University of Nebraska - Lincoln
DigitalCommons@University of Nebraska - Lincoln

Historical Materials from University of Nebraska-Lincoln Extension

Extension

1981

EC81-2400 Living with Nitrate

Gary W. Hergert
University of Nebraska-Lincoln, ghergert1@unl.edu

George W. Rehm

Follow this and additional works at: https://digitalcommons.unl.edu/extensionhist

Hergert, Gary W. and Rehm, George W., "EC81-2400 Living with Nitrate" (1981). *Historical Materials from University of Nebraska-Lincoln Extension*. 4361.
https://digitalcommons.unl.edu/extensionhist/4361

This Article is brought to you for free and open access by the Extension at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Historical Materials from University of Nebraska-Lincoln Extension by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

AGRI
S
85
E7
#81-2400

Nebr. Cooperative Extension Service—EC 81-2400

LIVING WITH NITRATE

Gary W. Hergert
Extension Specialist (Soils)

George W. Rehm
Extension Specialist (Soils)

Richard Davis, M.D.
University of Nebraska Medical Center

Constance Kies
Professor (Human Nutrition and Food Service Management)

Robert A. Britton
Associate Professor (Ruminant Biochemist)

Alex Hogg
Extension Veterinarian

William A. Lee
Public Health Engineer
State Health Department

Deon D. Axthelm
Water Resources Specialist

Issued in furtherance of Cooperative Extension work, Acts of May 8 and June 30, 1914, in cooperation with the U.S. Department of Agriculture. Leo E. Lucas, Director of Cooperative Extension Service, University of Nebraska, Institute of Agriculture and Natural Resources.

INTRODUCTION

Nitrogen is a normal part of the human environment. Within the soil system, natural biological processes ultimately convert all forms of nitrogen to the nitrate ion. Excess amounts of soluble nitrate-nitrogen can accumulate in groundwater and in plants. Thus, there exists a possibility for humans and animals to ingest nitrate-nitrogen in greater quantities than metabolic systems can tolerate.

State and University personnel are available to provide assistance to those concerned about nitrate problems.

This publication provides concise and factual information on the influence of excess nitrate on humans and animals and where to seek additional information on dealing with this situation. The contribution of all members of the Nitrate Task Force Committee on development of this publication is acknowledged.

Funds for the initial publication were provided as part of a grant from EPA Region VII through the Nebraska Department of Environmental Control.

Throughout this publication the term nitrate-nitrogen or its abbreviated form NO_3-N, which means nitrogen in the form of nitrate, will be used. Other abbreviations found will be N for nitrogen, NO_3 for nitrate, NO_2 for nitrite, and NO_2-N for nitrite-nitrogen.

For standardization, all concentrations of nitrate and nitrite will be given in terms of NO_3-N or NO_2-N.

K. D. Frank, Chairman
Nitrate Task Force Committee
Cooperative Extension Service
Institute of Agriculture and Natural Resources
University of Nebraska–Lincoln

Nitrogen in Our Environment

Gary W. Hergert
Extension Specialist (Soils)
George W. Rehm
Extension Specialist (Soils)

Nitrogen has a unique place in our environment. World-wide it is the plant nutrient most limiting for production of food and fiber. Throughout recorded history, man has always strived to add nitrogen to crops either by using animal manures, legume crops, or fertilizers. By world standards, the inexpensive food Americans enjoy can be largely attributed to the availability and use of nitrogen fertilizer.

Basic to Environment

Nitrogen is a basic part of our environment. It accumulates in soils during the process of soil formation. During the thousands of years of soil development nitrogen accumulated in soil from additions in rainfall and plant and microbial fixation of nitrogen gas from the atmosphere. Nitrogen accumulated in the soil organic matter which was produced from decaying plants and animal residues.

Many of our virgin prairie soils contained four to six thousand pounds of organically bound nitrogen when they were first plowed. However, once a soil is tilled and crops are grown, the organic matter and nitrogen content start to decrease.

The organic nitrogen in soils changes slowly to inorganic nitrogen during the growing season at a rate of about 1 to 2 percent per year. Soils that once contained 4 to 5 percent organic matter now contain 2 to 3 percent organic matter after 50 years of continuous cropping with no additions of nitrogen. Systems of agriculture which rely heavily on nitrogen reserves in the soil to meet the nitrogen requirements of plants cannot be efficient in producing high yields of crops for a very long period of time.

Slash and Burn

The primitive slash and burn system, or the practice of shifting cultivation used in many tropical areas, can be productive without nitrogen fertilizer for a few years but new soil must be brought into production as the repeated cropping depletes the reserves of soil nitrogen. Old fields are allowed to return to native vegetation for several years to build up reserves of soil nitrogen. In years before commercial fertilizer was available farmers used crop rotations that included legumes to restore depleted nitrogen.

The use of nitrogen fertilizer was a breakthrough for agriculture because it meant the same field could be farmed continuously for grain production and the soil organic matter level could be maintained or increased in many soils while crop yields remained high.

Although the large majority of the total amount of nitrogen in soils exists in organic forms, plants take up nitrogen in the mineral form as either nitrate (NO_3-) or ammonium (NH_4+) ions. Most nitrogen used by plants is absorbed as the nitrate ion. Plants do not take up organic forms of nitrogen. This means that organic sources of nitrogen must be converted to the nitrate form of nitrogen before they can be used by plants. Several steps are needed in the conversion of organic nitrogen to nitrate-nitrogen and these are shown in what is called the **nitrogen cycle.** The general features of the nitrogen cycle are shown in Figure 1.

Nitrogen that has potential for use by plants can enter this cycle at several points. Animal manures, compost, sewage sludge as well as legume crops are organic sources of nitrogen. Some nitrogen fertilizers already contain the nitrogen in the readily available nitrate form. The nitrogen in other types of fertilizers must be converted to the nitrate form.

Once nitrogen is added to the soil as fertilizer nitrogen, crop residues, legumes or manures, it becomes part of the soil N system and some is eventually converted to nitrate-nitrogen as depicted by the N cycle. It's also important to point out that plants cannot distinguish between the original source of the nitrate-nitrogen it uses. The nitrate that results from the decomposition of manure, for example, is not different from the nitrate that comes from commercial fertilizer.

The total amount of nitrate-nitrogen generated through the processes of the nitrogen cycle is not necessarily used by plants. When the supply of nitrate-nitrogen is greater than the amount used by plants, there is an increased potential for accumulation of nitrate nitrogen and for loss of nitrogen from the system.

Nitrate-Nitrogen Can Be Lost

Nitrate-Nitrogen can be lost from the soil system by: **a. leaching, b. denitrification, c. volatilization of nitrogen gases and tie-up by soil bacteria.** Nebraskans are most concerned about losses due to leaching. Leaching is nothing more than the downward movement of nitrate-nitrogen through the soil with water. Leaching cannot occur if there is no downward movement of water.

The potential for nitrogen leaching is not the same for all parts of Nebraska. Sandy soils are very permeable and will not hold much water. Other soils in the state will hold larger amounts of water. Therefore, the potential for leaching of nitrate-nitrogen is greater in our sandy soils, but can occur on our fine textured soils.

The nitrate-nitrogen which moves downward through soils is not necessarily all derived from nitrogen fertilizers. The breakdown of organic sources of nitrogen through the processes of the nitrogen cycle also produces nitrate-nitrogen. This nitrate-nitrogen can move through the soils in the same manner as nitrate-nitrogen supplied as nitrogen fertilizers. Research in Nebraska has shown that large accumulations of nitrate-nitrogen occur several feet below the surface of some soils which have never been farmed or fertilized. This nitrate-nitrogen accumulated during the geologic past and has remained in the soil because of our relatively dry climate.

Since the downward movement of nitrate-nitrogen through the soils was probably taking place before the presence of man in Nebraska and will continue, it's unreasonable to expect that this movement can be stopped or eliminated. Man's alteration of his environment to produce food, however, can increase the rate of this movement. There are, however, management practices which farmers can use to minimize the leaching of large amounts of nitrate-nitrogen from irrigated soils—especially sandy soils.

Prevent Excessive Leaching

The control of two factors will prevent excessive leaching of nitrate-nitrogen in irrigated sandy soils. Leaching of nitrate can be reduced substantially if adequate, but not excessive, rates of nitrogen fertilizer are used. The choice of the rate of nitrogen doesn't have to be left to chance. The practice of taking soil samples to a depth of at least three feet, combined with the selection of a realistic yield goal, can result in the selection of a rate of fertilizer nitrogen which will provide an adequate amount for maximum economic yield without supplying an excessive amount which would be subject to leaching.

Remember, leaching cannot occur if there is no downward movement of water. We cannot control the amount of rainfall. We can, however, control the amount of water supplied through the irrigation systems. Research has shown that the practice of irrigation scheduling will substantially reduce the amount of water that moves through soils. These two factors—proper nitrogen fertilizer rate based on a realistic yield goal and soil samples to at least three feet, plus improved irrigation water management based on irrigation scheduling—are the keys to slowing down and reducing the amount of nitrate that moves to the groundwater.

