

Nitrate Poisoning  
And  
Feeding Nitrate Feeds  
To  
Livestock

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## Introduction

Nitrate poisoning has been a concern for Alberta livestock producers for many years. Every year there is a report about a farmer or rancher who has lost animals to nitrate poisoning. However, almost all feeds containing nitrate can be safely fed to livestock if handled properly. Understanding why nitrate accumulates in plants and how nitrates affect livestock, are keys to managing the feed supply and developing a safe feeding program.

### 1.0 **Mechanism of Nitrate Poisoning**

Nitrates ( $\text{NO}_3^-$ ) themselves are not very toxic but nitrites ( $\text{NO}_2^-$ ) which they are converted to. In ruminant animals such as cattle, sheep and goats, the conversion of nitrate to nitrite is carried out by rumen bacteria. Nitrate that has been converted to nitrite is then converted to ammonia. Ammonia arising from the breakdown of the feed and from the metabolism of nitrite is converted to urea and either recycled to the digestive tract or voided in the urine. Very little ammonia is lost by belching. After an animal consumes feed that contains nitrate, rumen ammonia levels may increase significantly and it is unusual to have blood ammonia levels increase (Kemp 1977). When the nitrate – nitrite - ammonia cycle is in balance, there is no build up of nitrates in the rumen.

In the rumen, the reduction of nitrate to nitrite occurs rapidly whereas the detoxification of nitrite is a slower process. Initial development of poisoning occurs when the nitrite level in the rumen exceeds the microbe's capacity to convert it to ammonia. When this happens, rumen nitrate and nitrite are absorbed across the rumen wall into the bloodstream. When nitrite crosses the rumen wall and enters the bloodstream, it combines with haemoglobin in the red blood cells and forms methemoglobin. When methemoglobin forms, the ferrous ion of haemoglobin is converted into the ferric form and the capacity of the red blood cells to carry oxygen to body tissue is reduced. Nitrate in the bloodstream does not create toxicity problems initially, but can be recycled back into the rumen via saliva or gastro-intestinal secretions. The recycled nitrate can be converted to nitrite for reabsorption into the bloodstream (Pfister et al 1988).

Refer to Figure #1

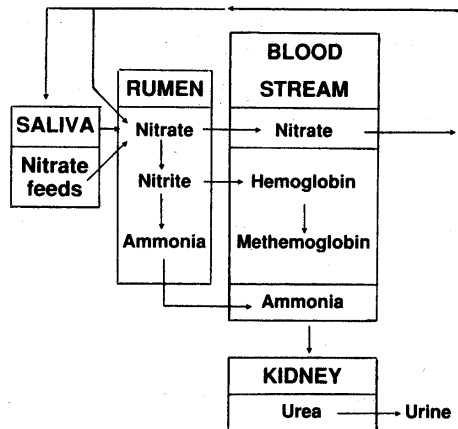


Figure 1. Nitrate Pathway In Ruminants

In contrast, monogastric animals like horses and pigs, conversion of nitrates to nitrites occurs in the intestine, closer to the end of the digestive tract. In this situation there is a less opportunity for the nitrites to be absorbed. It is this difference in site of conversion that makes nitrite poisoning much less of a concern with monogastrics than it is with ruminants.

The rate of conversion of hemoglobin into methemoglobin is directly affected by the transfer of nitrite from the rumen into the bloodstream, which is influenced by:

- rate of nitrate intake (amount of feed and how quickly it is consumed),
- rate of digestion of feed and subsequent release of nitrates,
- rate of conversion of nitrite to ammonia in the rumen, and
- movement of nitrite out of the rumen.

These factors influence the toxic nitrate intake level for each animal. Individual animals have different levels of tolerance to nitrates. This is reflected in the between animal variability in the amount of methemoglobin that can form before production or reproduction is affected, or death losses occur. Animal variability means that there is no single nitrate value that can be given to indicate what the toxic nitrate level is for a specific animal.

The release of plant cell contents into the rumen is dependent upon the digestibility of the ingested plant material, interference in the nitrate – ammonia pathway, and pH of the rumen. Highly digestible plant material such as ammonia pathway, and pH of the rumen. Highly digestible plant material such as young leaves will break down more rapidly than mature stems, thus a quicker release of nitrate into the rumen will occur. If one of the steps in the pathway in the reduction of nitrate to nitrite and finally to ammonia is blocked or is working to capacity and a substance or product is accumulating, this increases the amount of nitrite in the rumen and absorption through the rumen wall. Maximum nitrate reduction occurs when the rumen pH is 6.5 and nitrite reduction is maximal at pH 5.6. Thus, when rumen pH is favourable for nitrate reduction, nitrite appears very quickly. (Geurink, et al., 1979; Johnson et al., 1983).

