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RAMBLING AROUND THE FARM – NITRATE & NITRITE

By Andy Cowan

It was quite demoralizing when I went to check my stock one evening to find a number of dead animals. They were all quite healthy 6 hours before when I moved them into the new paddock. This happened to me a couple of years ago in 2002 during the last dry spell we had. I had drafted some older hinds from a mob that were to be slaughtered and put them in a paddock close to the yards for easy access. The paddock had a "reasonable amount" of capeweed present after quite a dry winter. I knew a little of the risk of nitrate/nitrite poisoning. The mob of about 90 hinds was in a 2 acre paddock for about 2 days before they were to be taken off to the abattoirs.

As soon as the cull mob was gone, I put the remainder of the herd in this paddock to clean it up. When I checked the mob about 6 hours later 4 of my good hinds were dead. I believed what had happened was that the first mob had selectively grazed the grasses and clover and possibly some of the capeweed. By the time the second mob was introduced to the paddock there was only capeweed left. Logically, the larger, dominant, more assertive hinds rushed to eat whatever looked lush. Having a limited choice of mainly capeweed with very little carbohydrates to balance this intake, they may have been poisoned.

Nitrate can accumulate in many weed and crop and pasture plants. Of the plants listed by Hungerford, capeweed is the plant that I have the most problem with. Combined with other predisposing conditions, nitrate accumulation in plants is a potential danger to grazing ruminant animals. It can cause two different disorders – nitrate poisoning and nitrite poisoning. Nitrates may cause inflammation of the gut when eaten in large quantities, but it is also important as a source of nitrite. Nitrites cause respiratory distress due to interference with oxygenation of blood and death may follow.

The rumen organisms rapidly reduce nitrate to nitrite. Under unusual circumstances, nitrites may build up to levels that are in excess of the body's capacity to convert them to ammonia. Animals can become acclimatized to large concentrations of nitrates if introduced to them gradually. Any sudden increase in nitrate intake can lead to poisoning because of the swamping or modification of the rumen's bacteria making them unable to deal with the high concentration of nitrate. In fact most of this winter I have been cell grazing pasture that would have been over 70% capeweed.

Nitrate in the diet of ruminants is normally broken down by microbial action in the rumen, first to nitrate and then to ammonia which then is converted to protein. Ruminants can tolerate fairly high levels of nitrate in their diet if the intake is spread over the whole of the feeding day and if their diet is also high in readily available carbohydrate, which is needed to fuel the microbial activity in the rumen. If these conditions are not met, the nitrate can accumulate, resulting in poisoning.

Most plants will accumulate nitrates when soil nitrate is high and conditions are not suitable for normal plant growth which would allow the nitrate to be converted quickly to protein. Normally plants contain relatively small amounts of nitrate as such, because the conversions take place fairly rapidly inside the leaf. During periods of drought, the amount of nitrate in the soil can increase greatly because of lack of leaching, reduced uptake by plants and decomposition of organic matter. Check your stock camps for capeweed after a drought. After the drought breaks, nitrate uptake by plants may be high. Predisposing conditions that limit growth and affect nitrate levels in plants can be drought, cloudy weather, cold weather, soils low in soil sulphur and moly, wilted plants, herbicide application and the application of nitrogenous fertilizers or combinations of any of these factors.

Balance is critical in all natural systems. Once nitrogen has entered the soil, it can be present in the following forms – mineral (NH_4 + NO_2 and NO_3); gaseous (N_2 , N_2O , NO, NH_3) and <u>trapped</u> NH_4 in clay minerals. These three combine to supply a relatively insignificant proportion of the total N available to plants.

The main source of nitrogen which is available to plants is derived from <u>organic</u> nitrogen (dead plants, animals and micro-organisms such as bacteria and fungi). One of the critical factors in the transformations that nitrogen undergoes in the nitrogen cycle is the level of energy or carbon available. It is the carbon/nitrogen ratio of the organic matter which determines how the nitrogen is held in the soil and the size of the microbial population in the soil. It is probably important to mention that, as with any natural system, something is always happening. Plant available nitrogen just does not sit around near the plant roots waiting to be absorbed. The products of organic matter breakdown are constantly being leached (I wish), being lost as a gas, or eaten by microbes as well as plants and then being broken down again when the conditions suit. These transformations are called the mineralization/immobilization process. For those of you that apply urea to your soil, you may be interested to know that a healthy permanent pasture, with 4% organic matter in the top 10cm, will contain over 5,000kg/ha total nitrogen.