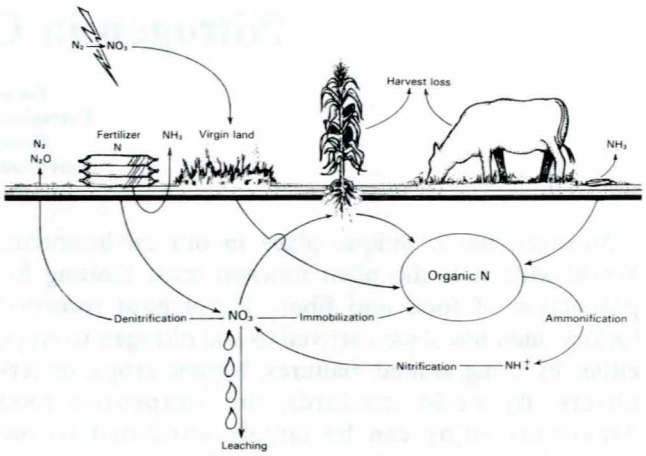

Figure 1. The nitrogen cycle.

It might appear that these management practices are too complex for use on a routine basis. However, many Nebraska farmers have shown that the use of these two practices is not difficult. In addition, yields have not been reduced when these practices are used. Farmers can use realistic yield goals for the soil and climate, soil testing for residual nitrate, irrigation scheduling, split application of nitrogen fertilizer, and nitrification inhibitors to obtain maximum use of fertilizer nitrogen without a reduction in yield while reducing the amount of nitrate that moves downward in the soil.

Nitrates, Nitrites and Methemoglobinemia

Richard Davis, M.D.
U.N. Medical Center

The hemoglobin of red blood cells serves as an oxygen carrier for body tissues. To function in this capacity, the iron in the hemoglobin molecule must be in the reduced or ferrous state. Oxidation of iron to the ferric state results in the formation of methemoglobin. Methemoglobin production occurs in normal individuals, but formation is counterbalanced by a more rapid reduction process. As a result, less than 1% of the total circulating hemoglobin in a healthy adult is present in the form of methemoglobin. The normal methemoglobin concentration in healthy infants is 2%.

In methemoglobinemia, the hemoglobin-methemoglobin equilibrium is disturbed, and methemoglobin accumulates as a brown pigment in the red cells, causing anoxemia and cyanosis (anoxemia is lack of oxygen supplied to tissues; cyanosis is blueness due to oxygen lack). Methemoglobinemia may arise as the result of a hereditary enzyme deficiency (1) a structural defect in the hemoglobin molecule, or a toxic substance which either oxidizes hemoglobin directly or facilitates its oxidation by molecular oxygen. The primary health hazard from nitrates relates to their potential for reduction to

nitrites. Nitrites, chlorates, quinones, sulfonamides, and aromatic amino and nitro compounds oxidize hemoglobin.

Nitrate in Drinking Water

Methemoglobinemia resulting from high nitrate concentrations in drinking water was first clinically recognized by Comly in 1945 (2). Drinking water may contain high concentrations of nitrogen salts as the result of pollution by organic materials or inorganic chemical fertilizers. The majority of reported cases have been in infants under the age of four months (3, 4).

Nitrates are usually absorbed before reaching the nitrate-reducing bacteria which commonly reside in the lower GI tract. The increased susceptibility of infants may be related to the lack of acidity in the gastric juices of newborns which allows nitrate-reducing bacteria to flourish in the upper GI tract as well. When nitrates are introduced directly into the colon, methemoglobinemia is readily produced (5). Most cases of infant methemoglobinemia due to contaminated water have been associated with nitrate concentrations (as NO_3) in excess of 40 ppm. As a result, the U.S. Public Health Service and the World Health Organization have recommended drinking water standards of not greater than 45 ppm (6). Remember 45 ppm NO_3 equals 10 ppm NO_3-N.

One case of adult methemoglobinemia due to contaminated water has been reported. The water, which contained 94 ppm (as NO_3-N) was being used by the patient in home dialysis. Methemoglobin was identified spectrophotometrically, but was not determined quantitatively.

Sausages and other processed meats have been reported to cause methemoglobinemia (7). Preservatives used in the preparation of sausage contain nitrites or nitrate salts, and the nitrates may be reduced to nitrites by bacteria or enzymes present in the meat. The U.S. Food and Drug Administration has set maximum allowable nitrate and nitrite concentrations in food at 500 ppm and 200 ppm, respectively as NO_3-NO_2.

Nitrates in Leafy Vegetables

Leafy vegetables such as spinach, cauliflower, cabbage and beets, have relatively high nitrate concentrations, and nitrate concentrations may be even higher because of certain fertilization practices. There have been several reports of methemoglobinemia following the consumption of spinach. However, studies have shown that the conversion of nitrates to nitrites during storage, rather than the nitrates themselves, was responsible for the development of methemoglobinemia (8). Animal studies have suggested that high-nitrate containing vegetables do not induce methemoglobinemia. Furthermore, it has been suggested that other compounds, possibly ascorbic acid, present in the leafy vegetables may provide protection against *in vivo* reduction of nitrates to nitrites (9).

Cases of methemoglobinemia have also been reported as a complication of silver nitrate therapy for burns (10), and the use of bismuth subnitrate in radiologic procedures (11).

References

1. Jaffe, ER and Heller P: Methemoglobinemia in Man. Prog. Hemat. 4:48, 1964.
2. Comly, H: Cyanosis in infants caused by nitrates in well water. JAMA 129: 112, 1945.
3. Rosenfield, A. B., and Huston, R: Infant Metheoglobinemia in Minnesota due to Nitrates in Well Water. Minn. Med. 33: 787, 1950.
4. Fassett, D. W.: Nitrates and Nitrites in: toxicants occurring naturally in foods. National Academy of Sciences, 2nd Edition. Washington, D.C. 1973.
5. Cornblath,M. and Hartman A. F.: Methemoglobinemia in infants. J. Ped. 33: 421, 1948.
6. Shuval H.: Epidemiological and toxicological aspects of nitrates and nitrites in the environment. Am. J. Publ. Health 62:1045, 1972.
7. Orgeron J. D. *et al.*: Methemoglobinemia from eating meat with high nitrite content. Pub. Hlth Rep 72:189, 1957.
8. Committee on Nutrition. Infant methemoglobinemia. Pediat. 46:475, 1970.
9. Stoewsand G. S., *et al.*: Nitrite-induced methemoglobinemia in guinea pigs: Influence of diets containing beets with varying amts. nitrate and effect of ascorbic acid and methionine. J. Nutr 103:419, 1973.
10. Fernberg J. L. and Luce, E.: Methemoglobinemia: A complication of silver nitrate treatment of burns. Surg. 63:328, 1968.
11. Roe H. E.: Methemoglobinemia following administration of bismuth subnitrate. JAMA 101:352, 1933.

Nitrates, Nitrites, N-Nitroso Compounds and Nutrition

Constance Kies, Professor
Dept. of Human Nutrition and Food Service Management

While nitrates, nitrites and N-nitroso compounds are known to be related, that nutrition also may have some involvement is less well recognized. Nitrates are widespread in food products and sometimes in water but are relatively harmless as such to the human (WHO, 1978).

However, nitrates can be reduced under certain conditions to nitrites either in food before being consumed or within the human body. High levels of nitrites, regardless of source, can cause the blood abnormality, methemoglobinemia, and possibly other abnormalities. Nitrites also can react with various nitrogen compounds to form potentially cancer-causing N-nitroso compounds at almost any site within the gastro-intestinal tract (WHO, 1978).

Human Nutrition Involved

Human nutrition becomes involved in the nitrate/nitrite/N-nitroso compound situation in several ways. These include: a. selection of foods and food patterns with low nitrate contents for those with concerns rela-

tive to high nitrate intake; b. treatment and care of food within the home to reduce the transformation of nitrates to nitrites; and c. delineation of involvement of nutrients in the conversion of nitrates to nitrites to N-nitroso compounds.

Plant products vary in their nitrate content depending upon the plant species, the part of the plant used, amount of nitrate in the soil (either residue or fertilization added), water nitrate levels, other environmental factors, and agricultural practices (Wolff and Wasserman, 1972; WHO, 1978; Viets and Hageman, 1971; NRC, 1972). Some plant species naturally have a very high nitrate content. However, within the same species, nitrate content varies because of genetic make-up and the aforementioned environmental and agricultural practices.

Nitrate in Food Products

In general, even food products which naturally contain high levels of nitrates contain proportionally much, much lower amounts of nitrites. Nitrates ordinarily found in food products may be changed to nitrites through microbiological action. This can occur in fresh or cooked vegetables which are allowed to stand at room temperatures for extended periods of time or during storage of improperly processed food products (Hall and Hicks, 1977; Phillips, 1968). Several incidences of methemoglobinemia have occurred in young infants fed spinach which was unrefrigerated (WHO, 1978; Keating *et al*., 1973).

