Copper Deficiency in Dairy Goats

Here in southern-most Southern California (San Bernardino, Riverside, Orange and San Diego counties) and most of Arizona we have severe primary copper (Cu) deficiency (low levels in soil and feed) problem in the Swiss breeds and LaMancha dairy goats. As in cattle, we feel that genetic difference in both breeds and blood lines within breeds affects goats susceptibility to both deficiency and toxicity (research shows that Simmental and Charolais cattle need more copper in their diet than Angus, because they are less efficient absorbers of copper from the small intestine. And field experience suggests Simmental, Maine Anjou, Limousin and Charolais benefit from 1.5 times the copper intake of traditional breeds - Dennis Herd, Texas A&M beef cattle nutritionist). At first it appeared that Nubians were holding their own with the available copper, we now have confirmation of copper deficiency in Nubians at the same rate as the Swiss breeds. Deficiency has also been confirmed in the Boer, Pygmy and Nigerian Dwarf breeds in Southern, Mid and Northern California and Arizona. We have little laboratory or other information on the hair breed goats, though there are some old studies and perhaps recent lab work that indicate they may be more sensitive to excess copper than the dairy, miniature, and meat breeds. We know of hypocupric problems in both cattle and horses (foals), and occasionally sheep, in the area as well. In the last decade deficiency has been confirmed (via necropsy and/or laboratory work) in mid-state areas. While we only have a small amount of laboratory and other confirmation of copper deficiency in animals/herds in the high desert areas north of San Bernardino (where they feed mostly locally grown alfalfa) what we do have has shown goats in that area (including Nubians, who account for 95% of the data we do have) to be just as deficient as those in the lower areas. Indications are that in some mid and northern California areas theirs is a secondary deficiency (inducted by high levels of Cu-antagonists in the diet) from an excess in soil molybdenum. NOTE: See lab statistics for local animals (limited area) updated March 2006.

Texas, Washington, Oregon, Missouri, Virginia, Colorado, South Dakota, Ohio, Wisconsin, Indiana, and the most of the New England states also have areas where copper deficiency in dairy, Pygmy, Boer, and Nigerian Dwarf goats has been confirmed.

Sandy soils have traditionally shown deficiencies, but high organic matter soils, degraded black soils, wooded calcareous and grey-wooded soils can also be severely deficient. Copper deficiency may occur when animals graze on soils deficient in copper, soils with high Molybdenum levels (+2PPM); copper intake should be 5 to 8 times molybdenum intake, pastures with high sulphate levels (+0.35% total sulfur), iron exceeding 250 to 300 PPM, or some combination of these. Water (desired level for minerals in livestock drinking water), usually well water or hot springs water, may have substantial amounts of sulfur that reduce availability of copper. Water from alkaline soils is more commonly high in sulfur, which may add to interference. While all minerals can be involved in interactions, the effect other minerals have on the need for copper is more specific and unique than with many of the other minerals.

*Copper’s availability is reduced by iron, sulfur, molybdenum & zinc. The zinc/copper interaction is alleviated to a certain extent by maintaining the zinc:copper between Soil copper is in two forms, Cu²⁺ and Cu(OH)⁺. In general the plant availability of copper in the soil decreases with an increase in pH of the soil. As pH rises adsorption increases and the solubility of the oxides decrease. Deficiencies can occur naturally in soils that are naturally high in pH or have been over limed. The opposite can occur in very acid soils. This is true of all the micronutrients except Selenium and Molybdenum. Studies have found that only 35% of pastures in the US have adequate copper levels.
Only a fraction of ingested copper is absorbed (average 4-5 per cent in adults) and is affected mainly by high levels of molybdenum, which binds with copper to make it insoluble, as well as high levels of iron and zinc.

Due to the large number of interactions, it is important to maintain a balance between trace minerals without over supplementing trace minerals.

Copper content of pasture can vary from spring to fall.

In general, the Western states had lower mean serum copper concentrations compared to other regions. The mean serum copper concentration for operations in the Western regions was 0.63 ppm, while the Midwest and Southern regions recorded 0.70 ppm.

Almost half of Canadian feeds analysed at the Agricultural Soil and Feed Testing Laboratory (Canada) contain less than the estimated RDA of 10 ppm. Also, in the US 28.7% to 57.8% of pastures had molybdenum (Mo) and iron (Fe) levels high enough to cause copper malabsorption. This can be added malabsorption through excessive sulfur intake.

NOTE: Alfalfa is notorious as a crop which is susceptible to copper deficiency. Wheat, barley and oats can also be deficient.

