Health hazards of nitrate in drinking water

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Abstract

This paper is concerned with the health effects of nitrate in drinking water and was compiled from the literature with the purpose to inform and raise interest in the subject. Drinking water containing nitrate-nitrogen in excess of 10 mg/ℓ can cause a (sometimes fatal) blood disorder called methemoglobinemia in infants under the age of six months. Consumption of nitrate has no apparent short-term effects on adults. Nitrate *per se* is not toxic, but is the precursor of nitrite which is produced through microbial reduction of nitrate in the intestine or in food preparations and which causes methemoglobinemia. Children exposed to excessive nitrate in their diet can have slightly retarded bodily growth and slower reflexes. The trouble is mainly confined to infants who are artificially fed with milk feeds made with water containing elevated nitrate levels. The exposure of the South African population to nitrates in drinking water is unknown, as are possible effects of it.

Introduction

Drinking water containing nitrate in excess of 10 mg/ℓ (as N) can cause a (sometimes fatal) blood disorder called methemoglobinemia in infants under the age of six months, especially those under three months. The exposure of the South African population to nitrates in drinking water is unknown as are the possible effects of it.

This literature review is concerned with the health effects of nitrate in drinking water and was compiled with the purpose to inform and to raise interest in this issue.

Sources of nitrates in drinking water

In France most of the high nitrate concentrations in water are found in ground water. Also, most of the wells affected are in highly developed agricultural areas (French Ministry of Health, 1983). The origin of nitrate in ground water can usually be traced to contamination by percolating water carrying nitrate from sources such as decaying plant or animal material, agricultural fertilisers, domestic sewage, areas of high density animal confinement or geological formations containing soluble nitrogen compounds (Adam, 1980).

It was found in the Netherlands that the relative contribution of urban, industrial and agricultural sources to nitrate concentrations in river water is 40% from urban sewage effluent and the rest from industrial and agricultural sources. Furthermore, it was estimated that of the total nitrate concentrations entering water sources from agricultural land, 20% comes from fertilisers, 15% from organic manures and 65% from mineralisation of soil organic matter (Wild, 1977). The primary source of nitrate in the environment is through the very familiar nitrogen cycle where, as a result of electrical discharges in the atmosphere, nitrogen and oxygen combine to form nitrate which is carried by rain to the soil for plant protein production. Herbivorous animals change plant protein to animal protein. Death of plants and animals and animal wastes produces organic matter on which bacteria act to release ammonia, nitrite and nitrate. Nitrogen gas re-enters the atmosphere mainly through the reduction of nitrites. Certain bacteria and algae also fix nitrogen from the atmosphere to produce plant protein.

The means by which nitrates find their way to water supplies were summarised by the International Standing Committee on Water Quality and Treatment (1972) and include the following:

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Surface waters

Nitrate concentrations in surface waters are influenced by:

- domestic sewage effluent and animal effluent from farms;
- discharges of domestic and industrial waste water (septic tanks, food processing wastes, refuse dumps);
- runoff and seepage water from the land containing the nitrified products of decayed vegetation and animal matter (agricultural and urban);
- excess material from oxidation of ammonia in agricultural fertilisers and from nitrates in these materials being washed into surface sources;
- the fixation of atmospheric nitrogen entering surface water from rain or washings from the land; and
- nitrogen fixation by leguminous plants such as peas and the nitrate so formed being washed into surface water.

Ground waters

The main sources of nitrates in ground waters are:

- seepage of water containing the nitrified products of decayed vegetation and animal matter;
- discharges of domestic and farm effluent into the aquifer;
- seepage of nitrification products of special crops grown in the catchment area, e.g. pea farming;
- excess of nitrogenous agricultural fertilisers applied to farm land percolating from the surface into the aquifer; and
- broad irrigation of sewage sludge and effluent on land and its consequent seepage into the ground.

Nitrate concentrations in surface waters are usually below 5 mgN/ℓ and much higher concentrations are sometimes found in ground water (Fraser *et al.*, 1980). For example, a survey of 2 000 wells in Canada showed that 31,6% of wells examined contained more than 20 mgN/ℓ nitrate (World Health Organisation, 1972).

