Diagnosing Common Vitamin and Mineral Abnormalities in Dairy Cattle

Jeffery O. Hall, D.V.M., Ph.D., Diplomat A.B.V.T.
Utah Veterinary Diagnostic Laboratory
Department of Animal, Dairy, and Veterinary Sciences
Utah State University
Email: Jeffery.Hall@usu.edu

INTRODUCTION

Many minerals and vitamins have been proven to be essential for optimal growth, physiologic function, and productivity in animals. Data from the Utah Veterinary Diagnostic Laboratory indicated a significant increase in incidence of vitamin and mineral deficiencies and excesses. Much of the deficiencies appear to be associated with producers decreasing or completely stopping the practice of vitamin-mineral supplementation of replacements due to the economy and costs. A common finding with many of the diagnosed deficiencies is a lack of vitamin-mineral supplementation to replacement heifers, resulting in them coming into their first lactation deficient. Mineral excesses are commonly associated with heavy use of chelated minerals in lactation total mixed rations (**TMR**).

Increasing incidence of adverse neonatal health effects, due to vitamin or mineral deficiencies are commonly encountered at the Veterinary Diagnostic Laboratory. A lack of or inadequate supplementation of replacement heifers results in maternal depletion of body reserves of minerals and, subsequently, in poor calf health.

Interestingly, the same deficiencies commonly identified in first lactation cows are only rarely observed in multiparous cows. In fact, excessive supplementation in the lactation rations tends to be more common and results in excessive mineral concentrations, especially for copper (**Cu**) and selenium (**Se**).

DEFICIENCY DIAGNOSES

Historically, testing for deficiencies has been performed on diets and/or dietary components to ensure *adequate* concentrations in the diet. However, general mineral analysis does not identify the chemical forms, which can dramatically alter their bioavailability and utilization. This is especially important with the increasing use of *chelated* minerals, as they can have significantly greater overall bioavailability than inorganic minerals.

Although not possible for some of the minerals, the most specific means of diagnosing a mineral deficiency is by testing animals for unique functional deficits or deficiencies of specific mineral-containing proteins or enzymes. This type of testing is often impractical from a field perspective, due to individual test costs or rigorous sample handling requirements. But, when possible, this type of testing eliminates the need to know the specific molecular characteristics of a dietary mineral and the potential for competitive interactions of antagonists of absorption/utilization. For minerals that do not have identified physiologic indices for testing, direct quantification from animal tissues or serum may provide a reliable indication of the overall mineral status of the animal or group.

Vitamin and mineral deficiencies can be suggestively diagnosed by the development of clinical disease or by post-mortem identification of tissue lesions. But, proof of deficiencies often requires analytical verification since most do not have very unique clinical signs or lesions. In some instances, circumstantial proof of a deficiency can be provided by positive response to supplementation of suspected deficient vitamins or minerals. But, positive response may have nothing to do with the supplementation, being just a correction of some other clinical condition.

An individual vitamin or mineral may have multiple means of measurement for identification of deficiencies, but most have one that is more specific than the others. For example, dietary concentrations may or may not be reflective of the amount that is bioavailable. Or, an individual tissue concentration may or may not reflect functional availability at the target or functional site.

The age of the animal being tested also is important for proper interpretation of status. For example, feti accumulate some minerals at different rates during gestation, necessitating adequate aging of the fetus for interpretation. In addition, some

minerals, for which little is provided in milk, accumulate at higher concentrations during gestation in order to provide neonates with adequate body reserves for survival until they begin foraging. This is especially prevalent with Cu, iron (**Fe**), Se, and zinc (**Zn**). Thus, the *normal ranges* for these minerals in body storage tissues would be higher in early neonates than in an adult animal. One must make sure that the testing laboratory is interpreting the results based on the age of the animals tested, as some interpret all as adults.