Healthy animals have normal methemoglobin levels that are relatively constant at 2-3% of total haemoglobin. When high nitrate feeds are consumed, moderate nitrate poisoning symptoms appear when 20-40% of the hemoglobin is converted to methemoglobin (Johnson, 1983, Pfister, 1988), and severe symptoms or death can occur when blood methemoglobin levels rise to 67-90% (Asbury, et al. 1964).

## 2.0 Interpreting Nitrate Test Results

Different laboratories use different methods to test for nitrate content in feeds. An interpretation guide is necessary for sound management decisions. Table 1 lists the different methods of analysis and associated risk factors involved.

<b>TABLE 1 METHOD OF NITRATE ANALYSIS AND DATA REPORTING</b>				
Category	% N0 <sup>3</sup>	%N0 <sup>3</sup> -N	%KN0 <sup>3</sup>	Remarks
1	0.5	0.12	0.81	Generally safe
2	0.5 – 1.0	0.12 - -0.23	0.81 – 1.63	Caution
3	1.0	0.23	1.63	High Nitrate Problems!

The values quoted above are on a dry (moisture free) basis.

Conversions: 1 N0<sup>3-</sup> N = 4.43 N0<sup>3-</sup> = 7.22 KN0<sup>3</sup> = 6.07 NaN0<sup>3</sup>

1 N0<sup>2-</sup> N + 3.29 N0<sup>2</sup> = 6.08KN0 = 4.93 NaN0<sup>2</sup>

Note: In category 2, some subclinical symptoms may appear.

In category 3, death losses and abortions can occur.

In the remainder of the paper, the term nitrate (N0<sup>3-</sup>) will be the reference unit used.

## 3.0 Acute Nitrate Poisoning

Acute nitrate poisoning occurs when nitrate is rapidly released from plant material and converted to nitrite in the rumen. Large amounts of nitrite are immediately transported across the rumen wall into the bloodstream.

The methemoglobin formation curve is sigmoid ("S" shaped). A small increase in nitrate consumption rate or diet concentration creates a large increase in blood methemoglobin levels. Increases of methemoglobin occur more rapidly than decreases (Crawford, et al., 1965). See Figure 2.

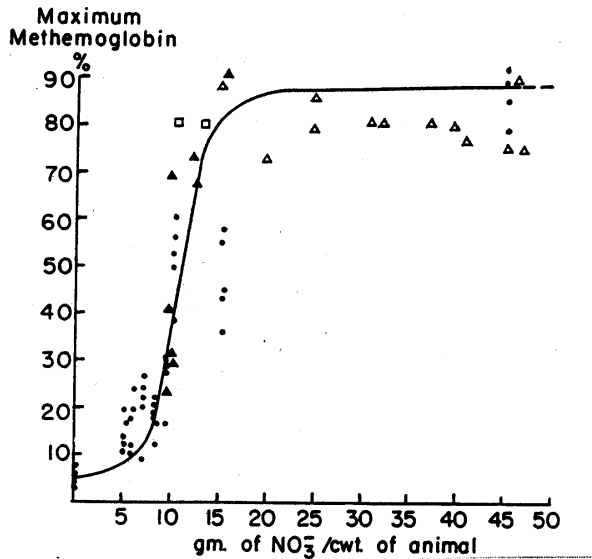


Figure 2. The sigmoid methemoglobin response curve obtained by drenching cattle with increasing levels of nitrate. (Cornell data, 1959: Bradley et al., <sup>2</sup> ▲ Stewart and Merilan, <sup>2</sup> 4 and ▼ Dodd and Corp. 7.)

When methemoglobin forms, the body's smooth muscles relax (including the smaller blood vessels) causing a drop in the animal's blood pressure. An increase in heart rate usually offsets the drop in blood pressure and brings conditions back to normal. A lack of oxygen supply to body tissues (asphyxia) is the main cause of death (Asbury et al. 1964).

Acute poisoning problems are intensified by tubular reabsorption of nitrite in the kidney which concentrates the nitrite in the blood.