There has been a gradual increase in nitrate levels during the past 20 years in many surface and underground water resources in England and Wales (Royal Commission on Environmental Pollution, 1979). The increasing use of nitrogenous fertilisers is considered to be a contributory factor (40 years ago inorganic fertilisers represented only 2,2% of the total nitrogen available annually, but by 1970 to 1972 the contribution from this source had increased tenfold). Changes in land use, particularly the conversion of pasture to arable land and the increased recycling of sewage effluent in low-land rivers, are also contributing to the rising levels of water-borne nitrate (Young and Gray, 1978). Similarly, in the

coastal aquifer of Israel, the source of some 20% of the country's water supply, observations made at hundreds of deep wells over the past 15 years indicate that nitrate (as NO_3) concentrations have increased at the rate of about 1 to 2 mg/ℓ.a (Soliternik, 1984). In certain parts of South Africa and of Namibia, ground waters also contain high levels of nitrate (as N), e.g. the Springbok Flats (Adam, 1980). A survey done in France showed that out of a population of 53 million, 280 000 have a water supply which exceeds 100 mg/ℓ of nitrate (as NO_3) at least once a year (French Ministry of Health, 1983). The average concentration of nitrate (as NO_3) in ground water in Denmark increased from 3,0 mg/ℓ to 13,3 mg/ℓ over the period 1940 to 1983 (Moller *et al.*, 1989).

Extensive data on nitrate (as NO_3) levels for different water drainage regions in South Africa are available. Levels in South Africa vary between less than 1 mg/ℓ and 40 mg/ℓ. The average nitrate level (as NO_3) for 230 drainage regions throughout South Africa falls between 2 and 5 mg/ℓ (Department of Water Affaris, 1990).

The recommended maximum allowable concentration for nitrates in drinking water has been a controversial issue for many years. The water quality criteria and standards for nitrate as given by the World Health Organisation (WHO) will be discussed under that heading in this paper.

Exposure of humans to nitrates

Man's major exposure to nitrates comes primarily from vegetables or water supplies that are high in nitrate content or from nitrates used as additives in the meat curing process (Wolff and Wasserman, 1972; Fan *et al.*, 1987).

Nitrates are natural constituents of plants. They are present in large quantities in many vegetables, but they occur in only minor amounts in fruit. Spinach, beets, radishes, eggplant, celery, lettuce, collards and turnip greens are among the vegetables that generally contain very high nitrate concentrations (Wolff and Wasserman, 1972).

The most important factors which favor large nitrate accumulation in vegetables according to Adam (1980) include:

- a nitrate-rich environment such as may be caused by high levels of fertilisation;
- species that are prone to accumulate nitrate;
- plant nutrient deficiencies;
- conditions of reduced light intensity during maturation;
- lack of water; and
- plant damage from chemical treatments.

Another important means of exposing humans to nitrates is through consuming cured meat products (Wolff and Wasserman, 1972). Nitrate and nitrite have been used since ancient times to preserve and cure meat and fish. Nitrite is added in large amounts to cured meat (up to 150 mg/kg) (Fraser *et al.*, 1980).

Exposure to nitrates through drinking water with high nitrate levels is a cause for concern. This nitrate comes from many sources as discussed earlier in this report. Health agencies have issued reports that, for water to be safe for domestic use, the nitrate concentration should not exceed 10 mgN/ℓ (equivalent to 45 mg/ℓ as NO_3) (Fraser *et al.*, 1980; Fan *et al.*, 1987). Numerous examples may be found in which the nitrate content of water supplies is in excess of this amount. This situation presents some hazard of potential toxicity (Wolff and Wasserman, 1972; Fan *et al.*, 1987).

In the quantities normally occurring in food and water, nitrates become toxic only under conditions in which they are or may be reduced to nitrites. Otherwise, at reasonable concentrations, nitrate ions are rapidly excreted in the urine (Wolff and Wasserman, 1972). But since nitrate is excreted by man in urine at the rate of say a gram or more a day (derived from food or water), infections of the bladder could lead to formation of nitrite and nitrosamines in the bladder by bacterial reduction (Lijinsky, 1976). The significance of exposure of the bladder to nitrite and its products in this way is not known at this time.

