Sulfate Toxicity in Cattle

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It is a hot summer's day in eastern Colorado, and as you are driving around your rangeland to check on the cattle, you notice that several calves seem depressed and do not run away when approached. In fact they seem blind and uncoordinated. Daily temperatures have been in the 90's for the past two weeks, and so you check the water supply to be sure this is not a problem due to water deprivation. The well is working well and water is plentiful. The cattle have been grazing a lot of the kochia weed along with other weeds as the grasses are not as plentiful as earlier in the year.

Your veterinarian, after examining the calves, diagnoses 'Polio' (polioencephalomalacia -PEM) as the cause of the blindness and depressed state of the calves. 'Polio' is often the result of a vitamin B1 (Thiamin) deficiency, and treatment with high doses of thiamin (10-20mg/kg body weight, the initial dose intravenously, followed by intramuscular inject 3 times a day for several days) is effective. However, after three days of treatment with high doses of thiamin the calves are not any better. Clearly there is more to the problem than just a thiamin deficiency!

Polioencephalomalacia in cattle and other ruminants can be due to a dietary thiamin deficiency caused by the ingestion of some plants that have an enzyme that destroys thiamine in the digestive system of the animal, or it is the result of sulfate toxicity. Both a thiamine deficiency and a sulfate toxicity may in combination cause the blindness and neurologic signs. In the scenario described above, the poor response to thiamin treatment suggests that the problem is more likely due to a sulfate toxicity.

Normally sulfate in the animal's diet is utilized by rumen bacteria to produce bacterial protein. Excess sulfate (SO_4) intake in ruminants results in the over-production of hydrogen sulfide by the rumen microflora. This highly toxic gas (H_2S) is inhaled when the animal eructates and is absorbed into the blood stream via the lungs. The sulfides inhibit cytochrome C. oxidase, a critical enzyme in the electron transport system of cells. Consequently, the brain that is highly dependent on energy metabolism, is energy deprived and undergoes malacia or softening, the main component of PEM. Cattle that have high levels of sulfide induced brain damage do not respond to thiamin treatment.

The potential for sulfate poisoning is increased when cattle consume forages, or grains, especially distiller's grain that is high in sulfates. Common annual weeds such as kochia weed thistle, pigweed, Canada thistle and grasses can accumulate significant levels of sulfate. The total sulfate intake can be compounded if the water also has high sulfate levels. The water from many

wells in Eastern Colorado, Kansas and Nebraska can have significant levels of sulfate. Ideally sulfate levels should be less than 1000 ppm for livestock. The total sulfate (sulfur) intake (including water) for cattle should not exceed 0.4% of the dry matter.

In summary, when the weather is hot in late summer and cattle drink more from wells high in sulfate (>1000ppm), and they are grazing weeds that may also be high in sulfate, it is entirely possible that their total sulfate intake exceeds 0.4% and signs of 'Polio' (PEM) develop. Feeding distiller's grains that are high in sulfate, and grains containing Monensin that promotes the formation of hydrogen sulfide in the rumen should be avoided. Treatment with thiamin will be ineffective once high levels of hydrogen sulfide are produced in the rumen. To prevent sulfate toxicity it is important to know the sulfate levels in well water, and provide low sulfate water especially in late summer. Removing cattle from areas where weeds are high in sulfates and monitoring the total sulfate in the ration so as not to exceed 0.4% is the best means of preventing sulfate poisoning.