As more elk are farmed in western Canada it is becoming evident that several diseases recognized in deer in New Zealand are going to be just as important here. One of these is copper deficiency. Reports from New Zealand indicate the elk and elk/red deer crosses are more susceptible to this problem than pure red deer. In Canada, analysis of blood, and liver samples of animals submitted for necropsy, have revealed copper levels that would be considered low in New Zealand. A few cases of probable copper deficiency in elk have surfaced at the Western College of Veterinary Medicine.

Cattle and sheep in western Canada are also susceptible to copper deficiency and it is not surprising that other livestock might show the same problem. Unfortunately we have no data that tell us what the proper dietary intake level of copper should be in elk or other game farm species.

There do appear to be marked similarities between species in the way that copper and other trace minerals in the diet interact and effect one another. Particularly, high dietary molybdenum and sulphur (especially if both are high) have been incriminated in the development of copper deficiency. High iron intake has also been recognized as causing an apparent copper deficiency, even where dietary copper appeared to be adequate.

Copper deficiency in deer species is characterized by being a disease of young adults, rather than calves. This is in sharp contrast to the situation in sheep, in which the disease is seen in new born or young lambs. The deer fetus is known to preferentially take up copper from the dam. In the face of a dietary deficiency it takes some time for the copper to reach inade-quate levels in the body. Alternatively, it may be that there is a seasonal variation in copper content of forage and also uptake, with lowest levels reported in late winter and early spring.

Copper deficiency can occur in two types of situation. The first is the straightforward one in which an inadequate amount of copper is present in the diet. This is probably less than 5-6 parts per million (ppm), and may be less than 10 ppm. The other is a situation in which the dietary copper is in some way prevented from becoming available to the animal. Molybdenum, sulphur and iron all form insoluble complexes with the copper and prevent its absorption by the animal. There may be other elements and factors that also reduce copper intake or absorption.

In the face of a lack of researched dietary needs one can only extrapolate from other sources. The National Research Council recommendation for copper levels in cattle feeds is 10 mg/kg (or ppm) of dry weight. As can be seen from the table below, nowhere in Canada does grass hay exceed this level and only in Saskatchewan does it meet it. Legume hay is consistently slightly below it, and oats are markedly deficient.

<table>
<thead>
<tr>
<th>Feed Copper levels in Canada</th>
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<tbody>
<tr>
<td>(ppm)</td>
</tr>
<tr>
<td>------</td>
</tr>
<tr>
<td>N Br</td>
</tr>
<tr>
<td>Ont</td>
</tr>
<tr>
<td>Man</td>
</tr>
<tr>
<td>Sask</td>
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<tr>
<td>Alb</td>
</tr>
<tr>
<td>BC</td>
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</table>
Diagnosis

Diagnosis of copper deficiency can be made in three main ways. First, the gathering of a herd and individual animal history, as well as diet and water analysis may indicate this problem as a cause of disease. The history will include clinical signs.

Clinical Signs

The most important clinical syndrome associated with copper deficiency is so-called “Enzootic Ataxia”. A number of other clinical signs are attributed to copper deficiency. Of definite importance to elk farmers is the report from Peter Gogan & others of marked antler abnormalities in copper deficient Tule elk in California.

The name Enzootic Ataxia is borrowed from the sheep literature and describes the clinical signs of uncoordinated gait. In sheep the condition is also known as “Swayback”.

![Bull elk with abnormal antler development due to copper deficiency. Photo credit, Dr. Dave Jessup](image)

The disease often has an insidious onset manifested at first by a slightly uncoordinated gait, often most easily seen in the hind limbs. As the disease progresses the animals may not be able to walk in a straight line. If distressed, for instance for yarding, the signs may be even more exaggerated.

Eventually the severely affected animal will be unable to stand, and may assume a sitting position, like a dog. By this time it will have lost considerable weight and may also have become the victim of harassment by other animals in the herd. Bruising and hair loss may be evident.

It is unlikely that the game farmer will have let animal get to this point without some intervention, but the unattended elk will probably continue to lose weight, and as it cannot walk it may eventually die either from starvation or trauma.

In North America there are other signs that have been associated with copper deficiency. Apart from the abnormal antler growth already mentioned, aberrant hoof growth has been associated with a lack of copper in the diet of Alaskan moose.

Clinical signs of copper deficiency
(in increasing severity)

Slight staggering gait (hind limbs esp.)
Increasing "Swayback"
Incoordination
"Dog sitting" posture
Trauma & death

Other signs include:- condition loss, hair loss, abnormal antler growth, abnormal hoof growth, possible "starry" coat and infertility.

Several other signs or symptoms have been linked, sometimes without good evidence, to copper deficiency. These include bone fragility, lameness and joint swellings, ill-thrift, reduced growth rates, rough "starry" coats, sudden death and reduced fertility.

Differential diagnosis

There are a number of other diseases that may show very similar symptoms, and these must be ruled out.

One of the most important of these is infection with the brain worm, *Parelaphostrongylus tenuis*. Continuing research at the University of Alberta, in the laboratories of Dr. Bill Samuel, has shown that this parasitic infection can produce almost identical symptoms. Some of Dr. Samuel's results have not yet been pub-
lished, but it would appear that elk infected with a moderate number of brain worm larvae may linger for a considerable period of time showing marked incoordination that could easily be confused with a copper deficiency.

Another parasite that might produce similar symptoms is the so-called muscle worm *Elaphostrongylus cervi*. This parasite is not recognized on the mainland of the North American continent, although it does occur caribou in Newfoundland. Vigilant quarantine measures will hopefully prevent its introduction. (see GF-S-2 for more information on both of these parasites).

Any other disease that effects the nervous system, or the muscles of locomotion, must be ruled out before a diagnosis of Enzootic Ataxia is made.