Clinical signs of acute poisoning are: increased heart rate, blue-grey mucous membranes, excess saliva and tear production, weakness, depression, laboured or violent breathing, staggered gait, frequent urination, vomiting, muscle tremors, low body temperature, disorientation and an inability to get up. Animals are found in a lying position after a short struggle. Feed intake in feedlot steers is reduced in sub-acute poisoning situations. When acute poisoning occurs, most animals are found dead before any signs of toxicity are observed. Death usually occurs within a few hours of feeding (Jainudeen, 1964, Burrows, 1987).

#### 4.0 Chronic Nitrate Toxicosis

Reduced weight gains, lowered milk production, (Jainudeen, et al. 1964) depressed appetites (Ferra, et al. 1971) and increased susceptibility to infections (Johnson, et al. 1983) are symptoms associated with diets containing moderate levels (0.5 – 1.0%) of nitrate. These production related problems or losses are not recognized in many operations. Since animals suffering from chronic nitrate poisoning may not show any obvious signs of distress.

Nitrates reduce thyroid gland activity and therefore, the metabolic rate of the animal. A lower thyroid activity level causes a depression in appetite and lower weight gains (Johnson, et al. 1983). In rats, lower thyroid activity reduces Vitamin A levels in the liver (Jainudeen, et al. 1964), but these conditions do not appear to affect cattle as long as dietary iodine levels are sufficient in the diet to maintain normal thyroid activity level.

Animals that exhibit signs of distress from chronic toxicosis may appear uncoordinated, belligerent, or have uncoordinated hind quarter movement (Smith, 1990) Diarrhea, eye lesions and silent estrus have been noted (Burrows, et al., 1987 Mittal, 1986). These "silent" heat cycles may be missed by herd bulls (Page, 1987). Subclinical (chronic) nitrate abortion is a complex process of interactions between maternal – fetal circulation, trace mineral and enzyme interactions (controlled by quantity and quality of feedstuffs), absorption time, rumen pH, rate of nitrate release from the feed and conversion rate of nitrate to nitrite (Johnson, et al. 1983). Almost all nitrate related reproduction problems are not recognized as a feed-related problem.

Feeding diets containing moderate levels of nitrate to dairy cows does not change milk protein or fat content. Feed consumption may decrease and a drop in milk production results. In one feeding trial done by Farra, (1971) cows placed on a 2% nitrate diet for 16 weeks produced 4 kg/day less milk and lost 20 kg of weight compared to the control animals. The control animals fed a nitrate free diet over the same stage of lactation gained 13 kg of weight.

Work done in Indiana shows that drought stressed corn silage/soybean mix containing high nitrate levels when fed to cattle resulted in a 4.5% improvement in average daily gain, but a reduction in feed conversion efficiency of 18.2% when compared to a normal corn silage. The drought stressed corn contained 85% less corn grain than normal silage, thus a lower energy level was present in the diet (Perry, 1984)

Reproductive problems which occur because of nitrate toxicity generally are due to an induced hormonal imbalance although other factors may be involved. High nitrate rations reduce progesterone levels in cycling cows and in early pregnancy (Smith, 1990). Nitrate interferes with the implantation of the egg in the uterus. When implantation does not occur, the fetus dies and is reabsorbed by the cow. Nitrate-related abortions occur within the first 100 days of pregnancy. During the first trimester of pregnancy, no obvious signs of an abortion are seen.

Nitrate related abortions can also occur in late pregnancy and may be directly caused by fetal oxygen deficiency or interference with nutrient transfer across the placenta. Ingested and absorbed nitrite also lowers blood pressure, and reduces blood flow (Asbury, 1964). This reduces the amounts of oxygen being transferred across the placenta and compounds the problem of less oxygen being supplied to the calf. To combat this problem, the cow temporarily stops

uterine contractions to aid in oxygen transfer to the fetus (Johnson, et al. 1983). At 177 days (six months) of pregnancy, 14% of the cardiac output of the cow goes to supply the uterus with blood. Of the oxygen supplied to the uterus, 78% is used to support uterine and placenta functions. Only 22% of the oxygen in uterine blood is for the fetus. Nitrate poisoning will reduce the amount of oxygen in the blood (caused by increased methemoglobin levels) and will cause the in utero calf to become oxygen deficient and death can result.

