

Watch Copper Levels In Jersey Rations

This article summarizes the first study completed on copper metabolism in Jerseys, supported by a grant from the AJCC Research Foundation to R.W. Hemken and Z. Du at the University of Kentucky.

Recently, sudden death losses occurred in at least five Jersey herds that had been feeding copper at four to five times (40-50 ppm) the recommended level (10 ppm) for Jerseys for a period of 6 to 12 months. However, the amount fed was less than half of the National Research Council safe level, which is 100 ppm. Similar problems with copper toxicity for Holsteins have not been reported. It seems that Jerseys are more susceptible to copper toxicity than Holsteins. The objective of this study was to compare the differences in copper tolerances between Holstein and Jersey cattle.

Copper toxicity generally occurs after the liver accumulates a high concentration of copper and can cause a high death loss. Ruminant animals are more susceptible to copper toxicity than monogastric animals. Currently very little information is available concerning differences in toxicity among the dairy breeds.

Conducting the Study

Two age groups of animals were used in the study: 8 Jersey and 8 Holstein two-year-old cows, and 8 Jersey and 8 Holstein heifers with an average initial age of 16.8 months. The study design was appropriate to compare the two breeds at the differing levels of copper intake. Copper was supplemented in the ration at 5 ppm or 80 ppm of feed dry matter. The lactating cows were individually fed corn silage, alfalfa silage, whole cottonseeds and concentrate at the ration of 25:25:10:40 on an as fed basis twice daily free choice. The heifers were fed 1.82 kg. head per day of concentrate and corn silage and alfalfa silage at 1:1 free choice. The basic diets contained about 6.1 ppm for both cows and heifers. Copper was supplemented individually after the first liver biopsy.

Blood samples were collected via jugular vein at the beginning of the experiment and at 15-day intervals during the experiment. After centrifugation, the plasma and serum copper concentration, along with the ceruloplasmin (a copper transport protein in the blood) oxidase activities were analyzed. Liver samples were taken at the beginning of the experiment and monthly during the experiment via biopsy, and stored at -20°C for analysis of copper concentration.

Results

The results of the liver and plasma copper test revealed that Jersey cattle had higher liver copper concentrations than Holsteins even though the difference was not statistically significant on day 0 and day

Z. Du, R. W. Hemken, and R. J. Harmon. Copper Metabolism of Holstein and Jersey Cows and Heifers Fed Diets High in Cupric Sulfate or Copper Proteinate. *J Dairy Sci* 1996 79: 1873-1880.

Eight Holstein and 8 Jersey primiparous cows (3 d postcalving) and 8 Holstein and 8 Jersey growing heifers were randomly assigned to 1 of 8 treatments in a 2 x 2 x 2 factorial arrangement to compare Cu metabolism between Holsteins and Jerseys and the bioavailabilities of Cu in Cu proteinate and CuSO₄. The variables were Holstein or Jersey, Cu supplementation at 5 or 80 mg/kg of DM, and supplements of CuSO₄ or Cu proteinate. Jerseys had higher hepatic Cu concentrations than did Holsteins on d 60 (346 vs. 303 µg/g of DM). At the high Cu supplementation, hepatic Cu increased more rapidly, and content was higher in Jerseys than in Holsteins by d 60 (520 vs. 439 µg/g of DM). On d 0, plasma Cu concentrations were 0.99 and 0.80 µg/ml, and, on d 60, concentrations were 0.96 and 0.88 µg/ml for Jerseys and Holsteins, respectively. Overall, serum ceruloplasmin oxidase activity was greater for Jerseys than for Holsteins. Jersey cows and heifers also had greater hepatic Fe (208 vs. 173 µg/g of DM) and lower hepatic Zn (82 vs. 91 µg/g of DM) than did Holstein cows and heifers at d 60. The bioavailability of Cu in Cu proteinate and CuSO₄ was the same. Plasma Cu concentration and ceruloplasmin have limited value as indicators of Cu status and availability to dairy cows and heifers.

30. This difference was significant at the end of the experiment. At the high copper supplemental level, Jersey cattle increased their liver copper concentration more rapidly than Holsteins during the experiment. By the end of the experiment, Jersey cattle had a higher liver concentration than Holsteins (520 vs. 439, P < .05). At the beginning of the experiment, plasma copper concentration was greater for Jersey cattle than Holsteins (P < .01). Unlike liver copper, plasma copper changed very little with time at both copper supplemental levels. At the end of the experiment, plasma copper of Jerseys was still higher than Holsteins.