Differential Diagnosis

Copper Deficiency in elk

- *P. tenuis* (or *E. cervi*) infection
- Spinal lesion e.g. tumour, abscess
- Muscle or skeletal injury
- Capture myopathy
- White muscle disease
- Others lesions of central nervous system

Tissue levels

Two different tissues are used to aid in the confirmation of a diagnosis of copper deficiency. The first of these is liver. A reliable and safe method of obtaining a liver biopsy from red deer has been described by Dr. Alex Hamilton in New Zealand. Levels under 100 μmol/kg (dry weight) are considered deficient, and under 60 μmol/kg would appear to be a “critical” level at which enzootic ataxia will occur in some animals.

A second tissue is blood. Serum harvested from blood samples has shown that in clinically affected animals copper concentrations are less than 2.5 mmol/L. More importantly Dr. Colin MacIntosh and co-workers have shown that in deer with liver levels under 100 mmol/kg 50% of deer will have serum concentrations less than 8 μmol/L.

These levels are very similar for those published for cattle in which copper deficiency has been recognized.

<table>
<thead>
<tr>
<th>Copper liver &amp; serum levels in cattle</th>
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<tbody>
<tr>
<td>Liver (μmol/kg)</td>
</tr>
<tr>
<td>Deficient</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Serum (μmol/L)</td>
</tr>
<tr>
<td>Deficient</td>
</tr>
<tr>
<td>Normal</td>
</tr>
</tbody>
</table>

Note. If lab reports give levels in parts per million (ppm) the conversion is as follows:

\[ ppm + 63.54 \times 1000 = \mu\text{mol/kg or } \mu\text{mol/L} \]

It is certain that the use of serum copper analysis is not a really reliable test if only small number of animals are to be tested. If a single animal were to show levels below 8 μmol/L, it would be reasonable to assume a deficiency that needed treatment. It is unfortunately possible for a marginally deficient deer or elk to show serum levels above 8. If copper deficiency is suspected a minimum of ten animals should be blood sampled. As the disease is a nutritional one, it is likely that an entire herd will be effected to some degree, so that this is not an unreasonable approach.

Pathology

The third avenue for diagnosis is a pathological examination. The gross post-mortem signs of copper deficiency are unremarkable. There may of course be signs of trauma, and the carcass is usually in poor condition.

Histopathological examination of the central nervous system will confirm the specific lesions of copper deficiency. The myelin sheath around the nerves is reduced to a greater or lesser degree. A post-mortem examination will also provide an opportunity for submission of liver samples for copper analysis.
Prevention & Treatment

As is always the case, prevention is the better of these two options. It is certainly possible to investigate the levels of copper and other elements in the soil, but this alone is of no value in the assessment of a copper deficiency. The reason is that copper in the soil may not be available to the plants. If soil pH is high molybdenum solubility increases, to the detriment of copper uptake. Limestone soils, or areas where repeated lime application has been made tend to have high pH. Some areas of BC and Manitoba are known to be high in molybdenum.

An important feature of any analysis is the testing of well water for certain components. In particular deep wells in Western Canada tend to have high concentrations of sulphates and iron, both of which could adversely effect copper metabolism.

Copper sulphate has been tried in various forms, but is not really recommended. If used in fertilizer, it will be expensive, and more seriously, in areas where the soil is alkaline (many parts of western Canada) the copper will not be available to the plants. Copper sulphate has also been given in the water supply, but palatability may be a problem. Dr. Peter Wilson reports that only about 70% of deer will accept licks to which copper sulphate has been added. Oral drenching with copper sulphate is also ineffective as only a very small quantity is absorbed.

Treatment of severely affected animals is unlikely to be effective. Copper supplementation will not restore damaged nervous tissue, but it may prevent further damage, so that if administered in time it may allow the animal to function more or less normally.

Copper chelate drenches have been described as being effective, and some Canadian feed companies have a organic copper chelates available use in cattle. These are probably more effective than the inorganic chelates, but there is no information on doses appropriate for elk or other game farm species.

In New Zealand the most effective treatment for copper deficiency appears to be dosing with copper oxide or oxidized copper wire particles, usually in a gelatin capsule, which are on the market as a Beecham product called "Copporal". They are not licensed in Canada, but they may possibly be available to veterinarians on an investigators license. In New Zealand they are used routinely to prevent problems from arising.

Another form of copper treatment that has been tried is the use of an injectable copper product. The forms so far tested have produced severe tissue reactions and are not recommended at this time.

If a copper deficiency is diagnosed on a game farm it is critical that regular monitoring of the situation be carried out. The variability in degree of severity, the role of competing elements, and the gaps in our knowledge of the disease all complicate the issue.

Copper poisoning

Excess copper is toxic to most animals. The most commonly recognized type of poisoning is an acute form characterized by salivation, purgation, violent abdominal pain, convulsions, collapse and death. Single lethal doses of copper do not cause a rise in blood or liver copper levels, so diagnosis must be based upon symptoms and history. Dr. Peter Wilson reports one case in which elk drenched with an anthelmintic containing a copper EDTA mixture suffered severe consequences. All the animals had diarrhea for several days, and four animals died.

Chronic, or more properly cumulative copper poisoning is also recognized. The condition has not been reported in game farm animals, but many other species are susceptible, and major losses in sheep have been attributed to the effects of long term exposure to copper. These have occurred after spraying of orchards, consumption of licks, and in the vicinity of copper mines or smelting works.

Symptoms of chronic copper poisoning are characteristic. Particularly, hemoglobinuria (coffee-coloured urine) and jaundice are seen. Liver copper values may be doubled the maximum normal, in excess of 15,000 μmol/kg.

Bibliography:
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