Fetal haemoglobin may be more easily oxidized to methemoglobin than maternal blood. Methemoglobin levels that appear to be acceptable for cows may be toxic to the in-utero calf. Clinical signs of nitrate poisoning are not apparent in an aborted calf (late pregnancy) or a calf that is born one or two weeks early (Johnson, et al. 1983).

Symptoms of nitrate toxicity in calves born live are that they are born 1-4 weeks early and often die within 18-24 hours of birth. Convulsions and seizures are associated with nitrate poisoning. A post-mortem examination of these apparently normal calves sometimes reveal heart defects and joint immobility. Hemorrhaging problems in the heart and trachea have been observed. Also, general vascular, ruminal, and abomasal congestion occur. Blood is dark red to coffee brown in color if examined within 1 – 2 hours of death (Blood and Radostits, Vet medicine, 7<sup>th</sup> Edition, 1989).

A study conducted in Nebraska (1982-83) examined 277 calves that were born dead or died shortly after birth. Thirty percent of the calves had some nitrate present in the eye fluid and 20% had indications of excessive exposure to nitrates (Johnson, et al. 1983). This study confirmed that nitrate poisoning of the calf does occur without causing death to the cow.

Lambs born to ewes fed a nitrate diet have reduced birth weights and have more problems suckling at birth. Higher lamb mortality rates and an impaired ability of the newborn to seek a teat have been cited (Johnson, et al. 1983).

#### 4.1 **Non Feed Sources**

Non feed commodities commonly found on the farm can create nitrate toxicity problems. Ammonium nitrate and urea fertilizers, have been implicated in poisoning cases. As cattle graze pastures, or forage around buildings, they will locate fertilizer spills and quickly consume the material.

##### 4.1.1 **UREA/ Ammonia Poisoning**

When urea fertilizer is consumed, the urea molecule is broke down into two ammonia units. Rumen and blood ammonia levels increase dramatically within 20 – 30 minutes of consumption. It is the blood ammonia concentrations that

cause the toxicity problems. Symptoms of ammonia fertilizer poisoning are animals that; bellow, appear to have sunken eyes, and the skin loses its elasticity due to dehydration, high temperatures, laboured respiration, muscle tremors or tetany, and a fluid filled rumen (pH of 8 – 9) also occur. Liver and heart damage has been reported (Horner, 1982). It is generally agreed that urea toxicity is equivalent to ammonia poisoning (Shirley, 1986)

Ammonia toxicity affects the red blood cell. While nitrites prevent the red blood cells from carrying oxygen to body tissue, ammonia toxicity prevents the release of carbon dioxide from the red blood cells. The antidote for a mature cow suffering from ammonia toxicity is the oral administration of 4L vinegar. This treatment may need to be repeated every 20-30 minutes until the symptoms disappear (Horner, 1982).

Toxicity problems are not usually associated with the ingestion of protein, but rather with ingestion of excess levels of urea. The utilization of ammonia depends upon the growth rate of ruminal microbes and is usually limited by the availability of readily fermentable carbohydrates (ie: grains).

#### **4.12. Nitrate Fertilizers**

Ammonium nitrate fertilizers are more complex in their mode of action. The ammonium portion, is immediately available in the rumen and is passed across the rumen wall. Blood ammonia levels increase very shortly after consumption. The nitrate fraction, accumulates in the rumen and can be converted to nitrite before crossing the rumen wall, creating nitrate poisoning conditions. The treatment for ammonium nitrate poisoning is a combination of vinegar (orally) and methylene blue (injected).

#### **5.0 Adjustment to nitrates in Feeds**

Ruminants are more susceptible to nitrate poisoning than monogastics. Sheep have the highest tolerance to nitrate poisoning, because they have the highest capability of all ruminants to reconvert methemoglobin to haemoglobin (O'Hara, et al. 1975). Differences between animals is also due to different levels of nitrate reductase enzyme activity (Pfister, 1988, Sinclair, 1964) in the rumen microbial population. Nitrate reductase is the enzyme responsible for the conversion of nitrate to nitrite and finally into ammonia. Nitrate reductase activity levels can change over time.

Cattle are not very efficient at converting nitrite to ammonia and therefore have the lowest tolerance to nitrate poisoning.

Individual ruminant animals have different capabilities to convert nitrate into nitrite and finally to ammonia. Different dosages, feed type, adaptation period, and intake interval influence what the toxic level is for a specific animal. Peak

rumen nitrate concentration influences the amount of nitrate or nitrite that will pass through the rumen wall at any given time.