NOTE: Molybdenum is common in alfalfa hays. Copper deficiency is likely if hay has less than four parts copper to each part molybdenum.

NOTE: Soil applied copper will generally have long-lasting residual effects. Beneficial effects from 1.3 to 2.7 pounds of copper per acre have persisted undiminished for up to 35 years (western Australia). Copper can be applied as organic compounds in the form of CuEDTA, copper ligninsulfonates, and copper polyflavonoids.

Copper deficiency in grazing livestock has been recognized in most developed countries especially across Europe and North America as well as in Australia (pioneering work was done in Australia in the 1930s). As far back as the 1930s localized cases of copper deficiency were discovered in Florida, The Netherlands, New Zealand and parts of Australia.

Trace mineral concentrations in forages can vary among regions, within a state and even within a ranch. The following table demonstrates the variation measured among four pastures on a ranch in southwestern Montana. The table below demonstrates the variation in the Mineral Content of forage samples from one Southwestern Montana ranch. Forages were sampled during the late spring and early summer months. These analyses indicate adequate copper, zinc and manganese. But, the antagonistic effects of iron, sulfur and molybdenum have the potential to negatively affect the utilization of these minerals.

<table>
<thead>
<tr>
<th>Pasture</th>
<th>Copper</th>
<th>Zinc</th>
<th>Manganese</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.5 ppm</td>
<td>15 ppm</td>
<td>1 ppm</td>
</tr>
<tr>
<td>2</td>
<td>1.5 ppm</td>
<td>10 ppm</td>
<td>0.5 ppm</td>
</tr>
<tr>
<td>3</td>
<td>3.0 ppm</td>
<td>20 ppm</td>
<td>1.5 ppm</td>
</tr>
<tr>
<td>4</td>
<td>2.0 ppm</td>
<td>12 ppm</td>
<td>0.8 ppm</td>
</tr>
</tbody>
</table>

COPPER DEFICIENCY A POSSIBLE CAUSE OF POLIOENCEPHALOMALACIA IN YOUNG GOATS

Prairie Diagnostic Services - Canada

During the spring of 1999, the Regina laboratory received submissions from a producer experiencing problems in pygmy kids between 1 1/2 - 2 months of age. The history presented for one kid included fever, depression, head pressing, circling and terminal opisthotonus. Another animal and several more at home exhibited generalized weakness and muscle tremors. Weakness was most pronounced in the hindquarters.

At necropsy, the kid with seizures had severe cerebral edema with laminar necrosis of the cerebral cortical gray matter (polioencephalomalacia). The
spinal cord from the kid with generalized weakness displayed extensive hypomyelination with neuronal chromatolysis and necrosis. Both kids had decreased numbers of Purkinje neurons and cells within the granular layer of the cerebellum with chromatolysis of medullary neurons. Hepatic copper level from the kid with weakness was 2.4 ppm, a level considered very deficient (normal range 25 - 150 ppm). A CBC indicated marked nonregenerative anemia (hemoglobin 82 g/L; hematocrit 0.10). Both goats had mild to moderate thyroid hyperplasia. One animal had moderate coccidiosis.

The owner housed sheep with the goats. Both were receiving hay, barley, sheep supplement and cobalt/iodized salt. Drinking water sulphate and phosphorus levels were within normal ranges.

When sheep and goats are fed together, it is not uncommon to feed supplements designed for sheep. The practice predisposes goats to copper deficiency as their requirements at 10 - 20 ppm are much higher than those for sheep at 5 - 10 ppm. Although dietary copper levels were not calculated, a copper deficient diet with respect to goat requirements was strongly suspected. Genetic or breed predisposition and the interfering role of dietary molybdenum were not ruled out. Thyroid hyperplasia may have also been genetically related as dietary iodine levels appeared normal.

Copper deficiency in young goats typically appears as "enzootic ataxia" related to spinal chord and cerebellar changes. Cerebellar changes noted in this case were consistent with copper deficiency. Low copper levels were suspected as contributing to polioencephalomalacia. Similar lesions have been reported in young lambs from England. Other causes of polioencephalomalacia include: thiamine deficiency, high sulphates, water deprivation, hypoxia and any condition causing cerebral edema.