The signs of nitrate poisoning are diarrhea, salivation and abdominal pain. Nitrates have a direct caustic action in the lining of the gut. The signs of nitrite poisoning are difficulty in breathing, with gasping, rapid pulse and breaths. Animals are weak and trembling and will stagger. Severely affected animals will go down, convulse and die. If conditions are not right for the conversion of nitrite to ammonia in the rumen, nitrite will accumulate and may be absorbed into the blood. The blood levels of nitrite may become sufficiently high to change the haemoglobin in the blood to a form which is incapable of oxygen transport. Tissues then starve of oxygen and signs of respiratory distress and sudden death can occur.

The best policy is to prevent poisoning. Animals can usually cope with grazing pastures that are high in nitrate providing intake is controlled. In practical terms, this means not grazing hungry stock on forages that are potentially dangerous, such as lush capeweed, or fodder crops such as oats or millet. Holding paddocks around shearing sheds often contain lush capeweed because of the high fertility of the soil and infrequent heavy grazing. In areas where grazing management is fairly intense (ie: on dairy farms), animals should be initially given access to safe pastures. When their appetite has been reduced they can, if it is necessary, be moved to high risk pastures later in the day. They are then less likely to gorge themselves on high-nitrate fodder.

Now if you are as confused as I am about animals dying in the paddock, I suggest that when you next talk to your Vet, ask him/her about enterotoxaemia. Clostridial organisms are, for the most part, normal flora. They only become problematic with dietary stress, injury, changes in management, parasitism or other unusual circumstances that set up a favourable growth environment resulting in production of potent toxins. The disease is characterized by sudden death in thrifty, well-fed calves. Some of the risk factors that can exacerbate the potential problem are aggressive eaters, feed change, rumen acidosis and excessive rumen fill which spills over into the intestine its contents of high soluble protein and carbohydrate levels. In fact, my Vet has not seen a case of nitrate poisoning in our area for 20 years. He believes that my problem was more likely than not to be caused by stock eating close to the ground and consuming greater quantities of the clostridial organisms, combined with the change of food intake.

If ever I find an animal that has recently died, I firstly check for physical signs of injury. Over the years I have found hides pierced by small lengths of re-growth of hard antler (some heal and some do not), rumen walls literally pealing and "burnt" (classic acidosis) and cud in the lungs and wind pipe. Rest assured that this sort of death will only happen to your best animals!!

References

Bertram, J.D.; Sneath, R.J.; Taylor, K.M.; Queensland Beef Industry Institute; Mills, B.D. Farming Systems Institute; McKenzie, R.A.; Taylor, J.D. DPI&F's Biosecurity Unit & Reichmann, J.G. Intensive Livestock Institute. *Cyanide (prussic acid) and nitrates in sorghum crops: Risk management.*

Cave, J. *Clostridial Diseases in Livestock*. AG0068. Updated: April 2007. ISSN 1329-8062

English, A.W. Diseases of Deer. Vade Mecum. Series A, Number 11.

Halpin, C; Hides, S. Maffra. *Nitrate and nitrite poisoning of livestock*. February 2002. AG0701. ISSN 1329-8062.

Hungerford, T.G. Diseases of Livestock. 8th Rev. Ed. 1975. McGraw-Hill Book Co. Aust. P/L.

McQuirk, S.M. *Managing Clostridial Diseases in Cattle*. University of Wisconsin, School of Veterinary Medicine.

Prime Fact 415. Nitrate and Nitrite Poisoning in Livestock 4. www.dpi.nsw.gov.au/primefacts

Robson, S. *Nitrate and Nitrite Poisoning in Livestock*. Regional Animal Health Leader, Animal & Plant Biosecurity, Wagga Wagga.

Seawright, A.A. Animal Health in Australia. Volume 2 – Chemical and Plant Poisons. Commonwealth of Australia 1982.