Exposure to nitrate and nitrate means that indirect exposure to nitrosamines which are potent carcinogens, is possible (Magee and Barnes, 1967). A well-known reaction of nitrites is that they form a variety of products (including nitrosamines) with amines in the presence of acid, depending on the nature of the amine (Lijinsky, 1976). Since high concentrations of nitrosamines have not been found in the environment, attention has focused on the formation of nitroso components in the human stomach, where conditions are favorable for these reactions. Formation of nitroso compounds inside the infected bladder is important but less common as the pH is not the optimum for nitrosation. N-nitroso compounds can also act transplacentally, can be freely distributed in the animal body, and could be present in the milk and urine of animals or people exposed to nitroso-compounds from within or without. This could be a hazard to others, e.g. for children through milk from mothers and cows (Lijinsky, 1976).

There is considerable variation between countries both in mean daily intake of nitrates and in relative contributions from different sources. In the United States, for example, the estimated mean daily intake of nitrate (as NO_3) is about 100 mg, 80% of which comes from vegetables and less than 10% from drinking water (Ross and Desforges, 1959). Nitrate intake is much high in Japan, the estimated mean daily intake being about 300 mg (as NO_3), most of which is derived from vegetables and very little from water (Fraser *et al.*, 1980). In Britain, the mean daily intake of nitrate (as NO_3) is 60 to 80 mg, of which vegetables contribute more than 50% of the total (Fraser *et al.*, 1980). It was demonstrated in a study in Denmark that drinking water may be the major source of total nitrate intake. The average Danish consumer is exposed to 90 mg/ℓ nitrate (as NO_3) per day (Moller *et al.*, 1989).

As highlighted in the previous section, man can be exposed to nitrates (and indirectly to nitrites and nitrosamines) through two major sources, i.e. food and water. As a result of the different concentrations of nitrates in foodstuff and the influence of external factors on these concentrations, together with the variations in quantities consumed, it is extremely difficult to calculate the actual nitrate intake for specific individuals. Comparisons between populations using water with high or low nitrate (specified as N or NO_3) concentrations (which can be accurately determined) could, however, be made and values could be used in epidemiological studies.

Health effects of nitrate in drinking water

Nitrate *per se* is considered to be relatively non-toxic and is readily excreted by the kidneys. The secondary products of it, namely nitrite and nitrosamines, however, present a health hazard to humans. Nitrites are known to cause methemoglobinemia in infants, while nitrosamines are carcinogenic and may play a role in the induction of certain gastrointestinal cancers (Hill *et al.*, 1973, Fan *et al.*, 1987).

Methemoglobinemia

Until 1945, nitrate in water was not considered to be a hazard,

but in that year Comly showed that a form of infantile cyanosis was due to the conversion of hemoglobin (Hb) to methemoglobin (MetHb) in the blood (Wild, 1977; International Standing Committee on Water Quality and Treatment, 1972; Wolff and Wasserman, 1972; Shuval and Gruener, 1977). The association between consumption of drinking water high in nitrate concentration and infant MetHb was then identified.

Methemoglobinemia is a condition resulting from the conversion of Hb, the oxygen carrier of mammalian blood, to MetHb which is unable to transport oxygen. Several chemicals such as nitrites, perchlorates, sulphonamides, aniline dyes, bismuth and others (Adam, 1980) can cause conversion of Hb to MetHb, in which the iron is in the ferric (Fe^{3+}) state, rendering the molecule unable to bind oxygen. The exact mechanism of this conversion is not known (Adam, 1980).

It is accepted by many workers that, under normal circumstances, less than 2% of the total Hb exists as MetHb (Shuval and Gruener, 1977). No external signs or symptoms are generally noted under 5% MetHb, whereas the first signs of cyanosis can be seen between 5% and 10% (Knotek and Schmidt, 1964). Hypoxic signs and symptoms may develop at levels above 20%, while death results at levels of 50% or higher (Shuval and Gruener, 1977). From 1945 to 1970 approximately 2 000 cases of infant methemoglobinemia were reported internationally. Fatality rates were 8% on average (Fraser and Chilvers, 1981).