<table>
<thead>
<tr>
<th>Mineral</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cu, ppm</td>
<td>7</td>
<td>19</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Zinc, ppm</td>
<td>36</td>
<td>36</td>
<td>24</td>
<td>37</td>
</tr>
<tr>
<td>Mn, ppm</td>
<td>57</td>
<td>65</td>
<td>55</td>
<td>41</td>
</tr>
<tr>
<td>Mo, ppm</td>
<td>0.62</td>
<td>0.19</td>
<td>4.1</td>
<td>-</td>
</tr>
<tr>
<td>S, %</td>
<td>0.26</td>
<td>0.47</td>
<td>0.46</td>
<td>0.36</td>
</tr>
<tr>
<td>Fe, ppm</td>
<td>457</td>
<td>385</td>
<td>179</td>
<td>136</td>
</tr>
</tbody>
</table>

There can be little doubt that copper deficiency in ruminants is a worldwide problem. A recent survey on the nutrition of grazing ruminants in tropical regions has indicated that copper deficiency is a serious problem in many tropical countries:

ARGENTINA  KENYA
BOLIVIA  INDONESIA
BRAZIL  PANAMA
Copper deficiency in Alaskan Moose and Finnish Reindeer have both been reported. In Finland the deficiency is so severe that winter survival is considerably impaired. Red Deer in North West Scotland and in the West Midlands of England have been found to suffer a swayback disease characterised by ataxia, paresis and spinal chord demyelination.

Copper is necessary for the absorption and utilization of Iron, it helps oxidize vitamin C and it works in conjunction with Vitamin C to form elastin, a chief component of muscle. It also helps with the formation of red blood cells and bone structure. A copper deficiency does not allow the bone marrow cells to reach maturity.

Sheep accumulate copper in the liver more readily than other farm animals and are highly susceptible to copper toxicity. Texel and Blue Faced Leicester sheep are known to be especially susceptible. Sheep should not be supplemented with copper above 10ppm, or allowed access to supplements containing high levels of copper.

Recent copper toxicity problems in sheep in Ontario, Canada
Chronic Copper Poisoning in Sheep
Selecting the Right Grain Ration for Sheep

Note: mineral mixes labeled for sheep AND goats will NOT contain adequate copper for goats. Generally goats should not be fed sheep minerals without some other form of copper supplementation.

Copper is actively transported through the intestinal wall and stored in the liver. Copper deficiency prevents iron from being incorporated in hemoglobin, resulting in anemia, indistinguishable from iron deficiency. Copper plays a role in iron absorption and mobilization. Copper deficiency impairs the formation of connective tissue proteins, collagen and elastin. Weak bones (osteoporosis), and defective arterial walls are the more obvious manifestations.

Many areas of the country with copper deficiency problems can correct a copper deficit by simply adding a mineral containing adequate copper for goats, such as a horse or cattle mineral mix.

Looking back to the early and mid 1980's we can see that the problem actually began to manifest itself that long ago with an occasional animal exhibiting what we now know were signs of deficiency. In the beginning many of us had few if any classic copper deficiency symptoms, but testing has shown ALL our
herds to be severely deficient. Some blood lines, and breeds appear to have more (Alpines) or less (Nubians) problems than others.

While liver biopsy is the most reliable test of the true copper status of the animal, few private practice or university affiliated veterinarians are willing to take the risk of performing liver biopsies on breeding stock. In the last few years there has been interest in the procedure and some successful caprine liver biopsies have been done at Texas A&M. The University of California at Davis has developed a procedure for doing liver biopsies on cattle that is proving successful. But for now, there is no reliable test readily available to the small goat keeper on a live animal; liver levels from the liver of deceased animals are still the only accurate indication of the Cu status that we have access to. Copper is carried in the blood in a variety of ways and conventional blood tests measure only the total copper content. Of this, usually only about 3% is available for use in enzymes. In cases of thiomolybdate toxicity, this will be grasped by the thiomolybdate ions. Blood serum and plasma copper levels are the last to fall, after Cu reserves fall to <30 mg/kg liver DM, so they seldom correlate to the actual levels in the liver. Blood levels can be normal, low, or even elevated, while the copper stores in the liver and kidneys are extremely deficient, blood testing for copper is a poor second choice, and we seldom test the blood levels anymore (NOTE: serum zinc levels can change rapidly in response to stress). Normal liver levels of copper for goats are 25ppm-150ppm. - unsupplemented animals in this area run 0.1ppm-15ppm. While liver copper and total blood copper are used alone as indicators of copper status, these do not take any account of the correction of the symptoms of clinical copper deficiency. The efficacy of copper supplementation of ruminants is the ability to correct the symptoms of clinical deficiency and should not be judged by the supplement's ability to raise the copper content of the body. Copper levels in hair samples are highly variable.