Nitrates and nitrites are sometimes added to food products, particularly meats. In the United States, amounts permitted are subjected to careful regulation. Nitrites in cured meat products do offer benefits in preventing growth of *Clostridium botulinum,* the toxin which causes the extremely serious type of food poisoning, botulism (WHO, 1978; Wolff, 1972).

The amounts of nitrates or nitrites actually consumed are in part determined by the level of these substances in the specific foods consumed and in part by the amount and frequency of consumption of these foods (White, 1975). For example, spinach is one food that naturally contains high amounts of nitrates. However, typically, spinach isn't eaten very often by most people or in very large amounts. Hence, for most people foods such as spinach and turnip greens which naturally have high nitrate contents do not contribute much nitrate to the diet simply because most people don't eat them very often or in large quantities. However, fresh cabbage or iceberg lettuce which contain lower amounts of nitrates than do fresh spinach or turnip greens probably contribute a fairly large amount of nitrates to the diet of the typical Nebraskan because they are generally well liked and are eaten frequently by many people.

Water Contains Nitrate

Water is a form of food which is consumed more frequently and in larger amounts than is any other food. For this reason, an elevation in the nitrate content of the water supply has a great impact on the total amount of nitrates consumed.

Obviously, when a drinking water supply is found to have a high nitrate content, the most effective approach is to find out why and to eliminate the problem. This isn't always possible. Other approaches include using bottled water or water purification systems. A third approach is to cut down on eating other nitrate foods so as to minimize the total, overall nitrate consumption. In Table 1, nitrate contents of some food products are listed. In Table 2 are some examples of high and low nitrate menus. These figures should only be viewed as estimates, not absolutes, since ranges in nitrate/nitrite content exist for all. Another problem in this approach is that it discourages the use of many food products which have positive nutritional attributes as well as adding color, taste, and variety to meals.

Food Preparation

Food preparation procedures also have an impact upon the nitrate/nitrite problem (WHO, 1978). Obviously, addition of high nitrate or nitrite-containing water to a food product in its preparation will increase the nitrate/nitrite content of the final product. Heating of the water either before its addition or as part of the preparation procedure will not reduce the nitrate/nitrite content. Use of minimal level nitrate/nitrite water in the preparation of infant formulas or foods, as well as low-nitrate food in general, is important since infants are prone to the sometimes fatal blood disorder, methemoglobinemia, caused by a high intake of nitrates/nitrites.

Since methemoglobinemia is caused by nitrites, rather than nitrates, conversion of nitrates to nitrites in food preparation procedures should be avoided. Do not allow fresh vegetables to stand at room temperature for extended periods of time after being harvested. For this reason "fresh" vegetables purchased in grocery stores are usually higher in nitrite content than are their frozen or canned counterparts. The latter are usually quickly processed following harvest allowing less time for conversion of nitrates to nitrites. Conventional home canning and freezing practices will minimize nitrate/nitrite conversion by eliminating or limiting microbial action which is responsible for the change.

Lack of Oxygen

Methemoglobinemia is caused by the reaction of nitrites with the hemoglobin in red blood cells to form methemoglobin which lacks the oxygen-carrying ability

Table 1. Nitrate contents of selected vegetables.[a]

Food	Nitrate content mg/100g food
Artichoke (frozen)	1.2
Asparagus (canned)	0.3
Asparagus (fresh)	2.1
Asparagus (frozen)	1.6
Beans (dry)	1.3
Beans, green (canned)	10.0
Beans, green (frozen)	27.0
Beans, lima (fresh)	5.4
Beans, lima (frozen)	2.7
Beans, snap (fresh)	25.3
Beets (canned)	145.0
Beets (fresh)	301.0
Broccoli (fresh)	78.3
Broccoli, spears (frozen)	46.4
Broccoli, chopped (frozen)	57.3
Brussel sprouts (frozen)	8.4
Cabbage (fresh)	78.4
Carrots (canned)	20.5
Carrots (fresh)	7.2
Carrots (frozen)	9.7
Cauliflower (fresh)	54.7
Cauliflower (frozen)	25.4
Celery (fresh)	234.0
Collard greens (canned)	264.0
Collard greens (frozen)	245.0
Corn (frozen)	4.5
Corn (fresh)	4.5
Cucumbers (fresh)	2.4
Eggplant (fresh)	30.2
Endive (fresh)	66.3
Kale (canned)	277.0
Kale (frozen)	160.0
Lettuce, iceberg (fresh)	110.0
Lettuce, romaine (fresh)	140.0
Melons, (fresh)	43.3
Mushrooms (fresh)	6.3
Mushrooms (whole canned)	1.7
Mushrooms (sliced canned)	0.6
Mustard greens (canned)	136.0
Mustard greens (frozen)	239.0
Okra (frozen)	7.4
Onions (fresh)	13.4
Onions (chopped frozen)	3.3
Onions (whole frozen)	12.8
Okra (canned)	0.2
Peas (fresh)	2.8
Peas, green (frozen)	2.0
Peas, green (canned)	0.6
Pea pods, Chinese (frozen)	1.3
Peas, blackeyed (frozen)	0.9
Peppers, sweet green (canned)	6.2
Peppers, sweet (frozen)	5.0
Pickles	5.9
Potatoes (fresh)	11.9
Potatoes, hash browns (frozen)	3.7
Potatoes, small whole (frozen)	15.0
Potatoes, whole (canned)	6.3
Potatoes, sliced (canned)	6.9
Radishes (fresh)	240.0
Pumpkin (fresh)	41.3
Salad, mixed (fresh)	81.9
Sauerkraut (fresh)	19.1
Sauerkraut (canned)	6.8
Spinach (canned)	57.3
Spinach (canned)	222.0
Spinach (frozen)	214.0
Squash, accorn (fresh)	3.4
Squash, butternut (fresh)	67.8
Squash, zucchini (fresh)	66.5
Squash, zucchini (frozen)	53.3
Squash (frozen)	16.0
Sweet peppers (fresh)	12.5
Sweet potatoes (fresh)	5.3
Tomatoes (fresh)	6.2
Turnip greens (frozen)	346.0
Turnip greens (canned)	223.0

[a]Values given were converted from values reported by McNamara *et al.* (1971), White (1975) and Siciliano (1975). All values are mean values, which, in some cases, represent considerable ranges. Since analyses were done by different laboratories at different times on different samples, values should be considered more relative than absolute.

of normal hemoglobin (Rodkey, 1978; WHO, 1978). This means that in methemoglobinemia, the blood lacks the ability to carry sufficient oxygen to individual cells of the body. Most **adult** humans have the ability to rapidly convert methemoglobin back to oxyhemoglobin; hence, the total amount of methemoglobin within red blood cells remains low in spite of relatively high levels of nitrate/nitrite intake. In the **young infant,** enzyme systems for reducing methemoglobin to oxyhemoglobin are incompletely developed; hence, methemoglobin within blood cells can build up with excessive nitrite intake and the forementioned serious condition of methemoglobinemia can occur. This also may happen in **older** individuals who genetically have impaired enzyme systems for the reduction of methemoglobin.

Nitrates in the diet may be converted to nitrites in the mouth, the small and the large intestines. This may also occur in the stomach if the contents are insufficiently acidic (Tannenbaum *et al.*, 1976, WHO, 1978). Typically, the stomach contents of the infant are less acidic

Table 2. Examples of high and low nitrate meals.

High nitrate meals	Low nitrate meals
Example 1	*Example 1*
Spinach salad	Cucumber salad
Sliced cold ham	Sliced cold beef
Whole wheat bread and butter	Whole wheat bread and butter
Ice cream	Ice cream
Milk	Milk
Example 2	*Example 2*
French fried potatoes	French fried potatoes
Frankfurter on bun	Ground beef patty on bun
Celery and radishes	Pickles
Chocolate cake	Chocolate cake
Milk	Milk
Example 3	*Example 3*
Beet salad	Pea salad
Knotwurst and turnip greens	Pork chops and sauerkraut
Boiled potatoes	Boiled potatoes
Milk	Cherry pie
Apple pie	Milk

than are those of the adult which suggests another reason for greater susceptibility of the infant to the dangers of high nitrate content. Both nitrates and nitrites may be absorbed into the body. Nitrates and nitrites which are absorbed may be excreted in the urine. Absorbed nitrates may also be recycled through the saliva where-microbial action in the mouth changes it to nitrites, and it passes into the gastro-intestinal system. Thus, saliva is the major source of nitrites to the human, but this nitrite really is simply dietary nitrate that is being recycled.

The effect of absorbed nitrate/nitrite content is unresolved (WHO, 1978). Animal studies suggest that this transfer is relatively low; however, there is considerable variability among animal species. Thus, the question of degree of risk of pregnant women or nursing mothers consuming high nitrate water/diets is unknown. Since most parents or future parents do not favor assuming unknown risks for their children, nursing mothers and pregnant women are generally included on lists of individuals who should avoid high nitrate water.