When individual animals are tested, the prior health status must be considered in interpreting vitamin and mineral concentration of tissues. Disease states can shift mineral from tissues to serum or serum to tissues. For example, diarrhea can result in significant loss of sodium (**Na**), potassium (**K**), and calcium (**Ca**) from the body. Or, acidosis will cause electrolyte shifts between tissues and circulating blood. It is known that infectious disease, stress, fever, endocrine dysfunction, and trauma can alter both tissue and circulating serum/blood concentrations of certain minerals and electrolytes. Thus, evaluation of multiple animals is much more reflective of mineral status within a group than testing individual animals that are ill or have died from other disease states.

LIVE ANIMAL SAMPLING

A variety of samples are available from live animals that can be analyzed for vitamin-mineral content. The most common samples from live animals are serum and whole blood. These samples are adequate for measurement of several minerals, but it must be recognized that some disease states, as well as feeding times, can result in altered or fluctuating concentrations. Other samples from live animals that are occasionally used include liver biopsies, urine, and milk. But, since milk mineral content can vary through lactation, across lactations, and be affected by disease it is not typically used to evaluate mineral status. Hydration status affects urinary mineral concentrations, rendering it a poor sample for status evaluation. For vitamin A and E, serum is the best sample from live animals.

Although some laboratories suggest hair sampling to evaluate mineral status or exposure, hair is a relatively poor matrix to give reliable data on mineral status, except for Se. Since body hair in livestock is notoriously contaminated with environmental contamination (dirt, manure, etc.), the hair MUST be cleaned thoroughly to remove contamination prior to testing. But, thorough cleaning can also leach out some of the true minerals in the hair, which results in measuring less than what was truly present.

Serum should be separated from the red/white blood cell clot within 1 to 2 hr of collection. If the serum sets on the clot for longer periods of time, minerals that have higher intracellular content than serum can leach into the serum and falsely increase the serum content. Minerals for which this commonly occurs include K and Zn. In addition, hemolysis from both natural disease and due to collection technique can result in increased serum concentrations of Fe, magnesium (**Mg**), manganese (**Mn**), phosphorous (**P**), K, Se, and Zn. Vitamin A and E can begin breaking down in serum if not separated from the red blood cells and frozen within 1 - 2 hr of collection. Serum for vitamin A and E analysis should be stored to prevent breakdown from sunlight exposure.

The best type of collection tube for serum or whole blood is royal blue-top vacutainer tubes, as they are certified trace-metal free. Typical red-top clot tubes can give abnormally increased results for Zn content as a Zn containing lubricant is commonly used on the rubber stoppers. For minerals other than Zn or vitamins A and E, serum samples from the typical red-top clot tubes or separator tubes are adequate.

Samples should be appropriately stored for adequate sample preservation. Liver biopsies, urine, and serum can be stored frozen long term or refrigerated if mineral analysis is to be completed within a few days. Whole blood and milk should be refrigerated but not frozen, as cell lysis or coagulation of solids, respectively, will result in loss of the overall integrity of the sample.

Liver biopsies, because of their small size, are susceptible to desiccation unless properly stored. Small biopsies should be placed into SMALL tubes, with the sample pushed all the way to the bottom. Small 1 to 2 ml micro-centrifuge tubes work well for this. By placing the sample at the very bottom of the tube, one minimizes the air to sample interface area and minimizes potential for desiccation. The samples can then be frozen for transport.

POST-MORTEM ANIMAL SAMPLING

A variety of post-mortem animal samples are available that can be analyzed for vitamin-mineral content. The most common tissue analyzed for mineral content is liver, as it is the primary storage

organ for many of the essential minerals. In addition, bone is used as the primary storage organ for Ca, P, and Mg. For vitamin A and E, liver is the tissue of choice for analysis, but it needs to be relatively fresh. Tissue degradation will correspondingly decrease the vitamin A and E present.

Post-mortem samples should be stored frozen until analyzed to prevent tissue degradation of the vitamins. If samples are to be analyzed within 1 – 2 d, they can be stored under refrigerated conditions.