Ceruloplasmin (Cp) was also tested to compare the oxidase activity between breeds. The oxidase activity was greater for Jersey cattle compared to Holsteins on days 0, 30 and 45 of the experiment (P < .10). These trends were similar with that of plasma copper content. Further, more research must be done to determine why Cp activity declined rather than increased during the experiment.

To determine if there was liver damage, two specific enzymes were studied. The activity of these enzymes, serum lactate dehydrogenase (LD) and glutamic oxaloacetate transaminase (GOT), were measured in the animals systems. The normal physiological range of these two enzymes activity indicated that there were no breakdowns (dystrophy) in the liver.

Breed Differences

The copper concentration on day 60, along with plasma copper level and ceruloplasmin activity clearly showed there was a difference in copper absorption and/or post-absorptional metabolism between Jersey and Holstein cattle. The difference more likely occurred at high dietary copper supplementation as indicated by liver copper on day 60. This was the first

experimental demonstration of genetic differences in liver concentration and copper tolerance among dairy cattle breeds. The breed difference may be related to the efficiency of dietary copper absorption or copper excretion.

Implications

Considering all liver and plasma copper concentration and CP activity, there was a difference in copper absorption and/or metabolism between Holstein and Jersey cattle. Assuming that clinical copper toxicosis (death of cattle) happens at the same liver copper level for both breeds, Jerseys should be more susceptible to copper toxicity based on the fact that their liver accumulated more copper than Holsteins at the same dietary copper level. However, this was not proven by this study, as the experimental period was relatively short and no clinical copper toxicosis (death due to copper toxicity) occurred during this experiment.

While this study did not answer all the questions concerning copper toxicity in Jerseys, it does point out the need to further investigate the copper tolerance levels for Jerseys. The results indicate that copper metabolism was different between Jersey and Holstein cattle. It also suggests that Jersey cattle could be more susceptible to copper toxicity.

From this initial research, to avoid copper toxicity in Jerseys, it is recommended that Jersey herds not be fed more than 20 ppm copper, which is twice the recommended level for dairy cattle. Feeding a ration containing 10 to 12 ppm may be even more advisable. Additional research must be done to establish the maximum safe level of copper in the ration for Jerseys.

Avoiding Copper Toxicity

If a little is good, a lot must be even better, right? Right if you're talking about money, wrong if you're talking about copper.

Trace minerals, including copper, are a vital part of nutrition. Since most cows don't consume enough in their natural diet, feeding a mineral supplement has become a common practice for many Jersey breeders. In recent years several Jersey herds have consumed too much copper, something that has proven to be a problem.

One Breeder's Experience

Having problems with cows consuming too much copper is something Dan Bansen of Forest Glen Jerseys, Dayton, Ore., remembers all too well. Over a six- to eight-month period, Forest Glen lost more than 60 cows. "Every breeder has one or two, maybe even three cows that die for no apparent reason, but 60, that's crazy," Bansen said. "Something was wrong and no one knew what."

The first thing Bansen did was send tissue and organ samples to Oregon State University (OSU). "Their first diagnosis was gossypol toxicity, so we stopped feeding cottonseed to all the cows," Bansen noted. And they still lost cows. So he started sending entire cows to Dr. John Maas at OSU. After doing some heavy metal tests, copper toxicity was the final diagnosis.

What is Copper Toxicity?

Copper toxicity occurs when the liver gets to the point where it can no longer absorb any more copper. The liver dumps the copper into the bloodstream and the blood copper level shoots up, causing a hemolytic crisis. In hemolytic crisis the blood loses its normal oxygen-carrying capability, ending in death.

Signs of Copper Toxicity

Death is the only sure sign. "Talking with producers that have had a problem, what happens is that a high producing cow suddenly gets sick, goes off feed within a week to 10 days, drops milk production, then dies," says Dr. Roger Hemken, professor emeritus at the University of Kentucky and chair of the 1989 National Research Council's *Nutrient Requirements of Dairy Cattle* publication. The only sure way to tell if it is copper toxicity is to have tissue biopsy. "All the people I have talked with said that was the only sure way they knew."

Hemken notes that a herd owner wouldn't lose a large number of cows feeding 40 parts per million (ppm) of copper. "It's not something that you are going to walk into your barn one day and 50 percent of the herd is dead," Hemken noted. "And for that reason it is hard to diagnose."

How Much Copper Is Too Much?