Carcinogenic Compounds

Nitrates and nitrites which are not absorbed or which are recycled back into the gastrointestinal tract may undergo further transformation with amines or amides to form nitrosamines or nitrosamides (N-nitroso compounds). Nitrosamines and nitrosamides are also found in small amounts in food and water; hence, may be directly consumed. These N-nitroso compounds may be absorbed into the body. The breakdown products of N-nitroso compounds or the N-nitroso compounds themselves for the most part are quickly removed from the body either in the urine or expired into the air (WHO, 1978). However, a high proportion of N-nitroso compounds have been shown to be carcinogens. The organ affected seems to be related to species, specific N-nitroso compound tested, level of dosage, and length of time of exposure.

Implications of the importance of N-nitroso compounds in the incidence of human cancer has not been fully defined. In animal studies designed to determine whether or not a substance can cause cancer, proportionally much higher levels of the test substance are fed than are realistically found in human diets. This is done in part because certain individuals within a human population may be far more susceptible to cancer triggering agents than are others. While this technique is very useful and is scientifically sound, it sometimes causes another problem, that of over-concern.

Nutrition has been found to influence the impact of the nitrate/nitrite/N-nitroso compound problem. However, research evidence on the role of specific nutrients is relatively slim. Ascorbic acid (Vitamin C) and fiber, substances often found in high nitrate vegetables, seem to hold some promise.

Ascorbic Acid May Help

Ascorbic acid of all nutrients seemingly holds greatest promise as a nitrate/nitrite/nitrosamine detoxifer. Production of carcinogenic N-nitroso compounds from nitrite and nitrosamines has been blocked under most but not all experimental conditions with ascorbate (Mirvish, *et al.*, 1972). Mutagenicity of fish extracts after incubation with nitrite also was prevented by ascorbic acid additions (Marquardt *et al.*, 1977). Dietary ascorbic acid has been found sometimes to be associated with reduction but not elimination of methemoglobin in guinea pigs (Hathcock, 1976, 1975; Kilgore *et al.*, 1964; Stoewsand *et al.*, 1973). The effect of concurrent methionine supplementation with the ascorbic acid was found to reduce nitrite-induced methemoglobinemia to an even greater extent, suggesting a protein involvement in the detoxification process. Nitrites have also been found to increase the ascorbic acid requirement of the guinea pig (Hathcock, 1975, 1976).

These results imply that the common practice of addition of ascorbic acid to cured meats for color enhancement might have unforeseen beneficial effects relative to the nitrate/nitrite problem. Furthermore, these results imply that individuals consuming marginal or inadequate amounts of ascorbic acid might be particularly susceptible to nitrate/nitrite hazards. Nitrate/nitrite toxic-prone individuals, including those with low stomach acidity such as young infants or individuals receiving selected medications, might also receive some protection via ascorbic acid dietary additions.

As previously mentioned, methionine in combination with ascorbic acid reduced incidence of methemoglobinemia in guinea pigs. A news release describing work by S.R. Tannenbaum suggests that increases in dietary nitrogen (protein) increases synthesis of nitrites in the upper, aerobic portion of the intestines of adult humans (Anonymous, 1978). Earlier reports sought to define nitrate to nitrate conversion in human saliva (Eisenbrand and Preussman, 1976; Tannenbaum *et al.,* 1976).

Other reports suggest that increases in dietary fiber offer a protection against possible carcinogenic effects of nitrosamines by acting as a diluent since fecal bulk is increased, decreasing time exposure of intestinal surface to carcinogens by decreasing fecal transit time, or by surface absorption of carcinogen on fiber materials (Burkitt *et al.*, 1971; Kies and Fox *et al.*, 1978). Possible interactions of nitrates/nitrites with vitamins E, A and D have also been suggested (Maneschu *et al.*, 1975).

Nutrition Can Help

In conclusion, for individuals living with a high nitrate water problem, nutrition cannot be expected to completely overcome the difficult situation. Young in-

fants, pregnant and nursing women are groups of particular concern.

Of more practical concern to the consumer relative to the nitrate/nitrite problem are food selection and preparation procedures within the home. Hazards associated with high nitrogen fertilization such as of home gardens, use of high nitrate/nitrite foods or possible conversion of nitrates to nitrites in high nitrate-containing vegetables due to improper food handling are situations with which the consumer should be aware.

References

1. Anon., 1978. Nitrosamines may form easier than suspected. Chem. and Eng. News 56:6.
2. Burkitt, D., Walker, D. and Painter, N. 1971. Effect of dietary fiber on stools and transit times and its role in the causation of disease. Lancet 2:1408.
3. Eisenbrand, G. and Preussman, R. 1976. Influence of dietary nitrate on nitrite content of human saliva; possible relevance to in vivo formation of N-nitroso compounds. Food and Cosmetic Toxicology, pg. 545.
4. Goaz, P.W. and Biswill, H.A. 1961. Nitrate reduction in whole saliva. J. Dent Res. 40:355.
5. Hall, C.B. and HIcks, C.B. 1977. Nitrites in inoculated carrot juice as a function of nitrate content and temperature. J. Fd. Sci. 42:549.
6. Harada, M., Ischuvata, H., Nakamura, Y., Tanimura, A., and Ishidate, M. In vivo formation of nitroso compounds. I. Changes of nitrite and nitrate concentration in human saliva after ingestion of salted Chinese cabbage. J. of The Food Hygienic Society of Japan.
7. Hathcock, J.H. 1976. Nutrition: toxicology and pharmacology. Nutr. Rev. 34:65.
8. Hathcock, J.H. 1975. in *Trace Substances in Environmental Health-IX* D.D. Hemphill, Editor, Univ. of Missouri, Columbia, MO.
9. Keating, J.P., Lell, M.E., Straus, A.W., Zarkowsky, H. and Smith, G.E. 1973. Infantile methemoglobinemia caused by carrot juice. N.E. J. Med. 288:825.
10. Kies, C. and Fox, H.M. 1978. Fiber and protein nutritional status. Cereal Foods World 23:249.
11. Kilgore, L. Stasch, A.R. and Barrentine, 1964. Relation of ascorbic acid to nitrate content of turnip greens and to methemoglobin formation. Am. J. Clin. Nutr. 14:52.
12. Kociba, R.J. and Sleight, S.D. 1970. Nitrite toxicosis in the ascorbic acid-deficient guinea pig. Toxical. Appl. Pharmacol. 16:424.
13. Maneschu, S. *et al.*, 1975. Effects of nitrates in drinking water on health of children. Igiena, 1975, 199–202. Abstract Nutr. Abstracts and Rev., 3457, 1977.
14. Marquardt, H., Rufino, F. and Weisburger, J.H. 1977. On the aetiology of gastric cancer: mutagenicity of food extracts after incubation with nitrite. Food and Cosmetic Toxicology 15:97.
15. Mirvish, S., Wallcave, L., Eagen, M. and Shubik. 1972. Ascorbic-nitrite reaction: possible means of blocking the formation of carcinogenic N-nitroso compounds. Science 177:65.
16. National Research Council, Committee on Nitrate Accumulation. 1972. *Accumulation of Nitrate.* National Academy of Sciences, Washington, D.C.
17. Philips, W.E.J. 1968. Changes in nitrate and nitrite contents of fresh and processed spinach during storage. J. Agricul. Food Chem. 16:88.
18. Rodkey, F.L. 1976. A mechanism for the conversion of oxyhemoglobin to methemoglobin by nitrite. Clin. Chem. 22:1986.
19. Roglin, J. 1978. Americans confused by food additive studies. Lincoln Journal, Oct. 9, 1978, p. 7.
20. Siciliano, J., Krulick, S., Heisler, E.G., Schwartz, J.H. and White, J.W. 1975. Nitrate and nitrite content of some fresh and processed market vegetables. J. Agric. Food Chem. 23:461.
21. Stoewsand, G.S., Anderson, J.L. and Lee, C.Y. 1973. Nitrite-induced methemoglobinemia in guinea pigs: influence of diets containing beets with varying amounts of nitrate, and the effect of ascorbic acid and methionine. J. Nutr. 103:419.
22. Tannenbaum, S.R., Weisman, M., and Fett, D. 1976. The effect of nitrate intake on nitrite formation in human saliva. Food & Cometic Toxiocology 14:549.
23. Tannebaum, S.R., Fett, D., Young, V.R., Land, P.D. and Bruce, W.R. 1978. Nitrate and nitrite are formed by endogenous synthesis in the human intestine. Science 200:1487.
24. Viets, F.G., Jr. and Hageman, R.H. 1971. *Factors affecting the accumulation of nitrate in soil, water and plants.* Agric. Handbook, No. 413, Agric. Research Service, U.S.D.A., Washington, D.C.
25. Wang, T., Kakizoe, T., Dion, P., Furrer, R. Varghese, A.J. and Bruce, W.R. 1978. Volatile nitrosamines in human feces. Nature 276:278.
26. White, J.W. 1975. Relative significance of dietary sources of nitrate and nitrite. J. Agric. and Fd. Chem. 23:888.
27. Wolff, J.A. and Wasserman. 1972. Nitrates, nitrites and nitrosamines. Science 177:15.
28. World Health Organization. 1978. *Nitrates, Nitrites and N-Nitroso Compounds.* Environmental Health Criteria 5, Geneva: WHO.