COPPER

Copper deficiency is one of the most commonly encountered nutritional problems in ruminants, but Cu excess is also commonly encountered, especially in sheep or in dairy cattle. Excessive Cu is a relatively common finding in multiparous dairy cattle, while most deficiencies are identified in calves or first lactation cows. In contrast, Cu deficiency is rare in non-ruminants. Clinical signs of deficiency can present as a large array of adverse effects, including reduced growth rates, decreased feed conversion, abomasal ulcers, lameness, poor immune function, sudden death, achromotrichia, poor lactation, and impaired reproductive performance.

Cows will do all they can to ensure adequate Cu is in calves when they are born. They can actually deplete their own body reserves to ensure neonatal adequacy. As such, neonates diagnosed with Cu deficiencies are proof of maternal deficiencies. With Cu being an essential component of the immune function, this maternal deficiency likely results in poor colostrum quality and inadequate neonatal protection even with adequate intake.

The best method for diagnosing Cu status is via analysis of liver tissue, although much testing is performed on serum. Deficiency within a herd will result in some animals that have low serum Cu concentrations, but serum content does not fall until liver Cu is significantly depleted. In herds that have had livers tested and found a high incidence of deficiency, it is not uncommon for a high percentage of the animals to have *normal* serum concentrations. At the Utah Veterinary Diagnostic Laboratory, it is commonly recommended that a minimum of 10 animals be tested in order to have a higher probability of diagnosing a Cu deficiency via serum quantification. Even with herd deficiency, low serum Cu concentrations may only be seen in less than 10% of the individuals. Herds that may be classified as marginally deficient based on liver testing may have predominantly *normal* serum Cu concentrations. Thus, serum Cu analysis should be viewed as a screening method only. Another factor that can influence diagnosis of Cu deficiency in serum is the presence of high serum (**Mo**). As the Cu-sulfur (**S**)-Mo complex that forms is not physiologically available for tissue use, *normal* serum Cu content in the presence of high serum Mo should always be considered suspect. In addition, the form of Se supplementation can alter the normal range for interpretation of serum Cu status, with selenite supplemented cows having a lowered normal range for serum Cu.

Excessive Cu in dairy cattle is a common finding at the Utah Veterinary Diagnostic Laboratory. Liver Cu concentrations greater than 200 ppm are routinely identified. But, in recent years, several cases of deficiencies also have been identified, due to poor mineral supplementation programs. These have most commonly been in first lactation cows that were not adequately supplemented in the growth, breeding, or pre-lactation period.

Excessive liver Cu has the potential of causing adverse health and production effects. Liver Cu concentrations greater than 150 - 200 ppm have been shown to cause increased liver enzyme leakage, indicating adverse effects on tissue health. Adverse liver health and function can adversely affect feed/energy utilization and overall productivity of an individual. As Cu concentrations get even higher, further liver damage can occur.

Over-supplementation of Cu, as indicated by excessive liver Cu, in dairy cows has been increasing over time. The nature of these findings tends to correspond to increasing use of chelated minerals in dairy rations. Due to the increased bioavailability of chelated minerals, adding them to rations at the same concentrations as inorganic Cu can result in over-supplementation. Excessive dietary Cu also plays a role in the adverse interaction with other minerals. For example, excessive dietary Cu adversely impacts the absorption of Zn.

The recommended adequate wet weight liver Cu concentration range in adult cattle is 25 to 100 ppm. In comparison, a late term fetal or early neonatal liver should have 65 to 150 ppm Cu to be considered normal.

MANGANESE

Manganese deficiency in ruminants is associated with impaired reproductive function, skeletal abnormalities, and less than optimal productivity. Cystic ovaries, silent heat, reduced conception rates, and abortions are reported reproductive effects. Neonates that are Mn deficient can be weak, small, and develop enlarged joints or limb deformities. Manganese deficiencies in beef cattle, although rare, would most commonly be seen in areas of highly alkaline soils, due to much poorer plant uptake, but this is not very commonly identified.

