrate. The USEPA Maximum Concentration Level (MCL) for nitrite indicates that the concentration at which it might be of concern is ten times less than the MCL for nitrate. The oxidation of high nitrite levels to nitrate therefore will not create an unacceptably high nitrate concentration in the water, unless the nitrate level is already high, or the nitrite level is extremely high.

Analytical Methods

Nitrate

Referee Method

Cadmium Reduction Method (APHA 4500-NO_3-E).

Some Alternative Methods

1. Ion Chromatography Method (APHA 4110B; USEPA 300.1).

2. Nitrate Electrode Method (APHA 4500-NO_3 D).

Nitrite

Referee Method

Colorimetric Method (APHA 4500-NO_2 B).

Some Alternative Methods

1. Ion Chromatography Method (APHA 4110 B; USEPA 300.1).

Health Considerations

For nitrate, the main sources of exposure are vegetables, especially leafy vegetables. Other food sources include baked and processed cereal products and cured meat. For an average adult consumer who lives in an area with low drinking-water contamination, total exposure to nitrate from food and water is estimated to be about 60 to 90 mg per person per day, of which at least 90% is from food. For high consumers of vegetables, the intake of nitrate may reach 200 mg per person per day. Similar intakes could result from high consumption of water contaminated with more than 50 mg/L nitrate (as NO_3^-).

For nitrite, the main source of exogenous human exposure is also food. Important sources include cereal products, vegetables and cured meat. Over the last 30 years, the relative contribution of cured meat to dietary exposure to nitrite for an average consumer has decreased from about 40% to about 20%. For high consumers of cured meat, the relative contribution may have reached 90%. The total intake of exogenous nitrite is estimated to be about 0.75 to 2.2 mg per day for an adult with an average food consumption pattern.

Ingested nitrate is absorbed readily and completely from the upper small intestine. Nitrite may be absorbed directly from the stomach as well as from the small intestine. When nitrate levels in drinking-water exceed 50 mg/L as NO_3^-, drinking-water may become the major source of total nitrate intake, especially for bottle-fed infants.

The toxicity of nitrate in humans is thought to be due solely to its reduction to nitrite. Bacteria are responsible for most of the conversion of nitrate to nitrite in the gastrointestinal system. Consequently, the risk of methemoglobinemia from ingestion of nitrate depends not only on the dose of nitrate, but also on the number and type of enteric bacteria. In healthy adults, available data suggest about 5% of a dose of nitrate is reduced to nitrite by bacteria in the mouth. Conversion of nitrate to nitrite may also occur in the stomach if the pH of the gastric fluid is sufficiently high (above pH 5) to permit bacterial growth. This is of concern in adults with diseases such as achlorhydria or atrophic gastritis. It is commonly accepted that infants younger than 3 months may be highly susceptible to gastric bacterial nitrate reduction, as their stomach pH is generally higher than in adults.

In Hungary in 1975 to 1977, 190 cases of methaemoglobinaemia were reported, 94% in infants less than three months of age. The nitrate level in drinking water was more than 100 mg/L in 92% of cases and between 40 and 100 mg/L in the remaining 8%. In 1982, 96 cases of methaemoglobinaemia were reported. All cases were associated with privately dug wells, and 92% of the patients were three months of age or younger. Nitrate levels in drinking water were above 100 mg/L in 93% of cases and between 40 and 100 mg/L in the remaining 7%. WHO (1985).