The presence of high concentrations of nitrates in water is the principle determinant for the occurrence of MetHb in infants, albeit other factors are also important in the pathogenesis of the disease, some of which may also explain why nitrate poisoning is limited to infants. These include (Ross and Desforges, 1959):

- Age (more prevalent in infants below one year of age).
- Presence of bacteria (microorganisms isolated from the mouth and gastro-intestinal tract are capable of reducing nitrate to nitrite and grow in media of pH 5 to 7).
- Gastric acidity (examinations of gastric juices of infants who developed appreciable levels of MetHb revealed that the pH was usually higher than 4).
- Gastro-intestinal disturbances (members of the family Enterobacteriaceae are able to reduce nitrate to nitrite. Such organisms can gain access to the upper intestine during gastro-intestinal disturbances, but their ability to become established in the stomach is dependent on pH. In the absence of nitrate-reducing bacteria in the stomach or upper intestine, most of the nitrate ingested is probably absorbed as nitrate before reaching the colon in which the nitrate-reducing bacteria are normally found).
- Type of powdered milk product used (studies in Czechoslovakia have indicated that the use of certain types of milk preparations has been suspected as the main cause for the development of methemoglobinemia in areas with high nitrate levels in water. They reported on cases of methemoglobinemia in infants due to feeding with various brands of regular powdered milk which contained spores of *B. subtilis,* a nitrate-reducing bacterium. Acidified milk powders which are often prepared by fermentation with lactic did not cause any disease).
- High fluid intake (infants may ingest a bigger average nitrate per gram of Hb than an adult due to their higher fluid intake per unit of body weight).
- Effects of nutrition (food composition can also affect the severity of the illness when food rich in nitrates (e.g. spinach, rhubarb etc.) is consumed. On the other hand, certain nutrients such as vitamin C can cure or prevent methemoglobinemia).
- Foetal hemoglobin (hemoglobin-F is oxidised more readily to MetHb. The fact that the blood of new-born babies consists of 80% haemoglobin-F, might explain their increased tendency to develop methemoglobinemia).
- MetHb reduction (MetHb reduction velocity in the presence of lactate or glucose is lower in cord-erythrocytes than in adult blood).

Since so many variables are involved in causing infant methemoglobinemia, it is difficult to specify tolerance levels to nitrate in drinking water. The individual variations in the effect of nitrate on infants using the same water supply also complicate the whole issue. Factors such as variation in dose which a specific infant eventually receives e.g. amount of fluid intake, which may explain these individual differences, should be kept in mind when studying a group of children.

Other toxic effects

Shuval and Gruener (1977) conclude from their studies that nitrates and nitrites may be more toxic than is generally recognised. Laboratory experiments in rats showed that transplacental passage of nitrites can occur, causing raised MetHb levels in the foetus and impaired growth. Exposure of mice to nitrites in drinking water caused behavioral effects such as lowered motor activity and increased aggression. Rats chronically exposed to nitrite showed thinning and ballooning of cardiac blood vessels. Nitrites were used in these studies rather than nitrate, as the animal model does not allow for nitrate to nitrite reduction in the gut.

Some evidence that nitrates may directly affect the central nervous system (CNS) in humans has been published (Petukhof and Ivanov, 1970). The slowing of conditioned motor reflexes in response to auditory and visual stimuli was documented in 39 Russian children whose drinking water contained 105 mg/ℓ nitrate (not specified as N or NO_3). Their reflexes were compared with those of a group of children whose drinking water contained 8 mg/ℓ nitrate. The concentration of MetHb did not exceed normal limits in the schoolchildren who drank low nitrate-containing water, whereas the children exposed to high nitrate-containing water, had an average of 5,3% MetHb in their blood. Methemoglobinemia in which there is less than about 10% of MetHb has been generally regarded as of no clinical significance. This report of a measureable effect on the CNS with only a 5% reduction in oxygen-carrying capcity (Petukhof and Ivanov, 1970) warrants further study.

The relationship between maternal exposure to nitrates in drinking water and the risk of delivering an infant with CNS malformation was examined in Canada. This study found no significant relationship between water nitrate levels (as NO_3) and risk of CNS defects, but found a positive increase in risk with exposure to nitrates from private well-water resources (Arbuckle *et al.,* 1988).

Some workers have suggested that chronic exposure to high levels of nitrate in drinking water may have adverse effects on the cardiovascular system. A state-wide study of municipal water supplies in Colorado in 1960 suggested that the significantly higher hypertension risk in the eastern plains might be due to higher nitrate concentrations in drinking water (Morton, 1971). This finding was further investigated and it was reported that an earlier onset of hypertension exists among residents of communites exposed to nitrate levels of 19 to 123 mg/ℓ (as NO_3), compared with communities exposed to nitrate-free drinking water (Fraser and Chilvers, 1981).

Cancer

The carcinogenic property of N-nitroso compounds has been recognised since 1967 and up to now 75% of the 120 N-nitroso compounds have been shown to be carcinogenic to animals (Gilli *et al.*, 1984).