Nitrate Toxicity in Livestock

Robert A. Britton
Associate Professor (Ruminant Biochemist)
Animal Science

Nitrate is a normal part of the nitrogen cycle in nature and is relatively nontoxic. Nitrite formed by reducing nitrate is quite toxic. The term nitrate toxicity, although not entirely correct, will be used in this report. Nitrate toxicity results from over-ingestion of nitrate or nitrite in feedstuffs, water, ingestion of nitrate fertilizer or a combination of these factors.

Plants absorb nitrate from soils. After absorption, plant nitrate is reduced to nitrite by nitrate reductase. The nitrite is reduced to ammonia by nitrite reductase. Nitrite and ammonia are as toxic to plants as they are to animals, therefore, their levels need to be controlled in the plant. This is accomplished by having the nitrate converted to nitrite rather slowly. The resulting low

levels of nitrite and subsequently ammonia are not harmful to the plant. The ammonia is used for amino acid and protein synthesis in the plant.

Levels of Nitrates

Levels of nitrates found in plants vary according to plant and environmental factors. The major plant factors are species, plant part, and stage of maturity. Certain weeds, such as lambsquarters, pigweed, and puncture vine accumulate high levels of nitrate. Corn, sorghum, millet and sudangrass will also accumulate nitrates. Perennial grasses and legumes are usually not important nitrate accumulators. Nitrate is located in the stalk with very little being found in the leaf, seeds, or grain. Nitrate levels in stalk are highest in the area closest to the soil and lowest in the top of the stalk. Nitrates are usually highest in young plants and decrease with increasing maturity.

Environmental conditions that enhance nitrate levels in plants are generally those that interfere with the growth of the plant without diminishing nitrate uptake by the root. These factors include drought, herbicide or frost damage, disease, and shading. Rate of fertilization is also a major factor.

Drought and frost can lead to nitrate accumulations in plants. Frost, hail, drought, or low temperature usually damages leaves. Reducing leaf area impairs the plant's ability to reduce nitrate and convert it to protein. Temperatures of 55°F or less inhibit nitrate reduction in leaves. Herbicides can cause short term increases in plant nitrates by interfering with the plant growth systems, but herbicide treatment also may kill nitrate accumulating weeds. Plant diseases can also have profound effects on nitrate accumulation. Northern leaf blight in corn can raise nitrate concentrations 3-fold in the plants. Shade increases plant nitrate levels because light is required for nitrate reduction to occur. Periods of cloudiness or location in dense stands can increase plant nitrates but shading is generally a minor factor compared to the other environmental factors discussed. Generally, any condition which will interfere with leaf function and not harm the root system of the plant will increase nitrate levels.

Health Hazards

Health hazards and economic losses resulting from the ingestion of nitrates and nitrites by man and animals results from total nitrate intake. There is nothing to show that nitrate from water is more harmful than nitrate found in plants. Therefore, total nitrate intake from all sources is the important criteria.

The toxic agent, as mentioned earlier, is nitrite rather than nitrate. Nitrates are converted to nitrites by bacteria in the rumen. Nitrites are absorbed from the rumen or stomach into the blood stream and react with hemogloblin to form methemoglobin. Methemoglobin cannot transport oxygen. Acute toxicity or lethal effects occur when 80 to 90 percent of the hemoglobin is converted to methemoglobin. Because of the blood's reduced ability to bring oxygen to body tissues the animal suffocates. Levels of NO_3-N needed to cause toxicity in animals vary according to the level and type of feed as well as the method of administration of the nitrate. Species of animal is also a factor. Monogastrics, such as pigs, are much less susceptible to nitrate toxicity than ruminant animals such as cattle or sheep. A toxic dose of nitrate for ruminants, when put directly in the rumen of animals fed poor quality hay, was 75-90 mg NO_3-N/kg body weight.

Ruminant animals are considerably more resistant to nitrate toxicity when they are ingesting a source of readily fermentable carbohydrates or have been adapted to nitrates. Rumen fluid from sheep adapted to nitrate reduced nitrate to ammonia more readily than unadapted sheep. What this means is much less nitrate accumulates in the rumen than can be absorbed and cause toxicity.

Nitrate toxicity for monogastric animals is related to its reduction to nitrite before ingestion. Monogastrics are quite tolerant to high nitrate levels. Pigs administered with 300 mg NO_3-N/kg body weight directly into the stomach were poisoned. The cause of death was not the typical methemoglobinemia, but rather gastric hemorrhages. Nitrite, on the other hand, is quite toxic to monogastrics (12 mg NO_2-N/kg body weight). Work reported from South Dakota indicated pigs consuming water containing 300 ppm NO_3-N (total consumption 1.18 gm NO_3-N per day) had no detrimental effects on performance.

Symptoms of Toxicity

Symptoms of nitrate toxicity are dark brown or chocolate colored blood, excess salivation, loss of coordination, vomiting, abdominal pain, diarrhea, cyanotic (blue) mucous membranes, rapid weak pulse and low tolerance to exercise. Clinical signs may appear at levels of 30 to 40% methemoglobin. Death can occur at 80 to 90% methemoglobin. Diagnosis and treatment should be made by a veterinarian. Treatment usually consists of intraveneously infusing methylene blue. The methylene blue converts the methemoglobin back to hemoglobin and negates the toxicity.

An important question in this area is whether there are effects on animal performance from low levels or chronic nitrate ingestion. Four areas of concern have surfaced: a. reproductive problems, b. vitamin A destruction, c. intake depression, and d. disrupting thyroid function.

Acute nitrate toxicity has caused abortions in ruminants. These abortions occur when methemoglobin levels approach 80-90%. Pregnant cows with methemoglobin levels of 40-50% showed no effects on maintenance of pregnancy. Levels of 300 ppm NO_3-N in the drinking water or .07% NO_3-N in the feed had no

ill effects on reproductive performance in swine. The effects of nitrate or nitrite on reproductive performance of dams appears to be only important when the methemoglobin levels approach 80-90%.

Nitrate has been implicated as causing Vitamin A deficiencies in animals. Nitrate does not destroy vitamin A, but nitrite under acidic conditions found in the stomach can cause destruction of vitamin A and carotene. It does not affect vitamin A utilization after absorption. In view of the types of diets fed to pigs (feed grains contain essentially no nitrate or nitrite) it is difficult to imagine practical situations in which these animals would be exposed to nitrite for long enough periods to cause a vitamin A deficiency. Nitrite can destroy vitamin A in both the rumen and abomasum, but it has been very difficult experimentally to produce a vitamin A deficiency by feeding nitrates to ruminants. Nitrate can be reduced to nitrite or silo gases in forages during ensiling. Both nitrite and silo gases destroy vitamin A and carotenes, so animals fed these silages should get dietary supplements of vitamin A. Subacute or low level nitrate in feeds or water causes no general problem of vitamin A storage.

Reductions in feed intake and performance have been demonstrated in ruminants. Forages used were generally about .25% NO_3-N which approaches toxicity levels for ruminants. In a large number of research studies levels of nitrate encompassing a wide range have been fed to most classes of livestock with no negative effects.

Thyroid glands of animals fed nitrate or nitrite were enlarged. These effects were short lived (2 to 4 weeks) and easily overcome with additional iodine in the diet.

In general, preformance problems associated with chronic nitrate ingestion have been hard to measure and often contradictory reports appear in the literature.

Producer Tips

In conclusion, nitrate toxicity is still a problem to producers. The most important part of the problem relates to the acute toxicity. Relatively little evidence has accumulated that implicates subacute or chronic nitrate toxicity as being important.

Dilute high nitrate feeds with known low nitrate sources such as grain, grass hay, legumes or nitrate tested forages. Don't allow hungry animals access to high nitrate forages and allow ruminants to adapt slowly to high nitrate forages.

Nitrates and Animal Health

Alex Hogg
Extension Veterinarian

Water quality standards of nitrate-nitrogen recommended for human consumption are impractical and uneconomical for livestock or poultry water supplies.

Table 1 gives the maximum limits of nitrate-nitrogen recommended for human and livestock consumption by three government agencies.

Common sources of nitrate poisoning are plants such as pigweed, kochia, sorghum-sudan hybrids and corn plants. This is particularly true when plants are grown under adverse conditions such as high-nitrate fertilization, drought, or are sprayed with sublethal amounts of herbicides such as 2,4-D.

Nitrates are quite stable in dry forages but will be reduced about 50% by the ensiling process.

Cattle More Susceptible

Acute nitrate poisoning is much more likely to occur in cattle than in swine and is nearly always due to nitrate in forage rather than nitrate in drinking water.

Table 1. Limits of nitrate-nitrogen recommended for human and livestock consumption.[a,b,c]

	U.S. EPA[a] (for humans)	NAS[b]	CAST[c]
NITRATE-N (PPM)	10	100	300

[a]U.S. Environmental Protection Agency.
[b]National Academy of Sciences.
[c]Council for Agricultural Science and Technology.