For over 40 years, there has existed a widespread belief that nitrate in drinking water is a primary cause of infantile methemoglobinemia. Hunter Comly originally proposed this theory in 1945 in a report in the Journal of the American Medical Association after treating several infantile methemoglobinemia victims. Comly proposed that because nitrite is known to react directly with hemoglobin to form methemoglobin, nitrate from drinking water must be converted to nitrite within the gastrointestinal tract of infants. Because many infants did not appear susceptible to methemoglobinemia from nitrate-contaminated water, Comly suggested that the nitrate-to-nitrite conversion might only occur in the presence of a bacterial infection of the upper gastrointestinal tract, where such reactions could occur before nitrate is absorbed. The nitrate-derived nitrite could then react with haemoglobin to form methemoglobin and, in sufficient quantities, lead to the cyanosis of methemoglobinemia. This theory was reinforced by the fact that cyanosis typically subsided once an infant was switched to an uncontaminated water supply. Comly's hypothesis became widely accepted as further research revealed a consistent pattern of elevated well water nitrate levels in infantile methemoglobinemia cases. Limiting infant exposure to nitrate was thus decided to be the most prudent approach to protecting infant health, and a committee from the American Public Health Association conducted a nationwide survey to determine a safe level of nitrate in water. A total of 278 cases with 39 deaths were compiled. The results showed that methemoglobinemia incidence correlated with increasing nitrate levels. Because no infantile methemoglobinemia cases were observed with concentrations <10 ppm nitrate-nitrogen, the United States and the World Health Organization established a maximum contaminant level (MCL) of 10 ppm nitrate-N for nitrate in drinking water. Over the last 20 years, however, a more complex picture of infantile

methemoglobinemia causation has emerged which indicates that current limits on drinking water nitrate may be unnecessarily strict. It is now well established that diarrhoeal illness and some gastrointestinal disturbances, typically accompanied by diarrhoea and/or vomiting, can lead to methemoglobinemia in young infants without exposure to high-nitrate drinking water or exposure to abnormal levels of nitrate through food. There are literally dozens of reported infantile methemoglobinemia cases associated with diarrhoea without exposure to nitrate-contaminated water. Because diarrhoea was a prominent symptom in the majority of drinking water linked methemoglobinemia cases, the evidence suggests that diarrhoea and/or gastrointestinal infection/inflammation, not ingested nitrate, are the principle causative factors in infantile methemoglobinemia. A survey in Germany found that 53% of 306 infantile methemoglobinemia cases reported diarrhoea. Contrary to some reports, diarrhoea and vomiting are not symptoms that typically accompany cyanosis, methemoglobinemia due to oxidant drug exposure, or genetic abnormalities in haemoglobin. Avery (1999), and discussed in WHO (2011).

Methaemoglobinaemia in infants appears to be associated with simultaneous diarrhoeal disease. Authorities should therefore be all the more vigilant that water to be used for bottle-fed infants is microbiologically safe when nitrate is present at concentrations near the guideline value or in the presence of endemic infantile diarrhoea. Water should not be used for bottle-fed infants if the concentration of nitrate is above 100 mg/L as NO_3 but can be used if the concentration is between 50 and 100 mg/L if the water is microbiologically safe and there is increased vigilance by medical authorities (WHO 2011).

The primary health concern regarding nitrate and nitrite is the formation of methaemoglobinaemia, so-called blue-baby syndrome. Nitrate is reduced to nitrite in the stomach of infants, and nitrite is able to oxidise haemoglobin (Hb) to methaemoglobin (metHb), which is then unable to transport oxygen around the body. The reduced oxygen transport becomes clinically manifest when metHb concentrations reach 10% or more of normal Hb concentrations; the condition, called methaemoglobinaemia, causes cyanosis and, at higher concentrations, asphyxia. The normal metHb level in infants under 3 months of age is less than 3%. Other susceptible groups include pregnant women and people with a deficiency of glucose-6-phosphate dehydrogenase or methaemoglobin reductase. Methaemoglobinaemia in infants also appears to be associated with simultaneous exposure to microbial contaminants, e.g. Addison and Benjamin (2004).

Walton (1951) described a survey performed by the American Public Health Association to identify clinical cases of infantile methemoglobinemia that were associated with ingestion of nitrate-contaminated water. A total of 278 cases of methemoglobinemia were reported. Of 214 cases for which data were available on nitrate levels in water, none occurred in infants consuming water containing <10 mg nitrate-nitrogen/L (1.6 mg nitrate-nitrogen/kg/day). There were 5 cases (2%) in infants exposed to 11 - 20 mg nitrate-nitrogen/L (1.8 - 3.2 mg/kg/day), 36 cases (17%) in infants exposed to 21 - 50 mg/L (3.4 - 8.0 mg/kg/day), and 173 (81%) in infants exposed to >50 mg/L (>8 mg/kg/day). Based on these studies of nitrate contamination and occurrence of methemoglobin the USEPA set the maximum contaminant level or reference dose for oral intake of 10 mg/L nitrate as N (USEPA 1987, revised 1991).