The losses to local goat breeders were immeasurable, one year in the early '90s a herd in Arizona lost all but three kids to copper deficiency (confirmed by Arizona U and Cornell), they were either stillborn or died within the first few weeks. The loss in mature animals was tremendous to herds experiencing the most severe problems. On the other hand, many of us did not have specific or classic copper deficiency symptoms, but rather a multitude of miscellaneous problems such as frequent staph lesions on the udder, nose, mouth, and chin (occasionally the entire body) and thin/rough/faded (achromotrichia) hair coats (odd "prickly" coats in the case of some Saanens), maybe nothing more than bald tail tips or light spots on the nose. Other herds had serious problems with increased cases of mastitis including gangrene mastitis (more than a dozen cases of gangrene mastitis in this area in a two year period, when it was virtually unheard of before - and since), ruptured uterus' and pre-pubic tendons (abdominal wall hernia), hematomas following injections or even a minor injury. What acts like spinal cord injuries in adults (osteoporosis), twisting or bending of the front legs and/or feet in kids and pregnant yearlings (osteoporosis - see photo #1), anemia .... just about anything you can imagine ..... we were all mystified (our veterinarians and professionals in academia included) that our seemingly well managed animals were so plagued; again, some herds experienced NO obvious problems. We now realize that many of these situations were/are a direct effect of a compromised immune system resulting from the hypocupric condition. While other problems (bone disorders - ataxia - hair - cardiovascular) were a direct result of the low levels of copper. Young kids are most often and severely affected, with everything from the classic symptoms of swayback (congenital copper deficiency) and enzootic ataxia (delayed-onset ataxia) caused by demyelination of the spinal cord (a breakdown of the insulating fatty coverings [myelin sheaths] that surround the nerves in the brain and spine), this ataxia is NOT reversible by treatment with copper. to light colored rings around the eyes, thin hair over the nose and/or around the eyes and/or ears, small size, general weakness or sore joints and general failure to thrive. The does are not able to get kids on the ground with adequate levels of copper to maintain them in good health. Often they are so extremely deficient that they suffer from osteoporosis (soft, porous bones that bend and fracture easily i.e neonatals with rib fractures), severe anemia, or other health problems, some are unable to survive at birth, some appear normal at birth with symptoms showing soon after or weeks/months later depending on the level of the deficiency and the individual animal.

If copper nutrition was as simple as determining the copper levels in the base diet and adding a highly available copper source/supplementation, copper deficiency would not be a problem. However, because copper absorption and metabolism can be affected by molybdenum, sulfur, calcium, zinc, iron, manganese, cobalt, lead, cadmium, and selenium, deciding how much supplemental copper is required is not always easy.

Early on (after we identified the problem) we tried via oral supplementation of different mineral mixes high in copper (up to 1100ppm) and feeding of other than goat specific feeds (horse pellets, horse minerals, etc.) to correct the problem, to date none of them has succeeded in bringing up the body stores of copper. Absorption of copper can vary from zero to as high as 75% (Linder, 1991) depending on a number of factors. Copper availability in most feedstuffs fed to farm animals is between 1% and 15% (Hemken et al. 1993). Most minerals contain copper oxide in powder form, availability is poor when used
in this form, the mineral passes through the gut with little absorption. *(note: other areas of the US have had excellent results with just the addition of a mineral mix high in copper)* in our area we have found copper boluses (copper oxide wire boluses) dosed to weight to be the most effective means of elevating the liver copper levels to within normal limits. We had the first boluses brought into the US from New Zealand in the spring of 1994; since that time we have found a source of cattle copper boluses that we can downsize to goat doses. In this area 2000 to 2500+ goats have been on these boluses for nine years now (early '02). Continuous laboratory work on bolused animals indicates we are achieving normal liver concentrations of copper. To this date (June 2006) we have not had a single case of copper toxicity, and only one elevated liver copper level. Liver concentrations remain in the low normal (30-80ppm) with only three animals testing above that range in the twelve years we've been using the boluses. We've found that the boluses need to be administered at 5-6 month intervals to maintain adequate levels. After about 4 months, liver stores start to fall rapidly. In order to best protect the neonatal kids, we strive to use the boluses at times that will keep the does levels up during her entire pregnancy. Minnesota research with mice showed that perinatal brain development was affected by copper concentration in the mothers diet. Mice born to copper deficient dams had permanent brain disorders even when fed adequate copper after birth. Some breeders are routinely giving boluses (0.625 to 1.35 grams) to kids early on (2-4 weeks old) and it's proven to be very satisfactory (I've done this the last six years in my herd). This is an ongoing program. We get additional/new information, ideas, etc., constantly. As time goes on the regime may change to less or more frequent bolusing or perhaps a completely different method of correcting the problem. After more than a decade we've still not found a more satisfactory source of copper supplementation than copper boluses, nor a solution to the primary source of the problem (hay/feed). Until then, its imperative that we work together to keep on top of the situation and keep our animals healthy. We have veterinarians, both private and university affiliated, still interested and working on the problem. Our laboratory work has been done primarily by the California Animal Health & Food Safety Laboratory System [ CAHFS/UCDavis, was California Veterinary Diagnostic Laboratory Sytem, CVDLS/UCDavis] in San Bernardino and Davis, and the University of Arizona. Unfortunately, there are still veterinarians in the area that are unaware of the problem and even argue against its existence, though as time goes by more and more are seeing both the problem and our results and are recommending copper supplementation to goats in these areas.