Manganese at sub-normal to deficient concentrations is identified routinely in dairy cows. Of interest is the fact that most testing of beef cattle (greater than 95 %) finds normal Mn concentrations in liver, blood, and serum; but in these same matrices many dairy cattle tested are below recommended normal concentrations (unpublished data, Utah Veterinary Diagnostic Laboratory). This may, in part, be due to high Ca and P content of dairy rations, which can be antagonistic to the bioavailability of Mn. Another potential factor that can play a role in Mn status differences in beef vs. dairy cattle can be the diet. Some dairy herds that are grazed on grass have a similar lack of deficiencies or sub-normal concentrations of Mn as those observed in beef cows. Grasses typically have higher Mn content than forbs and the chemical form of Mn may be more bio-available than inorganic Mn supplements.

Of the samples available, liver is the most indicative of whole body status, followed by whole blood and then serum. As red blood cells have higher Mn content than serum, hemolysis can result in increased serum content. Since the normal serum concentration of Mn is quite low, many laboratories do not offer this analysis because of inadequate sensitivity. Overall, response to supplementation has frequently been used as a means of verifying Mn deficiency, but it is critical that a bioavailable form be utilized.

Unlike Cu, Se, Fe, and Zn, late term feti and neonates have lower Mn content than adult animals. Calves generally will have similar normal ranges to adults by 5 - 6 mo of age. For wet weight normal liver Mn, adult normal range is 2.0 to 6.0 ppm, while a neonatal normal range is 0.9 to 4.5 ppm.

SELENIUM

As an essential mineral, Se is commonly identified as deficient in ruminants, but infrequently in dairy cattle. In dairy cattle, the most common finding of Se deficiency is in replacement and first lactation heifers. This type of deficiency has significantly increased over the past 5 yr. Selenium deficiency is associated with reduced growth rates, poor feed efficiency, poor immune function, impaired reproductive performance, and damage to muscle tissues. *White muscle disease*, a necrosis and scarring of cardiac and/or skeletal muscle, is linked to severe Se deficiency; although, it can be caused by vitamin E deficiency as well.

Cows will do all they can to ensure adequate Se is in calves when they are born. They will actually deplete their own body reserves to ensure neonatal adequacy. As such, neonates diagnosed with Se deficiencies are proof of maternal deficiencies. With Se being an essential component of the immune function, this maternal deficiency likely results in poor colostrum quality and inadequate neonatal protection even in calves that get adequate volumes of colostrum.

Diagnosis of a deficiency can be made by analysis of liver, whole blood, or serum for Se content or by analysis of whole blood for glutathione peroxidase, a Se-dependent enzyme. The most specific analysis is that of whole blood glutathione peroxidase, as it verifies true functional Se status. Liver is the optimal tissue to analyze for Se content as it is a primary storage tissue. With serum and whole blood, the former better reflects recent intake, while the latter better reflects longer term intake status. Since seleno-proteins are incorporated into the red blood cells when they are made and the cells have a long half-life, Se content of whole blood is a better reflection of intake over the previous months than serum.

In order to adequately diagnose Se deficiency, the dietary form of the Se intake by the animals is important. Natural Se, predominantly in the form of seleno-methionine is metabolized and incorporated into Se dependent proteins, but can also be incorporated into non-specific proteins in place of methionine. Inorganic Se is also metabolized and predominantly incorporated into Se-dependent proteins. Thus, *normal* concentrations in serum and whole blood differ depending on whether the dietary Se is a natural organic form or an inorganic supplement.

Selenium excess is commonly identified in multiparous dairy cows. If the Se excess is great enough, it can result in poorer reproductive performance, poor calf survival, and imbalances of other minerals. Excessive Se can also interfere with Zn absorption. The recommended adequate liver Se concentration range in adult cattle is 0.25 to 0.50 ppm, with late-term fetal or neonatal liver normal being 0.35 to 0.75 ppm.

ZINC

Zinc is an essential mineral that is required by all cells in animals. Zinc plays a role in numerous enzymatic reactions. Deficiencies of Zn are associated with reduced growth, poor immune function, diminished reproductive performance, and poor offspring viability, as well as skin lesions in severe cases. Tissue Zn concentrations do not reflect body status well. Of the common samples tested, liver and serum are the best indicators of Zn status. But, serum and liver Zn can be altered by age, infectious diseases, trauma, fever, and stress. Response to added Zn has shown that some animals with low-normal liver or serum Zn can show improvement in some clinical conditions.