Nitrate is not mutagenic in bacteria and mammalian cells *in vitro*. Chromosomal aberrations were observed in the bone marrow of rats after oral nitrate uptake, but this could have been due to exogenous N-nitroso compound formation. Nitrite is mutagenic, causing morphological transformations in in vitro systems.

IARC (2005) stated that "Ingested nitrate or nitrite under conditions that result in endogenous nitrosation is *probably carcinogenic to humans (Group 2A)*. The underlying mechanism is

endogenous nitrosation, which in the case of nitrate must be preceded by reduction to nitrite. Nitrate and nitrite are interconvertible *in vivo*. Nitrosating agents that arise from nitrite under acidic gastric conditions react readily with nitrosatable compounds, especially secondary amines and alkyl amides, to generate *N*-nitroso compounds. Many *N*-nitroso compounds are carcinogenic." However, the weight of evidence indicates that there is unlikely to be a causal association between gastric cancer and nitrate in drinking-water.

The reference dose or RfD (USEPA 1991/2009/2011) for nitrate as N is 1.6 mg/kg/d, and for nitrite as N it is 0.16 mg/kg/d (USEPA 1997/2009/2011).

As at October 2015 ATSDR (http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf) quotes a minimal risk level (MRL) for nitrate of:

4 mg/kg/d for acute-duration oral exposure (1 to 14 days)
4 mg/kg/day for intermediate-duration oral exposure (15 – 364 days)
4 mg/kg/day for chronic-duration oral exposure (>364 days).

As at October 2015 ATSDR (http://www.atsdr.cdc.gov/mrls/pdfs/atsdr_mrls.pdf) quotes a minimal risk level (MRL) for nitrite of:

0.14 mg/kg/d for acute-duration oral exposure (1 to 14 days)
0.1 mg/kg/day for intermediate-duration oral exposure (15 – 364 days)
0.1 mg/kg/day for chronic-duration oral exposure (>364 days).

The livestock guideline value for nitrite (as NO_2^-) is 30 mg/L. Nitrate (as NO_3^-) concentrations less than 400 mg/L in livestock drinking water should not be harmful to animal health; stock may tolerate higher nitrate concentrations in drinking water provided nitrate concentrations in feed are not high. Water containing more than 1500 mg/L nitrate (as NO_3^-) is likely to be toxic to animals and should be avoided (ANZECC/ARMCANZ 2000). These guidelines were to have been updated in 2012.

Derivation of Maximum Acceptable Values

Nitrate (short-term)

The MAV of 50 mg/L (as NO_3^-) is to protect against methaemoglobinaemia in bottle-fed infants (short-term exposure). In epidemiological studies, methaemoglobinaemia was not reported in infants in areas where drinking-water consistently contained less than 50 mg of nitrate per litre.

The epidemiological evidence for an association between dietary nitrate and cancer is insufficient, and the MAV for nitrate in drinking-water is established solely to prevent methaemoglobinaemia, which depends upon the conversion of nitrate to nitrite. Although bottle-fed babies are the most susceptible, occasional cases have been reported in some adult populations.

Nitrite (short-term)

The short-term MAV of 3 mg/L (as NO_2^-) is to protect against methaemoglobinaemia in bottle-fed infants. The WHO (2011) guideline value for nitrite of 3 mg/L as nitrite (or 0.9 mg/L if reported as nitrite-nitrogen) is based on human data showing that doses of nitrite that cause methaemoglobinaemia in infants range from 0.4 mg/kg body weight to more than 200 mg/kg body weight. By applying the lowest level of the range (0.4 mg/kg body weight), a body weight of 5 kg for an infant and a drinking-water consumption of 0.75 litre, a guideline value of 3 mg/L (rounded figure) can be derived.