An animal in good body condition, receiving a diet that meets daily nutrient requirements is able to convert nitrate to ammonia more efficiently than an animal that is inadequately fed or in poor condition (Page, 1987). The opposite also holds true; an animal in poor condition but well fed is more susceptible to nitrate poisoning.

Microorganisms are able to increase nitrate reductase activity in the rumen three to five times the normal levels, but a three to five day acclimatization period is required. If an animal is adjusted to a low nitrate level in a feed and a sudden increase to high nitrate occurs, an increase in nitrate conversion to nitrite can be expected (Pfister, 1988).

Microbes are also able to increase the rate of nitrite to ammonia conversion as well.

Animals that are accustomed or adapted to a ration that contains nitrate can quickly pass this adaptability to animals that are held in close physical proximity (ie: in the next pen) within 2-3 days. The mechanism is not known, but it does occur (Cheng. Et. Al., 1985).

Rumen flow rates, maximum nitrate concentration and movement of nitrate and nitrite across the rumen wall are part of the nitrate toxicosis process. Day-to-day variation of methaemoglobin formation, and between animal variation causes a range of values for what is considered to be a "safe" nitrate level. Over time, rumen microbes do adjust nitrate content, in feeds, and are able to function well in the new environment.

After a five to six week nitrate feeding period, cattle increase their haemoglobin concentrations by twenty eight percent compared to pre-adaptation blood levels. Hemoglobin is maintained at this concentration as long as the nitrates are present at constant levels in the ration. When nitrates are removed from the ration, haemoglobin levels return to original levels three weeks. There are no changes in blood protein levels but calcium, phosphorus and Vitamin A levels decrease as the longer the nitrate diet is fed (Alaboudi, 1985).

High nitrate intake can affect iron containing enzymes other than haemoglobin. As an example; a decrease in aldehyde oxidase reduces serotonin degradation. An accumulation of brain catecholamine and neurotransmitters may cause mild convulsions or ataxia (Johnson, 1983).

Sheep have the capability to increase red blood cell concentrations in blood. This can result in their higher capability to detoxify nitrate. The variable levels of red blood cells can result in extremely variable amounts of nitrate being absorbed

from the rumen into the bloodstream. Sheep do become acclimatized to nitrates and can adapt to rations that contain nitrate (Diven et al, 1964).

## **6.0 Changes in Rumen Function**

### **6.1 Short Term Adaptation**

Metabolic activity of the microbes controls the rate of rumen function. When potentially toxic dietary components are ingested by a ruminant, these toxins can be destroyed if enough enzymes are present (Cheng, et al. 1985).

Rumen microbial activity remains normal for the first three days after nitrate is introduced, but microbial digestion capability decreases and fibre digestion slows down from day 4 to day 7 when the animals are on all forage diet if 20-30% of the haemoglobin is converted to Methemoglobin. If grain provides a soluble carbohydrate or energy source is being fed, the reduction in microbial activity should not occur (Allison & Reddy, 1982). The additional time that the feed stays in the rumen, because of slower passage rates allows for more nitrate uptake across the rumen wall and more conversion of nitrate to nitrite in the rumen before crossing the wall. Slower passage rates can also predispose the rumen to ketosis or digestive upsets. However, after day 7, the microbes adjust to the new conditions and digestion rates return to normal (Jamieson, 1958).

Nitrate levels in a feed that are considered safe on the first day of a feeding program may not be suitable in the next few days of the initial adaptation period but will be safe again after rumen activity levels return to normal. Nitrate accumulation rates in the rumen can vary from day to day. As more nitrate is consumed, the total amount in the body increases. Susceptibility to poisoning increases with repeated feedings even though the animal is able to handle higher levels of nitrate (Pfister, 1988).

When nitrate is fed, blood methemoglobin levels rise for 4-5 days and then plateau. This corresponds to an increase of blood nitrate/nitrite levels that gradually stabilizes as well (Kemp, et al. 1977) The reconversion of methemoglobin to haemoglobin takes longer to occur than the original change of haemoglobin to methemoglobin, thus a cumulative effect occurs.