Over the past few years, the number of dairy cows found to have sub-normal to deficient Zn status has been increasing. It is important to note that almost all of these cases are in multiparous cows that are also found to have excessive Cu and Se in the liver. Thus, knowing that dietary excesses of both Cu and Se can interfere with Zn absorption, one must conclude that the low Zn is likely a secondary effect. In fact, several cases have corrected the low Zn by nothing more than decreasing the excessive Cu and Se in the rations.

VITAMIN A

Vitamin A is an essential fat soluble vitamin in ruminants. It is essential for all cell replications and is especially important in epithelial integrity. It plays an important role in tight junctions between cells, as well as being an important antioxidant in the body and in mucosal secretions. Vitamin A deficiency is associated with poor growth rates, poor feed intake, poor immune function, poor reproductive performance, and high incidences of diarrhea in calves. Loss of efficient tight junctions in the epithelial cell lining of the digestive tract allows opportunistic pathogens to invade and cause disease.

Vitamin A is provided in the diet via green growing vegetation or supplementation. Dead, brown forages have relatively no Vitamin A content. Thus, for grazing livestock, they must accumulate enough body reserves to carry them through the winter and have enough left to provide adequate vitamin A to their offspring. Therefore, it is more common to see vitamin A deficiencies in the spring after significant drought years, due to decreased time for body reserve accumulation. Unlike minerals, much of the vitamin A provided to the neonate is via the colostrum and in milk fats. Also, early calving in beef herds has increased the incidence of neonatal vitamin A deficiencies due to lack of green forage for the cows at the time of parturition.

Vitamin A deficiency is not generally encountered in dairy cows, but is occasionally seen in dairy calves. In vitamin A deficiency cases this author has investigated, most occur in very intensely managed herds. As dairy rations typically have good vitamin A supplementation, one would not expect to see these deficiencies in calves. But the potential for vitamin A loss in the processing of colostrum must be considered. Vitamin A can be broken down by heat. So, although not yet proven, one should consider the possibility that the vitamin A could be lost either in thawing colostrum too fast or when colostrum is pasteurized to prevent spread of disease.

Vitamin A analysis can be efficiently performed on serum or liver tissue. It is important that samples be stored frozen and protected from light to prevent degradation of the vitamin A.

VITAMIN E

Vitamin E is an essential fat soluble vitamin in ruminants. It is essential for all cells as an important antioxidant in the body in conjunction with Se. Vitamin E deficiency is associated with poor growth rates, poor immune function, poor reproductive performance, poor muscle function, poor cardiovascular function, and *white muscle disease*.

Vitamin E is provided in the diet via green growing vegetation or supplementation. Dead, brown forages have relatively no Vitamin E content. Thus, for grazing livestock, they must accumulate enough vitamin E to carry them through the winter and have enough left to provide adequate vitamin E to their offspring. Therefore, it is more common to see vitamin E deficiencies in the spring after significant drought years, due to decreased time for body reserve accumulation. Much of the vitamin E provided to the neonate is via the colostrum and in milk fats, although it is also transferred, a small amount, across the placenta. Also, early calving in

beef herds has increased the incidence of neonatal vitamin E deficiencies due to lack of green forage for the cows at the time of parturition.

Vitamin E deficiency is not generally encountered in dairy cows, but is occasionally seen in dairy calves. In vitamin E deficiency cases this author has investigated, most occur in very intensely managed herds. As dairy rations typically have good vitamin E supplementation, one would not expect to see these deficiencies in calves. But the potential for vitamin E loss in the processing of colostrum must be considered. Vitamin E can be broken down by heat. So, although not yet proven, one should consider the possibility that the vitamin E could be lost either in thawing colostrum too fast or when colostrum is pasteurized to prevent the spread of disease.

Vitamin E analysis can be efficiently performed on serum or liver tissue. It is important that samples be stored frozen and protected from light to prevent degradation of the vitamin E.