Nitrate in feed and water are additive and both sources should be considered when evaluating a toxicity situation.

Acute poisoning in cattle occurs one-half to four hours after the ingestion of 0.5 gram nitrate nitrogen per kilogram of body weight or may be delayed for five to eight days.

Clinical signs can be noted when 30 to 40% of the hemoglobin has been converted to methemoglobin and death comes at 80-90% levels of methemoglobin. Stress speeds death so affected animals should be handled slowly and gently.

Signs of Poisoning

Clinical signs of nitrate poisoning are those of lack of oxygen and are:

Labored breathing
Cyanosis (mucous membranes are blue)
Rapid heart beat
Blood — chocolate-brown color before death and about 2 hours after death
Recovered pregnant cows may abort several days later
Death — several hours to 12-24 hours after the beginning of clinical signs.

Diagnosis is by clinical signs, postmortem lesions and laboratory analyses.

The following can be analyzed for nitrate and nitrite:

Rumen or stomach contents
Plasma
Serum
Urine
Forage
Water

Treatment is the intravenous injection of methylene blue at the rate of 2 milligrams per lb of body weight. In addition, pumping several gallons of ice water containing antibiotics into the rumen to inhibit the bacteria which convert nitrate to nitrite may be beneficial.

CAUTION: An overdose of methylene blue also produces methemoglobin and should be avoided.

Acute nitrate poisoning in animals may be expected when nitrate-nitrogen exceeds 345 ppm in water or 0.23% nitrate-nitrogen in forage (dry weight basis).

The bulk of the evidence indicates that chronic nitrate poisoning is very rare and difficult to substantiate. On the other hand, moderate levels of nitrate in water continues to be incriminated in several animal health problems.

The following health problems have been cited as being the result of chronic nitrate poisoning:

1. Poor growth rate
2. Abortion
3. Infertility
4. Vitamin A deficiency
5. Interference with iodine deficiency
6. Higher susceptibility to infection.

However, experimental evidence to substantiate these claims is lacking.

Prevention

1. Test forage before feeding. Remember that ensiling will remove about 50% of the nitrate so perform the test just before starting to feed the ensilage.
2. Gradually introduce the high nitrate forage—ruminants do adapt to higher levels of nitrate.
3. Dilute high nitrate feed with other feeds such as alfalfa hay or grain.
4. Be sure drinking water does not contain more than 345 ppm of nitrate-nitrogen.
5. When cutting high nitrate forage raise the cutter bar and leave the lower stalks in the field.
6. Watch animals for the signs of toxicity which are rapid breathing and pulse, staggering, muscle tremors and dilated pupils.
7. Call your veterinarian immediately if you observe signs of nitrate toxicity. Remember treatment is available and to wait could mean several dead animals.

Home Treatment Alternatives for the Removal of Nitrate from Drinking Water

William A. Lee, Public Health Engineer
State Health Department
Deon D. Axthelm, Water Resources Specialist
Institute of Agriculture and Natural Resources

Nitrate can be removed from drinking water by three methods: **distillation, reverse osmosis, and deionization.** Home treatment equipment utilizing these processes is available from several manufacturers. The decision to buy a home treatment unit should not be made on the basis of a field test for nitrate in the water supply, but should be based upon an analysis by a reputable laboratory. Samples may be submitted to the State Health Department Laboratory or to one of several commercial laboratories.

Distillation

The **distillation** process involves heating the water to boiling and collecting and condensing the steam by means of a metal coil. Most impurities remain in the heating tank. Nitrate reduction of up to 99% can be obtained through this process.

Merely boiling water will *increase* rather than *decrease* the nitrate concentration. Pure water is obtained by collecting and condensing the steam which is generated as water is boiled.

The type of container used to store distilled water is important. First, the container must be sanitary, otherwise the water will become contaminated. Second, water which has been treated by an efficient, properly operated distillation unit is essentially mineral free. It is, therefore, highly corrosive. The container in which the water is stored must therefore be resistant to corrosion. Stainless steel is commonly used but glass containers may also be used.

Reverse Osmosis

Reverse osmosis, as the name implies, is the opposite to the natural process of osmosis. In osmosis, if water containing a high concentration of mineral impurities is separated from water containing a lower concentration of impurities by a semipermeable membrane, the water from the solution of lower concentration will move

through the membrane to the solution of higher concentration of impurities. In the process of **reverse osmosis** pressure is applied to the impure water forcing the higher concentration water in a reverse direction through the membrane. As the water passes through, the membrane filters out most of the impurities. According to manufacturers' literature, from 85-95% of the nitrate can be removed by this process. Actual removal rates, however, may vary somewhat depending upon the quality of the water.

A disadvantage of this method is that only about 30% of the water entering the reverse osmosis unit is recovered as treated water. The remaining 70% is discharged as waste along with the impurities which have been removed from the product water. The disposal of the waste water must be considered when a reverse osmosis unit is to be installed. Efficiency of a reverse osmosis unit can usually be increased by softening the water before treatment.

Some pressure is required to force the water in the reverse direction through the semipermeable membrane. Most home units operate under a pressure of about 200 lb per sq. inch. A pump included in the unit develops this pressure. There are also units available which operate under normal househould water pressure. These units have a lower nitrate removal efficiency but may be useful when a high level of removal is not required.

Deionization

The process of **deionization** utilizes the principle that impurities in water consist of chemical ions each containing a small electrical charge. The water is passed through a treatment tank filled with a bead-like resin. The resin contains the opposite charge to the impurity to be removed. Ions of opposite charge are attracted to the resin and will remain with the resin as the water passes through the unit. It is usually necessary for nitrate removal to soften water before passing it through a deionization unit.

Establish Need

All of the methods described here for the removal of nitrate are relatively expensive. The need for nitrate removal should be definitely established before investing in equipment. Equipment should be purchased only through reputable dealers and manufacturers.

Regardless of the quality of equipment purchased, it will not perform satisfactorily unless maintained in accordance with the manufacturers recommendations. Impurities must be drained from distillation units and the unit cleaned on the schedule recommended by the manufacturer. A reverse osmosis unit requires that the pump operate at the proper pressure and that the membrane be changed periodically. When deionization units are used, the resin must be changed on schedule. Failure to do so will result in a finished product with a higher nitrate concentration than the raw water. Resin tanks are changed by the manufacturer under a maintenance agreement.

Bottled water (not a home treatment) can be purchased in stores or direct from bottling companies. Users should assure themselves of a nitrate content and the general quality and bacterial purity of the product purchased. In all cases, the product water used must be handled and stored in a manner to prevent contamination.

References

The following list of references is reprinted from Special Report No. 34, The Nitrate Problem, printed by Iowa State University, Ames, Ia.

NITRATE AND NITRITE POISONING

Livestock

1. Bradley, W. H., Eppson, H. F. and Beath, O. A. Livestock poisoning by oat hay and other plants containing nitrate. Wyo. Agr. Exp. Sta. Bul. 241. 1942.
2. Case, A. A. Some aspects of nitrate intoxication in livestock. Jour. Amer. Vet. Med. Assn., 130, April 15, 1957, 323-329.
3. Case, A. A. Cornstalk intoxication. Sheep Breeder, p. 14. 1954.
4. Crawford, R. F. and Kennedy, W. K. Nitrates in forage crops and silages; benefits, hazards, precautions. Cornell Misc. Bul. 37, 1960.
5. Garner, G. B. Learn to live with nitrate. Mo. Agr. Exp. Sta. Bul. 708. 1958.
6. Garner, G. B. Nitrate levels in bovine and porcine serum. Journ. Animal Sci., 19:1322 (Abstract). 1960.
7. Gilbert, C. S., Eppson, H. E., Bradley, W. B. and Beath, O. A. Nitrate accumulations in cultivated plants and weeds. Wyo. Agr. Exp. Sta. Bul. 277. 1946.
8. Hopkins. L. L., Jr., Story, C. D. and Dougherty, F. C. In vitro evaluations of nitrogen fertilized forages and their extracts. Jour. Animal Sci., 19:1306 (Abstract), 1960.
9. Lewis, D. The metabolism of nitrate reduction by rumen microorganisms in vitro. Biochem. Jour. 49:149. 1951a.
10. Lewis, D. The metabolism of nitrate and nitrite in the sheep. I. The reduction of nitrate in the rumen of sheep. Biochem. Jour., 48:175. 195lb.
11. Mayo, N. S. Cattle poisoning by nitrate of potash. Kan. Agr. Exp. Sta. Bul. 49. 1895.
12. O'Dell, L. B., Erek, Z., Flynn, L., Garner, G. B. and Muhrer, M. E. Effects of nitrate containing rations in producing vitamin A and vitamin E deficiencies in rats. Jour. Animal Sci., 19:1280; (Abstract). 1960.
13. Olson, O. E. and Moxon, O. L. Nitrate reduction in relation to oat hay poisoning. Jour. Amer. Vet. Med. Assn., 100:403. 1942.
14. Perez, C. B., Jr. and Story, C. D. The effect of nitrate in nitrogen fertilized hay and fermentation in vitro. Jour. Animal Sci. 19:1311 (Abstract). 1960.
15. Pfander, W. H., Garner, G. B., Ellis, W. C. and Muhrer, M. E. The etiology of "Nitrate Poisoning" in sheep. Mo. Agr. Exp. Sta. Res. Bul. 637. 1957.
16. Simon, J., Sund, J. M., Wright, M. J. and Douglas, F. D. Prevention of noninfectious abortion in cattle by weed control and fer-