Monensin sodium (Rumensin®) modifies rumen function and shifts volatile fatty acid production in favour of propionic acid in growing and fattening cattle. Specifically, the rumen acetate: propionate rate is reduced, but the butyric acid level remain relatively constant. This reduction in the acetate: propionate ratio caused by the feeding of Rumensin®, along with the further reduction when nitrates are in the ration may be too much for some animals to handle. Changes in the rumen environment (due to nitrates present and Rumensin®) causes a rapid shift in the type of microbes to those which produce nitrites (Rogers, 1980) or increases the passage rates of nitrite into the bloodstream (Malone 1987).

Caution is advised when high nitrate feeds are provided in conjunction with Rumensin®.

Chlortetracycline may provide limited protection for a short period of time. Feeding chlortetracycline at the rate of 22 mg/kg of feed will reduce the amount of nitrate being converted into nitrite for about a week. It is not always effective. This product is only a short term solution. A change in feeding management is required (Emerick, 1961).

## 6.2 Long Term Adaptations

The “safe” nitrate level for an adapted animal is 3-5 times higher than that for an adapted animal (Kemp, 1977, Geurink et al, 1979). Ruminants adapt to the sub-lethal doses of nitrate in the diet by increasing haemoglobin levels, hematocrits and blood volumes (Jainudeen, 1964). The type of roughage (fresh vs dry), and feed composition (hay vs silage) may influence the rumen microbes ability to:

- digest the material
- Change with the nitrate – nitrite- ammonia reduction pattern and which will influence the formation of methemoglobin in the blood (Geurink, et al. 1979)

Inclusion of feeds containing nitrates ( $\text{NO}_3^-$ ) at levels between 0.7 to 4.0% of the diet, affects the production of various volatile fatty acids in the rumen. Animals that are adapted to nitrates in their diet produce less methane, but the same amount of total volatile fatty acids. The proportions of these acids change. The amount of acetate formed increases while propionate and butyrate levels decrease. See Table 2.

	Prior	During	Change	
Acetate	62.3	80.2	+22.3	
Propionate	19.6	14.7	-25.0	
Butyrate	16.1	5.0	-69.0	(Farra, 1971)

## 6.3 Feedstuffs/Feed Type

The rate of feed intake and type of forage being offered, influences whether or not an animal will succumb to nitrate toxicity. The faster an animal consumes an allotment of feed, the greater the amount of haemoglobin converted to methaemoglobin. A dry hay contains less water than a fresh forage and is consumed more quickly (on a dry basis). Dry hay releases nitrate into the rumen more rapidly than fresh forage because the cell walls in the hay are ruptured during drying, allowing for a rapid release of cell contents, including nitrate, into

the rumen. Thirty percent of the nitrate in a fresh forage is released in twenty minutes while eighty percent of the nitrate is released from a dry hay over the same time period (Geurink, et al. 1979).

Specific feeds will also influence the effects of nitrate in a diet. When soybean meal is included in a diet that contains nitrate, the average daily gain for lambs was reduced compared to urea supplemented diet (Clark, 1970). The blood nitrate levels for the soybean supplemented cattle was significantly higher than for the urea-supplemented cattle. The difference of blood nitrate values may indicate that the rumen microorganisms in cattle receiving urea were utilizing nitrate to a greater extent than those cattle receiving soybean meal.

## **7.0 Factors Affecting Plant Nitrate Concentrations**

### **7.1 Nitrogen Uptake**

Soil nitrate level greatly influence [plant nitrate content. Large applications of nitrogen fertilizer or manure increase soil nitrate and thus nitrate availability to the plant. At high soil nitrate concentration surplus quantities of nitrate are taken up and accumulation occurs.

The amount of nitrate used by plants depends on its' metabolic activity level. Nitrates combines with carbohydrates during metabolic processes to form the amino acids glutamine and asparagine. These two amino acids are the base units for the formation of all other plant amino acids and subsequent protein. Nitrate that is brought into the plant accumulates in the stems and leaves when the glutamine and asparagine pathways are saturated (Knott. 1988).

Plant enzymes require near perfect conditions to function at or near maximum capacity. The nitrate-utilization pathway depends on:

- an adequate water supply
- energy from sunlight
- warm temperatures (above 13 degrees C)

If all of the conditions are not optimal, the conversion rate of nitrate into amino acids is impaired. The plant usually uses up all available plant sugars, and this results in the plant accumulating soluble nitrogen as nitrate and ammonia (Knott, 1988).