As discussed earlier, nitrates and secondary amines and amides are the precursors of nitrosamines. The process by which nitrates seem to develop their carcinogenic effects is associated with transformations and interactions which they undergo *in vivo*. The nitrates, in fact, when introduced by foodstuffs and drinking water, are rapidly absorbed in the superior gastro-intestinal tract. They enter the bloodstream and are partially excreted by urine and partially by salivary glands into the oral cavity, where some microorganisms reduce a part of nitrate to nitrite, upon which both return to the stomach by salivary flow (Gilli *et al.*, 1984).

In animals, the N-nitrosamides and N-nitrosureas are proximate carcinogens acting directly at the site of application. In contrast, the N-nitrosamines require activation and are organotropic tending to produce tumors distal to their site of application. The site at which a tumor is formed following treatment of an animal with an N-nitrosamine depends both on the nature of the nitrosamine and on the animal species (Magee and Barnes, 1967). For example, in the rat, dimethylnitrosamine causes renal cancer, dibutylnitrosamine bladder cancer, N-nitrosopiperidine liver cancer and N-nitrosomorpholine lung cancer. N-nitrosopyrrolidine causes cancer of the liver in the rat, but of the lung in the hamster. Thus, in attempting to identify the target organ of a specific nitrosamine it is not even possible to extrapolate results from rodent to rodent, much less from rodent to man (Fraser *et al.*, 1980). As there are no appropriate animal models available that are relevant to the human situation, it is difficult to assess the carcinogenic action of N-nitroso compounds in humans.

As the amount of N-nitroso compounds formed in humans depends in part on the amount of nitrate ingested, studying the cancer risk in populations ingesting varying amounts of nitrate, may provide evidence for the carcinogenicity of it in man (Fraser *et al.*, 1980). Several epidemiological studies have been done in this field. With the exception of the study of oesophageal cancer in Iran, and a few studies which have considered cancer risk in general, most epidemiological studies have examined the carcinogenic activities of nitrates (nitrosamines) in relation to gastric cancer (Fraser *et al.*, 1980; Mirvish, 1983). The most persuasive evidence for a link between gastric cancer and high nitrate ingestion comes from Colombia, parts of which have a very high incidence of gastric cancer. Wells with nitrate (as NO_3) concentrations up to 300 mg/ℓ are a feature of several of the high risk areas (Fraser and Chilvers, 1981). In Chile, a strong association between fertiliser usage and gastric cancer mortality was documented (Fraser *et al.*, 1980). A study done in the UK in 253 urban areas could not find a positive association between nitrate in drinking water and the risk of stomach cancer (Beresford, 1985).

There is no unequivocal epidemiological evidence that nitrosamines (or their precursors) are carcinogenic in man, but it is probable that man is sensitive to the carcinogenic action of these compounds. The type and quantity of nitrosamines which are carcinogenic to man have still to be determined, as is the amount taken in or produced in the body. Epidemiological studies are complicated as subjects possibly consume diverse carcinogens which induce the same type of cancer (Adam, 1980).

The evidence suggests that in the high risk areas for gastric cancer there is an environmental factor, the effect of which is so overwhelming that it overrides the effect of other factors, such as sex and social class, which is clearly relevant in populations at lower risk. Whether or not this factor is related to nitrate ingestion must await the results of further studies. On the epidemiological evidence to date, the hypothesis that high nitrate ingestion is involved in the aetiology of gastric cancer should not be lightly discarded. High nitrate ingestion *per se*, cannot be incriminated as a cause of oesophageal cancer in Iran, but lack of nitrosation inhibitors in an impoverished diet cannot be excluded. At present, there is too little information available to draw any conclusions about the relationship between high nitrate ingestion and any other human cancer (Fraser *et al.*, 1980; Mirvish, 1983).

South African studies - past and present

Very few studies have been published on the health effects of nitrates in drinking water in South Africa. Probably the only epidemiological study published is that by Super *et al.* (1981) on a group of Namibian infants. Methemoglobinemia is frequently encountered in some areas of Nimibia. This well-planned and executed study involved 486 infants consuming well water with various nitrate concentrations. A strong correlation between actual nitrate intake and MetHb levels was found. Super *et al.* (1981) also found that the regular administration of vitamin C to infants is more important than the effects of age in lowering MetHb levels.