tilization practices on lowland pastures. Jour. Amer. Vet. Med. Assn., 135:315. 1959.
17. Simon, J., Sund, J. M., Douglas, F. D., Wright, M. J. and Kowalazyk, T. The effect of nitrate or nitrite when placed in the rumen of pregnant dairy cows. Jour. Amer. Vet. Med. Assn., 135:315. 1959.
18. Sokolowski, J. H., Garrigus, U. S. and Hatfield, E. E. Some effects of varied levels of potassium nitrate ingestion by lambs. Jour. Animal Sci., 19:1295 (Abstract). 1960.
19. Sokolowski, J. H., Garrigus, U. S. and Hatfield, E. E. Nitrate poisoning, A study of possible relationships between nitrate, vitamin A and carotene. Ill. Agr. Exp. Sta. Sheep Day, SH 46. 1960.
20. Tollett, J. T., Becker, D. E., Jensen, A. H. and Terrill, S. W. Effect of dietary nitrate on growth and reproductive performance of swine. Journ. Animal Sci., 19:1295 (Abstract). 1960.

SOURCES OF NITRATES

Nitrates in Plants and Feeds

1. Anderson, A. J. and Spencer, D. Sulfur in nitrogen metabolism of legumes and non-legumes. Austral. Jour. Sci. Res. Ser. B. 3(4):431-449. 1952.
2. ap Griffith, G. The nitrate content of some grass species and strains. Nature, Lond. 182:1099-1100. 1958.
3. ap Griffith, G. Nitrate content of herbage at different manurial levels. Nature, Lond. 185:627-628. 1960.
4. ap Griffith, G. and Johnston, T. D. The nitrogen content of herbage. 1. Observations on some herbage species. Jour. Sci. of Food and Agr. 11:622-626. 1960.
5. ap Griffith, G. and Johnston, T. D. The nitrogen content of herbage. II. Effect of different levels of application of sulphate of ammonia on the nitrate content of herbage. Jour. Sci. of Food and Agr. 11:626-629. 1960.
6. ap Griffith, G. and Johnston, T. D. The nitrogen content of herbage. III. the mineral nitrate content of rape and kale. Jour. Sci. of Food and Agr. 12:348-352. 1961. Rep. Welsh Plant Breed Sta. 1959:94-98. 1960.
7. ap Griffith, G. Studies on the nitrate nitrogen contents of herbage.
8. Azam, Gul and Kolp, B. J. Accumulation of nitrates in several oat varieties at various stages of growth. Agron. Jour. 52:504-506. 1960.
9. Balks, R. and Plate, E. Untersuchungen über den NItratgehalt von Futterpflanzen. Landwirt. Forsch 7:203-211. 1955.
10. Bathurst, N. O. and Mitchell, K. J. The effect of light and temperature on the chemical composition of pasture plants. New Zealand Jour. Agr. Res. 1:540-552. 1958.
11. Berg, R. T. and McElroy, L. W. Effect of 2,4-D on the nitrate content of forage crops and weeds. Can. Jour. Agr. Sci. 33:354-358. 1953.
12. Bradley, W. B., Eppson, H. F. and Beath, O. A. Livestock poisoning by oat hay and other plants containing nitrate. Wyo. Agr. Exp. Sta. Bul. 241, 1940.
13. Breniman, G. W., Neumann, A. L., Smith, G. S. and Jordon, H. A. Nitrate and nitrite contents of corn forages and silages as influenced by nitrogen fertility, seeding rate, and various silage additions. Jour. Animal Sci. 20:684. 1961.
14. Breniman, G. W., Neumann, A. L., Smith, G. S. and Zimmerman, J. E. Factors affecting the nitrate content of forages. Journ. Amimal Sci. 20:926. 1961.
15. Burström, H. Photosynthesis and assimilation of nitrate by wheat leaves. Ann. Roy. Agr. Coll. Sweden. 11:1-50. 1943.
16. Burström, H. The nitrate nutrition of plants. Ann. Roy. Agr. Coll. Sweden. 13:1-86. 1946.
17. Burt, R. F. Some factors influencing the accumulation of nitrate by plants. M.S. thesis, Mann Library, N. Y. State College of Agriculture, Cornell University. 1963.
18. Carey, V., Mitchell, H. L. and Anderson, K. Effect of nitrogen fertilizer on the chemical composition of bromegrass. Agron. Jour. 44:467-469. 1952.
19. Carter, L. P. Effectiveness of inorganic nitrogen as a replacement for legumes grown in association with forage grasses. Ph.D. thesis, Iowa State University Library, Ames, Iowa.
20. Case, A. A. Forage poisoning in Missouri due to excessive amounts of nitrate. Mo. Agr. Exp. Sta. Bul. 652:19-20. 1955.
21. Crawford, R. F., Kennedy, W. K. and Johnson, W. C. Some factors that affect nitrate accumulation in forages. Agron. Jour. 53:159-162. 1961.
22. Doughty, J. L. and Warder, F. G. The accumulation of nitrates in oat straw. Sci. Agr. 23:233-236. 1942.
23. Ferguson, W. S. and Terry, R. A. The effect of nitrogenous fertilizers on the non-protein nitrogenous fraction of grassland herbage. Jour. Agr. Sci. 48:149-152. 1956.
24. Finnell, H. H. Factors affecting the accumulation of nitrate nitrogen in high plains soils. Okla. Agr. Exp. Sta. Bul. 203. 1932.
25. Flynn, L. M., Gehrke, G. W., Muhrer, M. E., Smith, G. E. and Zuber, M. S. Effects of temperature, rainfall and fertilizer on yields and composition of corn plants, with special reference to toxic nitrate levels. Mo. Agr. Exp. Sta. Res. Bul. 620. 1957.
26. Garner, C. B. Learn to live with nitrates. Mo. Exp. Sta. Bul. 708. 1958.
27. Gilbert, C. S., Eppson, H. F., Bradley, W. B. and Beath, O. A. Nitrate accumulation in cultivated plants and weeds. Wyo. Agr. Exp. Sta. Bul. 277. 1946.
28. Gordon, C. H., Decker, A. M. and Wiseman, H. G. Some effects of nitrogen fertilizer, maturity and light on the composition of orchardgrass. Agron. Jour. 54:376-378. 1962.
29. Hageman, R. H. and Flesher, D. Nitrate reductase activity in corn seedlings as affected by light and nitrate content of nutrient media. Plant Physiol. 35:700-708. 1960.
30. Hageman, R. H., Flesher, D. and Gitter, A. Diurnal variation and other light effects influencing the activity of nitrate reductase and nitrogen metabolism in corn. Crop Sci. 1:201-204. 1961.
31. Hageman, R. H., Cresswell, C. F. and Hewitt, E. J. Reduction of nitrate, nitrite and hydroxylamine to ammonia by enzymes extracted from higher plants. Nature 193:247-250. 1962.
32. Hagerman, R. H., Zieserl, J. F. and Leng, E. R. Levels of nitrate reductase activity in inbred lines and F_1 hybrids in maize. Nature 197:263-265. 1963.
33. Hanway, J. J. and Englehorn, A. J. Nitrate accumulation in some Iowa crop plants. Agron. Jour. 50:331-334. 1958.
34. Hanway, J. J. Corn growth and composition in relation to soil fertility. III. Percentages of N, P and K in different plant parts in relation to stage of growth. Agron. Jour. 54:222-229. 1962.
35. Hewitt, E. J., Agarwala, S. C. and Williams, A. M. Molybdenum as a plant nutrient. VIII. The effects of different molybdenum levels and nitrogen supplies on the nitrogenous fractions in cauliflower plants grown in soil culture. Jour. Hort. Sci. 32:34. 1957.
36. Kretschmer. A. E., Jr. Nitrate accumulation in Everglades forages. Agron. Jour. 50:314-316. 1958.
37. Mayo, N. S. Cattle poisoning by potassium nitrate. Kan. Agr. Exp. Sta. Bul. 49, 1895.
38. Muhrer, M. E. and Pfander, W. H. Forage poisoning in Missouri due to excessive amounts of nitrate. Mo. Agr. Exp. Sta. Bul. 52:19-20. 1955.
39. Muhrer, M. E., Case, A. A., Garner, G. B. and Pfander, W. H. Toxic forages produced in drought area. Jour. Animal Sci. 14:1251. 1955.
40. Muhrer, M. E., Case, A. A., Garner, G. B. and Pfander, W. H. Forage poisoning in Missouri due to excessive amounts of nitrate. Mo. Exp. Sta. Bul. 652. 1955.
41. Nightingale, G. T., Schermerhorn, L. G. and Robbins, W. R. Some effects of potassium deficiency on the histological structure and nitrogenous and carbohydrate constituents of plants. N. J. Agr. Exp. Sta. Bul. 499. 1930.
42. Nowakowski, T. Z. The effect of different nitrogenous fertilizer, applied as solids or solutions, on the yield and nitrate-N content of established grass and newly sown ryegrass. Jour. Agr. Sci. 56:287-292. 1961.
43. Olson, O. E. and Whitehead, E. I. Nitrate content of some South Dakota plants. Proc. S. Dak. Acad. Sci. 20:95-101. 1940.
44. Peterson, W. H., Burris, R. H., Sant, R. and Little, H. N. Production of toxic gas (nitrogen oxides) in silage making. Agr. and Food Chem. 6:121-126. 1958.
45. Scalleti, J. V., Gates, C. E., Briggs, R. A. and Shuman, L. M.