Earlier, WHO had stated that animal studies were inappropriate to establish a firm No-Observable-Adverse-Effect Level (NOAEL) for methaemoglobinaemia in rats. Therefore, a pragmatic approach

was followed, accepting a relative potency for nitrite and nitrate with respect to methaemoglobin formation of 10:1 (on a molar basis), and a provisional MAV of of 3 mg/L had been adopted for nitrite.

Nitrite (long-term)

The long-term MAV had been based on the next paragraph. WHO (2011) now states: "However, owing to the uncertainty surrounding the susceptibility of humans compared with experimental animals, this value which was considered provisional has now been suspended and is being subjected to review in light of evidence on the differences in nitrite metabolism between laboratory rodents and humans."

The 0.2 mg/L (as NO_2^-) MAV in the 2008 DWSNZ for long-term exposure for chronic effects of nitrite was considered provisional owing to uncertainty surrounding the relevance of the observed adverse health effects for humans and the susceptibility of humans compared with animals. The occurrence of nitrite in the distribution system as a consequence of chloramine use will be intermittent, and average exposures over time should not exceed the provisional MAV. The nitrite MAV (long-term exposure) is based on allocation to drinking-water of 10% of JECFA ADI of 0.06 mg/kg of body weight per day, based on nitrite-induced morphological changes in the adrenals, heart and lungs in laboratory animal studies.

Nitrate:Nitrite ratio

Because of the possibility of simultaneous occurrence of nitrite and nitrate in drinking-water, the sum of the ratio of the concentration of each to their short-term MAVs, as shown in the following formula, should not exceed 1:

$$\frac{C(NO_2^-)}{MAV(NO_2^-)} + \frac{C(NO_3^-)}{MAV(NO_3^-)} \leq 1$$

where C = concentration, and MAV = Maximum Acceptable Value

Note related to short-term MAVs.

The short-term MAVs for nitrite and nitrate have been established to protect the health of infants, particularly those that are bottle-fed. Community water suppliers providing drinking-water that exceeds the short-term MAVs will need to find a procedure for advising parents of new-born babies. The WHO (2007) states that in areas where household wells are common, health authorities may wish to take a number of steps to ensure that nitrate contamination is not or does not become a problem. Such steps could include targeting mothers, particularly expectant mothers, with appropriate information about water safety, assisting with visual inspection of wells to determine whether a problem may exist, providing testing facilities where a problem is suspected, providing guidance on disinfecting water or where nitrate levels are particularly high, providing bottled water from safe sources or providing advice as to where such water can be obtained.

The MAV for nitrate in the 1995 and 2000 DWSNZ was 50 mg/L as NO_3^-, and the MAV for nitrite was 3 mg/L as NO_2^-. The 1995 datasheet stated:

> The epidemiological evidence for an association between dietary nitrate and cancer is insufficient, and the MAV for nitrate in drinking-water is established solely to prevent methaemoglobinaemia, which depends on the conversion of nitrate to nitrite. Although bottle-fed babies are the most susceptible, occasional cases have been reported in some adult populations.

As a result of recent evidence of the presence of nitrite in some water supplies, it was concluded that a MAV for nitrite should be proposed. However, the animal studies were inappropriate to establish a firm NOAEL for methaemoglobinaemia in rats. Therefore a pragmatic approach was followed, accepting a relative potency for nitrate and nitrite with respect to methaemoglobin formation of 10:1 (on a molar basis), and a PMAV for nitrite of 3 mg/L is proposed. (The paragraph and formula related to the ratios of each appeared in the 1995 Guidelines as well.)

The Minnesota Department of Health (MDH) has developed health-based rules and guidance to evaluate potential human health risks from exposures to chemicals in groundwater. The chronic health risk limit (exposure greater than 10% of a lifetime) for nitrate is 10 mg/L as N.

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