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MINERAL Writes

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What do calcium levels influence in transition dairy cows?

When dairy cows are diagnosed as hypocalcemic, the blood calcium levels are lower than normal at the beginning of the lactation cycle. This presents as either clinical hypocalcemia (milk fever) or subclinical hypocalcemia. Subclinical hypocalcemia is much more prevalent than clinical milk fever. A great deal of research has been devoted to preventing milk fever and the resulting consequences.

A recent study by Chamberlin et al. (2013) looked at the related question to the established research to determine the related questions about the associations between subclinical hypocalcemia, lipid metabolism, and animal health at postpartum, milk production and milk quality and fertility in postparturient multiparous Holstein cows. This research follows the trend of studying the occurrence of multiple postpartum disorders. The studies are finding that the disorders have common factors that influence their occurrences. Instances of clinical and subclinical hypocalcemia are proving to put cows at increased risk for many different diseases and disorders including: lipid related complications from increased levels of non-esterified fatty acids (NEFA) & BHBA including ketosis and fatty liver. Additional diseases can occur such as mastitis, metritis and increased risk of developing a displaced absomasum.

Experiment & Findings

Chamberlin et al. (2013), studied 100 cows over two years, dividing them into two groups based on whole-blood calcium concentrations, the hypocalcemic (n = 51) and normocalcemic cows (n = 49). Subclinical hypocalcemia occurs when the whole-blood total calcium is less than 8.0 mg/dL in dairy cows; in this study, the researchers chose the level of 4.0 mg/dL as the standard to determine the occurrence of subclinical hypocalcemia from normocalcemia. This study focused on instances of subclinical hypocalcemia and did not look at animals within clinical hypocalcemia levels.

On the day of calving, the researchers found the expected results where the hypocalcemic cows had lower whole-blood calcium concentrations and plasma total calcium concentrations compared to the normocalcemic cows. Milk protein percentage from hypocalcemic cows was lower at sampling times during the first 35 days of lactation. Hypocalcemic cows had higher mean plasma NEFA levels at day of calving and at d 21. The liver lipid percentage was also significantly higher in these animals at d 7 and d 35.

There was a negative correlation between the liver lipid percentages and plasma glucose concentrations and there was a positive correlation between the liver lipid percentage and amino acid metabolizing enzyme activity. The researchers also found a positive correlation between plasma NEFA concentrations and the same amino acid enzyme activity. Plasma NEFA concentrations and liver lipid percentage were also

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positively associated with blood ionized calcium levels on day of calving. Calcium concentrations were not significantly associated with the risk of animals developing displaced abomasum. In addition, no increased incidences of clinical mastitis, dystocia, retained placenta or metritis were associated with subclinical hypocalcemia. Low blood calcium and higher plasma NEFA concentrations often result when hypocalcemic cows are trying to produce more milk than in normocalcemic animals.

The relationship between subclinical hypocalcemia and plasma NEFA concentrations in humans is explained as the point when fat cells are stimulated by chemicals that bind calcium to calcium receptors and the breakdown of lipids is decreased. This suggests an increase in lipid/fat breakdown when calcium is depleted (Cifuentes and Rojas, 2008).

In dairy cattle, calcium is also an important part of energy metabolism in liver cells. The authors of the current experiment (Chamberlin et al., 2013) suggest that changes in plasma ionized calcium levels may influence calcium levels in liver cells which may inhibit oxidative metabolism because the intercellular calcium levels are insufficient. This will ultimately result in continued decrease in carbohydrate metabolism thus contributing to more negative energy balance, which triggers the release of NEFA in an attempt to generate energy (Shire and Beebe, 2014). Due to the increased NEFA levels in the blood, liver tryglicerides accumulate, resulting in "fatty liver."

Calcium also contributes as a messenger for immune responses (Shire and Beebe, 2014). Once a cow reaches a hypocalcemic state, her immune system declines to defend some infections. This can lead to potential increases in cases of mastitis. Adequate calcium levels are necessary to activate immune responses and muscle contractions for teat closure. This also correlates with increased metritis due to low blood calcium levels, especially with cows that have difficulty at calving.

Subclinical hypocalcemia may be associated with changes in lipid metabolism which is observed as increased plasma NEFA concentrations at calving and increased lipid deposits in the liver throughout the initial 35 days in milk. Additionally in this study, there was no association between subclinical hypocalcemia and changes in peak milk yield, milk fat percentage, SCC or fertility measures. The authors also found no differences in the association of negative energy balance and hypocalcemia and common postpartum disorders between normocalcemic and subclinical hypocalcemic cows.