Nitrogen dioxide production from silage. I. Field survey. Agron. Journ. 52:369-372. 1960.
46. Scharrer, K. and Siebel, W. Über der Einfluss der Einähsung und Belichtung auf den Nitratgehalt von Futterpflanzen, Landw. Forsch 9:168-178. 1956.
47. Simon, J., Sund, J. M., Wright, M.J. and Winter, A. Prevention of non-infectious abortion in cattle by weed control and fertilization practices on lowland pastures. Jour. Amer. Vet. Med. Assn. 135:315-317. 1959.
48. Spencer, Donald. The reduction and accumulation of nitrate. Handbuch des Pflangenphysiologie pp. 201-211. 1948.
49. Stahler, L. M. and Whitehead, E. I. The effect of 2,4-D on potassium-nitrate levels in leaves of sugar beets. Science 112:749. 1950.
50. Sund, J. M. and Wright, M. J. Weeds containing nitrates cause abortions in cattle. Agron. Jour. 49:278-279. 1957.
51. Whitehead, E. I. and Moxon, A. L. Nitrate poisoning. S. Dak. Agr. Exp. Sta. Bul. 424. 1952.
52. Whitehead, E. I., Viets, F. G., Jr. and Moxon, A. L. Nitrogen distribution in the corn plant. S. Dak. Agr. Exp. Sta. Tech. Bul. 7. 1948.
53. Whitehead, E. I. and Olson, O. E. Factors affecting the nitrate content of plants. Proc. S. Dak. Acad. Sci. 21:67-72. 1941.
54. Whitehead, E. I. Nitrate poisons in silage. S. Dak. Farm and Home Research 12(3):8. 1961.
55. Whitehead, E. I., Kersten, J. and Jacobsen, D. The effect of 2,4-D spray on nitrate content of sugar beet and mustard plants. Proc. S. Dak. Acad. Sci. 35:106. 1956.
56. Wilson, J. H. Nitrate in plants: Its relation to fertilizer injury, changes during silage making, and indirect toxicity to animals. Jour. Amer. Soc. Agron. 35:279-290. 1943.
57. Wright, Neal, Trautman, R. J. and Streetman, L. J. Nitrate accumulation in blue panicgrass. Agron. Jour. 52:671-672. 1960.
58. Wright, N. and Trautman, R. J. Influence of management on nitrate accumulation in blue panicgrass. Agron. Jour. 54:363-364. 1962.
59. Zieserl, J. F., Jr., and Hageman, R. H. Effect of genetic composition on nitrate reductase activity in maize. Crop Sci. 2:512-515. 1962.
60. Zieserl, J. F., Ravenbark, W. L. and Hageman, R. H. Nitrate reductase activity, protein content and yield of four maize hybrids at varying plant populations. Crop. Sci. (in press) Feb. 1963.

Nitrates in Water

1. Betke, K. and Kleihourer, E. Water containing nitrate as a cause of methemoglobinemia. (German.) Water Poll. Abst., 33:113. 1960.
2. Borts, I. H. Water-borne diseases. Amer. Jour. Pub. Health. 39:974-978. 1949.
3. Bosch, H. M., Rosenfield, A. B., Huston, R., Shipman, H. R. and Woodward, R. L. Methemoglobinemia and Minnesota well supplies. Jour. Amer. Water Works A. 42: pp. 161-170, July 1950.
4. Burden, E. H. W. J. The toxicology of nitrates and nitrites with particular reference to the potability of water supplies. The Analyst 86:420. 1961.
5. Caballero, P. J. Discussion sober las normas de calidad para aqua potable. Organo Official de la Associacion Interamericana de Ingenieria Sanitaria 3: pp. 53-64. July 1949-June 1950.
6. Campbell, W. A. B. Methemoglobinemia due to nitrates in well water. Brit. Med. Jour., 2: pp. 371-373 (1952).
7. Case, A. A. The nitrate problem. National Hog Farmer. March 1963.
8. Chapen, F. J. Methemoglobinemia from nitrates in well water. Jour. Mich. State. Med. Soc. 46:938. 1947.
9. Chute, W. D. Cyanosis in an infant caused by nitrates in well water. Jour. Mo. State Med. Assn. 47:42-45. 1950.
10. Comly, H. H. Cyanosis in infants caused by nitrates in well water. Jour. Amer. Med. Assn. 129:112-116. 1945.
11. Donahoe, W. E. Cyanosis in infants with nitrates in drinking water as cause. Pediat. 3: pp. 308-311. 1949.
12. Garner, R. J. Veterinary toxicology. Second Ed. Williams and Wilkins Company. p. 111. 1961.
13. Holm, L. W., Johnson, L. F. and Critchlow, J. K. Experimental poisoning of cattle and sheep with dynamite. Cornell Vet. 42:91. 1952.
14. Horn, K. Uber gesundheitsstorungen durch nitrathaltiges trinkwasser vornehmlich bei saughlinger unter berucksichtigung der orthsygienischen Verhaltnisse. Stadchygiene 9: pp. 2-21-25. 1958.
15. Johnston, G., Kurz, A., Cerny, J., Anderson, A. and Matlock, G. Nitrate levels in water from rural Iowa wells. Jour. Iowa Med. Soc. 36:4-7. 1946.
16. Johnston, R. A. The incidence of nitrates in rural Ontario well waters. Can. J. Pub. Health 46:30. 1955.
17. Maxcy, K. F. Relation of nitrate nitrogen concentration in well water to the occurrence of methemoglobinemia in infants. U. S. Armed Forces Med. Jour. 1:1007-1015. 1950.
18. Rosenfeld, A. B. and Huston, R. Infant methemoglobinemia in Minnesota due to nitrates in well water. Minn. Med. 33:787-796. 1950.
19. Sollman, T. S. A manual of pharmacology. Sixth ed. W. B. Saunders Company. p. 825. 1942.
20. U. S. Dept. of Health, Education and Welfare, Pub. Health Service, Drinking water standards. Pub. Health Service Pub. No. 956. 1962.
21. Walton, G. Survey of literature relating to infant methemoglobinemia due to nitrate-contaminated water. Amer. Jour. Pub. Health 41: pp. 986-996, August 1951.
22. Waring, F. H. Significance of nitrates in water supplies. Jour. Amer. Water Works Assn. 41:147-150. 1949.

Testing for Nitrates

1. American Public Health Assn., Inc. Standard methods for the examination of water, sewage, and industrial wastes. APHA, 1790 Broadway, New York 19, N. Y.
2. Bray, R. H. Nitrate tests for soils and plant tissues. Soil Sci. 60:219-221. 1945.
3. Garner, G. B., Baumstark, J. S., Muhrer, M. E. and Pfander, W. H. Microbiological determination of nitrate. Anal. Chem. 28:589. 1956.
4. Jackson, M. L. Soil chemical analysis. Prentice-Hall, Inc., Englewood Cliffs, N. J. 1958.
5. Johnson, C. M. and Ulrich, A. Analytical methods for use in plant analysis. Calif. Agr. Exp. Sta. Bul. 766. 1959.
6. Johnson, C. M., Ulrich, A. Determination of nitrate in plant material. Anal. Chem. 22:1526-1529. 1950.
7. Keeney, D. R. and Bremmer, J. M. Determination of inorganic forms of nitrogen in soils: I. Exchangeable ammonium, nitrite, and nitrate. (To be submitted for publication in The Soil Sci. Soc. Amer. Proc.)
8. Kitchen, H. B., editor. Diagnostic techniques for soils and crops. Washington, D. C., American Potash Institute. 1948.
9. Nelson, J. L., Kurtz, L. T. and Bray, R. H. Rapid determination of nitrates and nitrites. Anal. Chem. 26:1081-82. 1954.
10. Taylor, E. W. Examination of water and water supplies. The Blakiston Co., Philadelphia, Pa.
11. Ward, G. M. and Johnston, F. B. Chemical methods of plant analysis. Canada Dept. Agr. (Research Branch, Ottawa, Ontario) Pub. 1064. 1960.

The Cooperative Extension Service provides information
and educational programs to all people without regard
to race, color or national origin.

Issued February 1981, 3,500
Reprinted October 1981, 5,000