Excess nitrates also accumulates in stressed plants. Drought or hot winds put forage under water stress, often resulting in nitrate accumulation. Damage caused by hail and frost, impairs photosynthesis resulting in an excess of nitrates. Cool cloudy weather can also create a problem. When any of these conditions exist within a few days of harvest or grazing, the potential for nitrate

poisoning exists. If the stress is removed and the plants recover, nitrate levels should return to normal within several days.

Cereal hays are associated with nitrate problems. These crops are usually planted into well fertilized, manured or recently plowed/broken grass land or pasture. These crops are also harvested at an early stage of development (milk to dough stage) when they contain the highest nitrate content.

Nitrate concentration vary in different parts of the plant. The highest nitrate levels are found in the lower one third of the stalk or stem. Concentrations in the leaves and flowers are lower and the seeds or grain kernels are usually nitrate free (Knott, 1988).

### **7.2 Plant Maturity**

After seeding, much of the nitrate taken up by the plant is used for root and shoot development. During the initial growth of the stand, 40% of the dry matter is retained as root material. At this stage of growth, the roots are able to take up more nitrate than is required and it accumulates in the plant stem and leaves. As the plant grows and matures, the top growth develops and the plant is able to convert more nitrate to amino acids. This change in forage to root ratio improves the nitrogen balance and less "surplus" nitrate is found in the plant (Parwinkal, 1976).

### **7.3 Plant Injury**

Crops hurt by frost or hail reduced photosynthetic capacity. After a frost or hail, the roots are unaffected and are able to supply the same amount of nitrate to the upper plant as they did prior to the injury. Frost usually occurs just before sunrise, when plant nitrate levels are the highest. Nitrate accumulates in the plant as long as the roots supply more nitrate than the plant can use. Nitrate concentrations in the plant remain high until the plant totally recovers and is able to utilize the nitrates which are present. If the plant dies, nitrate will remain in the plant material.

### **7.4 Other Factors Affecting Nitrate Accumulation**

Herbicide application may disrupt or interfere with normal plant function, photosynthesis or nutrient movement within the plant. Also livestock prefer the taste of sprayed or dying weeds and will selectively graze for them. These plants can have a high nitrate content (Cited by Westra, et al. 1983).

## 7.5 Nitrate Accumulators

Various plants can become nitrate accumulators if certain conditions exist. Some of the more common accumulators are:

Commerical Crops	Weeds
Barley greenfeed      Wheat greenfeed	Bull thistle      Canada thistle
Canola plants      Rye greenfeed	Fire weed      Kochia
Sugar beet tops	Mustards      Nightshade
Oat greenfeed	Pigweed      Russian thistle
Sorghum	Smartweed      White ragweed
Beet tops	Lambs quarters      Millet
Flax	Wild Sunflower

(Johnson, et al. 1983)

## 8.0 Effects of Ensiling on Nitrates

During the initial ensiling period, nitrate from the fresh plant material is degraded to ammonia, nitrate and nitrous oxides. Plant nitrate reductase enzymes, which are active when the plant is growing, are less active when the pH drops below 7 and are not active below a pH of 5.5. The highest nitrite levels occur when the silage pH is 4.9 to 5.7 (Spoelstra, 1985)

The breakdown of nitrate during the ensiling fermentation is caused by nitrate reductase enzymes, found in Enterobacteria, Clostridia and Lactobacilli species. Enterobacteria is mainly responsible for degradation of nitrate in silage (Spoelstra, 1987). Similarly, Clostridia can reduce nitrite to ammonia but this is only achieved when pH levels are relatively high. Enterobacteria, commonly found on wilted plant material, disappear as the pH drops. Lactobacilli reduce nitrate and cause lactate to be reduced to acetic acid, thus raising the final pH level in the silage. Nitrate reduction in a silage counteracts the acidification process (Spoelstra, 1985) resulting in a poorer quality product.

Some research from the United States indicates that nitrate levels can be reduced by the ensiling process. This can occur but not without an associated cost. The quality of the silage is dramatically reduced before nitrate dissipates. It is more important to ensile a high quality product, than be concerned with a reduction in nitrate content. Relying on a longer ensiling period to reduce nitrate levels in a silage is detrimental to silage quality (more energy and protein is lost from the feed).

The type of plant material being ensiled and management techniques used (fresh cut vs wilted material) influences nitrate losses. When nitrate reduction occurs, it is usually during the first few days of storage when the ensiling process is

starting. Nitrate reduction occurs when silage pH is above 5.0. About seven to ten days is required for the pH to drop below 5.0 in a high quality silage.