Practical Applications:

Subclinical hypocalcemia is the more common occurrence of calcium imbalances in lactating and transition cows compared to incidences of clinical hypocalcemia (Shire & Beebe, 2014). Producers need to understand that during the transition period, calcium demand doubles due to the shift in biological processes as lactation starts. The negative shifts in blood calcium concentrations alter the homeostatic functions of cells. The calcium imbalance may be connected to the increased risks of secondary health disorders postpartum, such as displaced abomasum.

To counter the potential negative effects, producers need to be proactive about hypocalcemia and in particular, subclinical hypocalcemia. Paying attention to the management programs of transition cows is essential to continued success.

Information for this article taken from:

Chamberlin, W.G., J.R. Middleton, J.N. Spain, G.C. Johnson, M.R. Ellersieck, and P. Pithua. 2013. Subclinical hypocalcemia, plasma biochemical parameters, lipid metabolism, postpartum disease, and fertility in postparturient dairy cows. *J. Dairy Sci.* 96:7001-7013. http://dx.doi.org/10.3168/jds.2013-6901.

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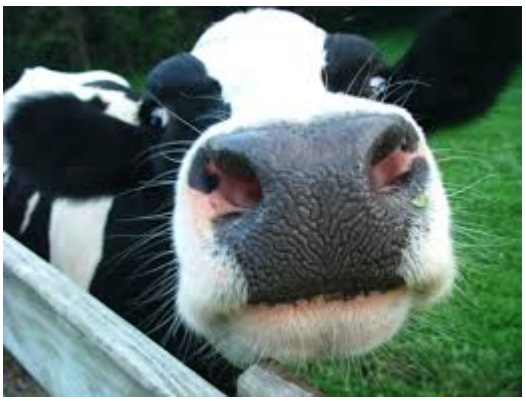
Shire, J. A and D.K. Beede. 2014. Hypocalcemia is more than a "low calcium" problem. *Hoard's Dairyman.* February 10, 2014. p. 81.



Bone remodeling induced by fatty liver disorder in laying hens

Osteoporosis commonly occurs in caged laying hens during the laying phase. The formation and resorption of bone is key to maintain any animal's or human's skeletal physiology and structural integrity. The balance between the two processes (bone homeostasis) is very important. When there is an imbalance, osteoporosis may occur. In humans, osteoporosis and obesity have been linked in repeated studies. The amount of visceral and total abdominal fat inversely associates with bone strength and bone mineral density (Science Daily, 2012).

Nutritional imbalances resulting from hens excessively consuming high-energy diets may induce chicken fatty liver syndrome (or hepatic steatosis). This means the lipoproteins have accumulated in the liver because hepatic lipogenesis exceeds the rate of fat mobilization (Hermier et al. 1994; Lee et al, 2010). Gradually, the liver cells lose their functions and are ultimately destroyed as a result of the positive energy balance that is causing the fat accumulation.

This article reviews a recent study by Jiang et al., 2013 in which they examined the effects on bone metabolism and blood parameters of fatty liver disorder induced by the consumption of high energy and low protein diets.

The Experiment

The researchers used 100, 63-wk-old Hy-line Brown laying hens. The birds were allocated to either a control diet which was a regular layer diet or the experimental diet, a high energy and low protein diet for 80 days.

Egg production and dead bird count was recorded daily. Feed intake was recorded at regular intervals throughout the trial. Body weight was recorded at the beginning and end of the trial. Blood samples were collected from 10 randomly selected birds from each treatment group at d 0, 60 and 80. At the end of the experiment, the liver, abdominal fat pad, tibias and tissue samples were collected from the keel bone of 10 birds from each group (n=20). Blood samples were analyzed for total calcium, inorganic phosphate, and total alkaline phosphatase (**ALP**) activity, estrogen (**ES**) concentrations, leptin-like protein and total osteocalcin (**OC**).

Findings

Average egg production was reduced in the hens fed the treatment diet. The high energy low protein hens had lower feed intakes compared to the hens fed the control (regular layer) diet. Before and after the experiment, there was no difference in body weights between the two treatments. Liver weights between control and treatment hens were not different. Hens fed the treatment diet had a greater abdominal fat pad weight and liver fat content.