Additional information, copies of laboratory work and veterinary information is available (your veterinarian is welcome to contact the veterinarians working with us for additional/specific information and confirmation as well).

**One Example**

(Photos & history of Toggenburg buck by Linda Colquitt - Eders Toggs, Alpines & Colquitt's Saanens)

This Toggenburg buck was born 3/92 in a herd having severe copper deficiency problems. Other animals in this herd were put down due to limb paralysis. This buck was a normal medium brown Toggenburg color at birth and left the herd at 3 months. The entire herd was blood tested for copper several months later and his twin sister was the lowest in the herd (0.06ppm). Only 6 of 17 animals were in the normal range of 0.8- 1.2ppm. At six months of age the buck was entirely white.

After seeing the blood tests above, he was given a MolyCu injection of 0.25mL. There were no other changes made in his care or nutrition. In about 10 days, he had dark roots. The picture below was taken
about 1 month after the injection. In February 93 he was given a copper Bolus of 2.5gms (the only size available at the time). In May, he was clipped for the shows and was a normal Toggenburg brown. The clippings showed a definite change in color with 1/3 of the hair nearest the skin being dark and the other 2/3 nearly white.

It is important to stress that color change might not be present in animals with a copper deficiency and that more serious problems can result from copper deficiency.

HOW THE COPPER BOLUSES WORK

References:
"Veterinary Drug Therapy" by Thomas Barragry '94.
"Cap With Confidence" Copacaps/Rhone Merieux Animal Health, New Zealand.
"Copper deficiency in sheep and cattle" Western Australia Dept of Agriculture

When copper deficiency has been recognized, attempts to remedy it by provision of extra oral copper has proved unsatisfactory because of the unpredictable intake, rapid excretion, and variable effect. With an element such as copper, which is a cumulative poison, the risk of chronic copper poisoning from parenteral or oral copper treatment is positively correlated with its effectiveness in combating deficiency. Existing methods of treatment for copper deficiency have limitations. Mineral licks and supplementals are unpredictable because of the individual refusal of some animals and over indulgence of others. Copper sulfate (CuSo4) drenches are not only astringent (Cu sulphate drench, if it accidentally enters the lungs, can cause shock and death) but more than 90% of the copper is rapidly excreted from the body. Animals need to be drenched every 2-3 weeks. Boluses (glass) of copper that lodge in the rumen or reticulum can form unusable complexes with molybdenum, sulfur and iron. Compounding copper salts with concentrate rations can be effective (though it has not proven so with our animals). Injectable copper (copper glycinate, CuCa-EDTA, copper methionates and Cu-oxyquin) can be acutely toxic (seen most often w/Cu EDTA which is no longer available), so injectable doses must be limited, the dose is often partly encapsulated at the injection site and thus prevented from achieving its objective. Side effects such as injection site abscesses (copper glycinate) and hepatic necrosis are potential problems with this method of treatment. Repeated injections are needed to maintain adequate protection. note: we only use copper glycinate (Molycu) injections in emergency situation, usually in young kids from unbolused dams.

Gelatin capsules containing copper oxide needles provide relatively long term protection against copper deficiency. The sustained activity after oral dosing with copper oxide needles as a means of alleviating hypocupremia in goats has been widely reported.
The gelatin capsules contain thousands of minute, blunt copper oxide rods. When given orally, the gelatin capsule dissolves in the rumen, releasing the copper oxide rods, which then pass into the abomasum where they lodge. There they release copper for the animal's immediate requirements and reserves. The rods dissolve completely over a period of time.