The National Research Council in its publication, *Nutrient Requirements of Dairy Cattle*, states that 100 ppm is considered safe to feed some breeds of dairy cattle. In research conducted since the NRC

publication, it has been discovered that although 100 ppm may be safe for some breeds of dairy cattle, it may be too high for Jerseys. A feed analysis showed that Forest Glen was supplementing between 40 and 50 ppm of copper, well below the 100 ppm considered safe by NRC.

"In most of the cases of copper toxicity that I have worked with, all were feeding less than 50 ppm," Hemken noted.

Monitoring the amount of copper that is supplemented is not a foolproof method for monitoring the copper level in an individual cow. There are other factors that affect the absorption of copper.

In a study conducted at the University of Kentucky, researchers found that higher levels of whole cottonseed (and cottonseed meal) increased the amount of copper that was absorbed. "We believe that it is the gossypol in the cottonseed that helps the absorption," Hemken said.

Another factor contributing to copper absorption is iron. It has already been proven that iron interferes with copper absorption. In another University of Kentucky study, researchers fed copper at levels higher than 40 ppm to Jersey and Holstein steers in order to create toxicity. By the end of the study, no steers had died and it was discovered that they were fed rations with iron levels as high as 500 ppm. The high levels of iron had interfered with the copper absorption preventing toxicity.

How to Fix the Problem

"Most cows will probably die before a breeder can do something," Hemken said. "But once it has been diagnosed, the problem can be fixed."

The first thing a breeder needs to do is stop supplementing copper. After that a breeder could feed higher levels of sulfur and molybdenum for a few weeks. Hemken did just that for a Jersey herd in Tennessee when copper toxicity was discovered. "That breeder in Tennessee is convinced we saved some cows by doing just this," Hemken noted.

Although Bansen didn't feed any sulfur or molybdenum, he did stop feeding all copper. "There are no good blood tests for copper so we instead monitored the culled cows and their copper levels," Bansen said. He continued to monitor the copper levels and when the herd was back to a comfortable level, he added a little copper back into the ration. "We are back to about 10 to 15 ppm and do a copper check about every six months."

Hemken does have a concern that herd owners who experience copper toxicity will inadvertently create the opposite problem, copper deficiency.

"Taking out all copper would be a natural defense for anyone who has had copper toxicity," Hemken commented. "I'm not sure that is good either. Copper deficiency has been found to depress the immune response system, making the animal less resistant to disease, and lowers reproductive ability. In addition research has shown that cows which are copper deficient are more

susceptible to mastitis."

Something else that concerns Hemken is when herd owners supplement trace minerals with mineralized salts and nothing else. "Many of these salts still contain copper oxide, an inferior form of copper, which isn't high enough to add anything to the cow's diet," Hemken said. "In my opinion, these are inadequate for optimum production."

What's Being Done

Through research funded by the AJCC Research Foundation, Hemken completed several studies comparing the differences between Jerseys and Holsteins and how they absorb and metabolize copper.

In the first study, Hemken and colleagues found that Jerseys absorbed more copper in the liver than did Holsteins. At a higher copper supplemental level, Jersey cattle increased their liver copper concentration more rapidly than Holsteins. This was the first experiment ever completed demonstrating a difference in copper tolerance among dairy breeds.

That study help explains one common situation, according to Dr. Hemken.

"Dr. Bob James of Virginia Tech talked to me about a herd he was consulting that was a mixed herd of Jerseys and Holsteins. The same amount of copper was fed to both and the breeder lost some Jerseys but didn't lose any Holsteins," Hemken said. Hemken also noted talking to a Jersey breeder in Georgia who purchased his feed from a Holstein milk producer. The Jersey breeder lost cows but the Holstein producer never did.

In another study conducted at Kentucky, researchers found that steers fed whole cottonseed had higher copper concentrations than those that were not fed cottonseed. Principal investigator Patrick French found that whole cottonseed might be a factor in copper toxicity if it is fed over an extended period of time when the liver copper concentration is reaching a toxic level.

"Someone in Michigan asked if Jerseys have a lower copper requirement than Holsteins," Hemken noted. "The answer is, 'We don't know.' There is still research to be done and questions to be answered."

What You Should Remember

- Jersey dairy managers need to be aware of this potential problem.
- Feed companies and nutrition consultants need to recognize this potential problem.
- An occasional death in a herd, or an increased death rate could be due to copper toxicity. The first thing herd owners need to do is look at the level of copper being supplemented. Higher levels need to be investigated further.
- If you have had a problem, don't cut back too much on the amount of copper you are supplementing. Copper toxicity can be controlled with proper management.

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