Heavy fertilization and thus high nitrate uptake by the plant increases the buffering capability of the forage. Thus it takes longer to obtain a stable product which influences the quality of the preserved forage. Silages that contain high nitrates have a higher final pH. Ammonia, butyric acid and non protein nitrogen levels of the silage increase when nitrates are present in the silage crop.

Crops ensiled with a high soluble sugar content, ie: cereal grains, have a rapid fermentation process. The rapid drop in pH precludes these crops from nitrate reduction during the ensiling process. Checking silage for nitrates when the pit is being filled, will usually provide an accurate indication of plant nitrate content.

In some of the slower fermenting crops, ie: drought stressed corn and grasses, and the unpredictability of nitrate disappearance makes nitrate estimation in silage from the fresh, green material unreliable (unless the green material is of low nitrate content in the first place), (Spoelstra, 1987).

Higher temperature during the ensiling process increases the opportunity for nitrate to be converted to nitrite. A temperature rise is expected during ensiling, but if temperatures remain over 40 degrees C for more than a few days, a check for nitrates and nitrites should be done before feeding any of the silage to livestock. Protein availability in the silage will be lower than normal (high ADIP levels). Lower ensiling temperatures ie: 20 deg C vs 38 degree C increases the amount of time for the pH to drop (6 days vs 2.5 days). When the pH drops below 4.2, nitrate no longer accumulates.

Degradation of nitrate can be complete in poor quality silage, but this is a poor way to manage nitrates. The production of low quality silage creates many more management problems than it solves. Emphasis should be on the production of a high quality silage with nitrate content being a lesser concern.

## **9.0 Mineral Nutrition Changes**

### **9.1 Sulphur**

Sulphur requirements in the ruminant diet are increased when nitrate feeds are in the ration. Nitrates and sulphates are reduced by the same type of bacteria in the rumen, and this increases the competition for reducing electrons. Therefore a limited supply of available electrons in the rumen could restrict the reduction of nitrate and sulphate by the organisms. With rapid increases of nitrate in the rumen, sulphate reduction rate drops considerably. To keep adequate levels of sulphur available for the growth of the bacteria and production of microbial protein, the addition of sulphur (0.1%) to the ration is recommended. This may also help prevent nitrate

toxicity problems. When nitrate levels are 0.4 – 0.8% in the diet, cellulose digestion is reduced, and the addition of 0.1 – 0.4% sulphate-sulphur is required to maintain cellulolytic bacteria activity (Spears, 1977).

### 9.2 **Molybdenum**

If the ration contains nitrate and a high molybdenum (4-8 mg/kg), the sulphur requirements are further increased. Molybdenum inhibits the reduction of sulphate to sulphide and reduces the incorporation of sulphide into microbial cells (Spears, 1977).

### 9.3 **Magnesium/Potassium – Grass Tetany**

Diets low in magnesium (along with manganese, and high in potassium) have been associated with grass tetany problems. Hypomagnesemia or tetany, results when a diet high in protein and low in structural carbohydrate increase the occurrence of tetany. If the total nitrogen: water soluble carbohydrates ratio was greater than 0.3, the chances for tetany problems is large. Nitrates can be part of the total nitrogen content and contribute to the tetany problem. (Ross, 1978)

## 10.9 **Other Nitrate Sources**

### 10.1 **Water**

Nitrate from sources other than plant material can be poisonous. Water runoff from feedlot grounds can be high in nitrite. Nitrite in water is 10 times more toxic to animals compared to nitrate in feeds due to the rapid absorption across the rumen wall into the bloodstream.

Some species of algae are nitrate producers. Water contaminated with these algae species have been implicated (but usually not confirmed) as the cause of cattle deaths.

## 11.0 **Treatment of Nitrate Poisoning**

Veterinarians have various drugs and antidotes that can be given to animals to relieve acute poisoning symptoms. Chronic cases are not cured by the administration of these products (Page, 1987)

Methylene Blue is able to convert methaemoglobin back to haemoglobin. The dosage must be within a specified narrow range otherwise it can intensify the problem. Intravenous injection of methylene blue in saline solution (4%) for horses must be in the 1-2 mg/kg range while cattle and sheep require 20 mg/kg to obtain satisfactory results (Blood et al, 1989).

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