Osteocalcin protein concentrations in the keel bone were decreased in hens fed the treatment diet compared to those on the control diet. Osteocalcin is a biomarker for evaluating the osteoblast (bone forming) activity. It also regulates energy metabolism.

Concentrations of calcium and phosphorus were similar between the two treatment groups of hens. The high-energy low protein hens had higher serum ALP activity, OC, leptin-like protein and ES concentrations at d 80 compared to control hens. The ALP activity and OC concentrations increased at d 60 in high energy low protein hens. Serum OC concentrations positively correlated to the serum leptin-like protein concentrations in both treatments. A positive relationship was found between the concentrations of serum OC and serum ES in high energy low protein hens. In control hens, the serum OC concentrations significantly decreased with age, but not in the high energy low protein hens.

What this means

The liver synthesizes proteins and peptides that may function as hormones or enzymes that are involved in energy and bone metabolism. This experiment found that the high energy low protein diet successfully induced fatty liver disorder in laying hens. The results were upregulated bone turnover which resulted in increased skeletal damage. This supports the hypothesis that as fat metabolizes it decreases skeletal health and enhances the development of osteoporosis in laying hens.

The high energy low protein hens were found to have significantly higher liver fat content, abdominal fat pad weight, and concentrations of leptin-like protein and estrogen. Egg production was lower in those hens.

The findings suggest that changes associated with fatty liver disorder interfere with the homeostasis of OC as it relates to bone turnover, thus, reducing bone health. The increased serum OC concentrations in the high energy low protein hens and decreased bone OC concentrations indicate the hens fed the treatment diet experienced greater bone turnover where OC was released from bone into the blood (Figure 1). Higher ALP activity is related to increased osteoblastic (bone forming) activity. The negative correlation between ALP activity and bone stiffness found in this experiment, shows that the increased ALP activity in the treatment hens likely was indicative of higher bone remodeling with weaker bone strength.

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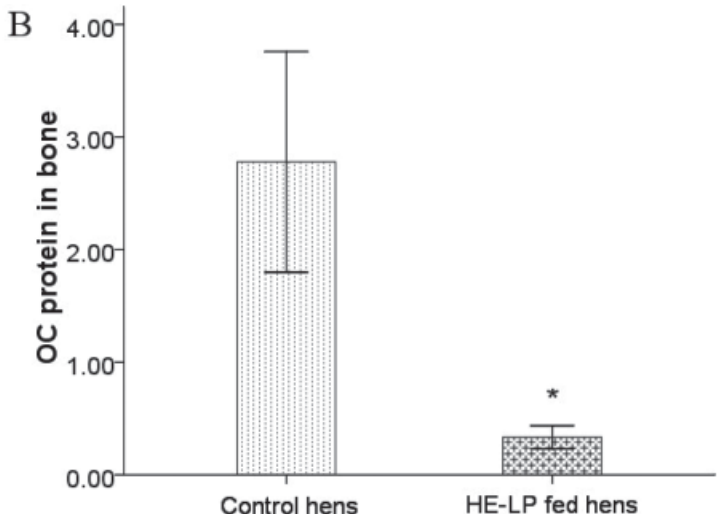
Bone remodeling in laying hens...



Estrogen concentrations declined with age in control hens and the high energy low protein hens had higher ES concentrations at d 80. The treatment hens also demonstrated a positive correlation between ES and serum OC concentrations. The author's position is that the fat intake associated change in blood ES concentrations relates to osteoblast activities. The result is the increased blood OC concentrations. Estrogen increases osteoblast activities differently in humans and chickens. Wilson and Thorp, 1998, found estrogen increases bone formation but does not improve bone strength in hens.

High energy low protein diets induce fatty liver disorder in laying hens. The birds may experience high bone turnover, but their bone health may not improve, and the incidence of skeletal damage rises. The positive correlation found between estrogen, leptin-like protein, and serum OC concentrations indicate that proteins secreted by adipose tissue and bone metabolism are directly linked in the regulation of bone remodeling in hens.

This study provides evidence that diets can be modified to control bone damage and the incidence of osteoporosis in laying hens.

Information for this article taken from:

Jiang, S., H.W. Cheng, L.Y. Cui, Z.L. Zhou, and J.F. Hou. 2013. Changes of blood parameters associated with bone remodeling following experimentally induced fatty liver disorder in laying hens. *Poultry Science* 92:1443-1453. http://dx.doi.org/10.3382/ps.2012-02800.

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