Water quality criteria and standards

The first federal drinking-water standard for nitrate was established in 1962. Table 1 lists the maximum concentrations in drinking water as recommended in several countries (Adam, 1980; Fan *et al.*, 1987). The current water standard for nitrate is based on protection from methemoglobinemia.

Evidently, 10 mg/ℓ nitrate (as N) is regarded as a safe limit by the majority of health authorities. Only when the concentration of nitrate in drinking water reaches 20 mg/ℓ (as N) will it become the main component of the total nitrate intake of adult persons. This indicates that the limit of 10 mgN/ℓ in drinking water may be overconservative for adults, but that it should not be raised to more than 20 mgN/ℓ.

At present, several million people in Europe depend on drinking water having a nitrate level in excess of the WHO guideline value of 10 mg/ℓ as N and the number is likely to increase sharply within the next few years. Owing to the carcinogenicity of a number of N-nitroso compounds to animals, there has been concern about their production in humans. This may happen, particularly in individuals suffering from atrophic gastritis, whose gastric pH exceeds 4, with resulting high populations of bacteria in the stomach.

The WHO group considered that there is no convincing evidence of a relationship between gastric cancer and consumption of drinking water containing up to 10 mgN/ℓ. There is no firm epidemiological evidence at higher levels, but a link cannot be ruled out due to the inadequacy of data available. However, in most countries, gastric cancer is declining and any risk from nitrate would appear to be restricted to individuals with conditions associated with low gastric acidity, rather than to the population in general (Waddington, 1985). The working group recommended endorsement of the WHO guidelines of 10 mgN/ℓ. They considered that in cases where the value is exceeded, low-nitrate bottled water might be provided for infants. Physico-chemical and biological treatment not only removes nitrates from water, but can also drastically change the overall composition of the water and may increase treatment costs several fold. There is thus some justification in raising the water standard for nitrate. This has, however, not been done yet. The current standards are adequate to protect the very young from nitrate induced toxicity, both pre- and postnatally (Fan *et al.*, 1987).

TABLE 1
RECOMMENDED NITRATE LIMITS FOR DRINKING WATER IN SEVERAL COUNTRIES

Country	mg/ℓ*
USA Public Health Service	10
USA National Academy of Science	10
USA Environmental Protection Agency	10
Japan	10
South African Bureau of Standards	10
Australia	10
WHO European	<11,5
WHO International	10
Britain	20
Russia	9,0
West Germany	11,3
Israel	10,2
Austria	9,0
East Germany	6,8
Denmark	5,7
Switzerland	4,5
Czechoslovakia	3,4

*To convert the nitrate (as N) value to a nitrate (as NO_3) value multiply by 4,43

Concluding remarks

Research activities on the problems of the health effects of nitrates in drinking water have been carried out in the field (epidemiological) and in the laboratory (experimental animal models). Both of these study methods have provided invaluable information regarding the potential and actual effects of nitrate on humans.

The results of several epidemiological studies indicate that there is a relationship between concentration of nitrates in drinking water consumed mainly as powdered milk and raised MetHb levels in infants up to 24 months. Local nutritional practices as well as possible genetic factors may have a bearing on the prevalence of methemoglobinemia. The susceptibility is enhanced by several factors such as high fluid intake, inability to produce sufficient gastric acidity, Hb present as Hb-F, the supplementary food taken by the infant, medicines used containing such substances as bismuth substrate etc. A study of liquid intake among infants aged 1 to 5 months indicated that while during the cool months 90% of the liquid intake is made up of milk, as much as 50% can be in the form of tap water during the hottest month (Shuval and Gruener, 1975). This finding may have considerable influence on the actual nitrate intake of infants. The low level of MetHb reductase in infants at birth and for the first few months of life may provide a partial explanation of the particular susceptibility of this age group. Vitamin C was demonstrated to provide partial protection against methemoglobinemia in experimental animals and humans.

From both the field and the laboratory studies, evidence has been gathered that nitrates and nitrites may be more toxic than generally considered. The fact that a significant effect can be detected in infants consuming water having only slightly more nitrates than the current standard, raises the question as to the margin of safety provided by that standard. We are faced with evaluting the potential hazard of nitrate, nitrite and nitrosamines in our environment. There is ample room for carefully planned and sufficiently executed health studies to evaluate the effects of nitrates in drinking water on the general population.

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