NOTE: There is at least one study (Attempted Induction of Chronic Copper Poisoning in Boma Confined Impala, Research and Development, Kruger National Park, Skukuza, South Africa, '99) that indicates, via fecal copper concentrations, that a good portion of the copper oxide particles are excreted from the body. Despite deliberate attempts to overdose the study Impalas with one time doses ranging between 125 mg/kg to 1000 mg/kg, less than 20% of the animals were found to have elevated liver copper levels after 52 and 105 days.

Copper oxide needles are brittle rods (1 to 8 mm long, and 0.5 +/- 0.1 mm in diameter) made by oxidizing fine copper wire. They are nontoxic when given orally, and they can be given in doses sufficient to establish long-lasting reserves of copper in the liver. Their properties were discovered by Australian scientists, who found that a combination of small particle size and high specific gravity (2.0 and 7.0) caused them to become trapped in the folds of the abomasum. Copper oxide particles, released in the rumen pass through to the abomasum where they remain in the folds of the abomasum. CSIRO (1978) and Judson et al., (1982) demonstrated that the particles remained for a period of at least 32 days. CSIRO (1978) showed that the excretion rate of copper from the copper oxide particles was about 0.2 grams by weight per day which allowed for the safe absorption of copper without toxicity being apparent. The accumulated hepatic stores of the absorbed copper can protect the animal against copper deficiency for periods of months (our lab work indicates 4.5-6 months). To be effective the Copper particles must be swallowed, administer by a conventional balling gun which delivers the capsule direct into the gullet. The gauge and weight of the copper particles is calculated so that they sink and lodge properly. Chewing rods/wires/particles will change both the gauge, weight, specific gravity, causing the particles to pass on through the animal in greater amounts than the dose is adjusted for.

To get the most out of your copper supplementation program

Heavy worm burdens can affect copper uptake by altering the pH in the gut, making the copper less soluble. An effective worming program is therefore an important aspect of copper supplementation. Internal parasites can:

- Reduce the solubility of copper in the abomasum (fourth stomach), by up to 70 per cent.
- Reduce the subsequent uptake of dissolved copper by the liver by up to 50 per cent.
- Increase copper losses from the animal.
While the use of cupric oxide rods has been shown to produce significant anthelmintic effects, their efficacy may be reduced by a heavy abomasal parasite burden.

It is important that adequate selenium (Se) levels are also maintained. See: U.S. Geological Survey Selenium in Counties of the Conterminous States. Selenium testing: Whole blood (EDTA or heparin) is the best sample since most of the selenium is located on red blood cells. Serum selenium analysis is possible but does not reflect long-term status of the animal.

**North Dakota:** Areas With High Concentrations of Selenium in the Soil and Forage Produce Beef With Enhanced Concentrations of Selenium

**PAY ATTENTION!**

**Copper can be toxic,** it is important to stress again, that this is a local problem and solution, and though both primary and secondary Cu deficiency problems of different magnitudes may be found in other areas we do not recommend supplementation using these methods or doses without complete evaluation of your herd’s copper status via laboratory work and veterinary consultation. Dose rates:

The animals are dosed to weight at the rate of 1 gram copper oxide in bolus form per 22 pounds at five to six month intervals, laboratory work has shown that liver and kidney concentrations start to fall rapidly after about four months. [Copasure downsize guide](#)

**Sources - Update: 2004**

Valley Vet Supply in Kansas carries both Copasure boluses and the #13, 1/8th oz empty gel caps. 800-360-4838

[http://saanendoah.com/cubolusdosechart.html](http://saanendoah.com/cubolusdosechart.html)

Jeffers - Dothan, Alabama 1-800-533-3377 - fax 1-334-793-5179 has Copasure boluses on an on and off basis.

Animal Health Express, Tucson, Arizona, 1-800-533-8115 has Copasure boluses in both 12.5 gram and 25 gram sizes.

Walco International in Ontario, California (1-909-947-4957) has Copasure boluses (25 gram only) in stock. **NOTE:** The Walco in Ontario, California will NOT sell for use in goats.

**Smaller size capsules:**

"000" (1.37 mL) - "00" (0.95 mL) - "0" (0.68 mL)

"1" (0.50 mL) - "2" (0.37 mL) - "3" (0.30 mL) - "4" (0.21 mL)

For smaller doses or multiples (an adult dose of 6.25 grams or 1/2 of a 12.5 gram boluse will fit into two "000" capsules). These can be found at health food stores, pharmacys or online at various herb and vitamin sites like [Herbal Remedies](#)