EFFECTS ON IMMUNE STATUS

Deficiencies in vitamins and minerals have a 2-part impact on immune function in neonates. Firstly, since neonates are still developing their immune capabilities, these deficiencies have a direct negative impact on that development. And, indirect immune compromise is via the mother's poor immune function. At the time in which it is essential that mothers be immune competent in order to produce antibodies for the colostrum, when inadequately supplemented they are often deficient due to depletion from the movement of minerals to the fetus. Additionally, poor immune function at the time of vaccination can result in very poor vaccine response, which in turn results in poor immune memory and antibody production necessary for good quality colostrum. Thus, herd deficiencies would be expected to result in poor colostrum quality. This poor quality equates to a higher incidence of disease in the offspring due to poor maternal protection. Often this is seen as high incidence of neonatal diarrheas and/or high incidence of neonatal/juvenile pneumonias.

SUMMARY

A variety of samples can be tested for vitamin-mineral content, but may not provide any indication of the overall mineral status of the animal. Appropriate diagnosis of mineral status involves thorough evaluation of groups of animals. The evaluation should include a thorough health history, animal ages, feeding history, supplementation history, and analysis of several animals (at least 6-10 recommended per similarly fed group, depending on the group size) for their mineral status.

Dietary mineral evaluation should only be used to augment the mineral evaluation of animal groups. If minerals are deemed to be adequate in the diet, but the animals are found to be deficient, antagonistic interactive effects of other minerals and true average daily per animal intake of the supplements need to be investigated. As an example, high S or Fe can cause deficiencies in Cu and Se even when there are adequate concentrations in the diet. Or excessive Cu and Se can adversely impact the Zn status. If a free choice supplement is used instead of the supplement being incorporated into a TMR, true intake should be measured.

Overall, common vitamin-mineral deficiencies/excesses are significant hindrances to profitability in the livestock industry. Poor reproductive performance results in increased incidence of culling open cows. Poorer than optimal feed efficiency and productivity impact the bottom line in terms of pounds of milk sales. And, poor calf health results in deaths and disease. The resultant increased disease incidence results in lost income in terms of treatment costs and poorer overall growth rates and productivity in affected animals. Beef herds have been followed where deficiencies were observed, then corrected in which breed-back efficiency has improved by 10 % or more and weaning weight averages have improved by as much as 30 to 70 lb/calf or more. These changes amount to significant improvements in profitability in cattle operations.

In dairy operations, one must correctly identify cause of mineral status abnormalities. For example, if calves are identified to be Cu and Se deficient (indicating deficiencies in the cows) the lactation ration should not immediately have increased supplementation. In several cases the author has investigated, these deficiencies were in herds that were actually OVER-supplementing Cu and Se in the lactation ration. But, the deficiencies were all in calves from first calf heifers which were not being adequately supplemented by the heifer raising facility. Similarly finding excessive Cu and/or Se in multiparous cows does not indicate that mineral supplementation should be cut back for all groups of cows (replacement heifers on dairies are often fed quite different rations than those fed to the lactating herd).

Understanding and Optimizing the Jungle of Bypass Fats

Kevin J. Harvatine, Ph.D.
Penn State University, University Park, PA
Email: kharvatine@psu.edu

ABSTRACT

Nutritionists attempt to meet nutrient requirements and regulate energy balance through dietary interventions. Traditionally, fat supplementation has been used to increase dietary energy density without increasing diet fermentability. Nearly all dietary ingredients contribute some fat to the diet and ingredients with a low fat intake that are fed at high rates are commonly overlooked, but contribute greatly to fat intake. Feeding high fat byproducts and the development of varieties selected for a specific fatty acid (**FA**) profile is quickly changing the FA profile of many fat sources. Fatty acids are well known to be bioactive nutrients that modify metabolism and physiology. The use of lipids as an energy source, substrate for cellular membrane synthesis, substrate for signaling factor synthesis, and their bioactivity make determination of *requirements* difficult. Important aspects of fat supplements are their digestibility, effect on intake and milk production, and their ability to modify physiology. The massive growth in the palm oil industry has broadened the type of products available and highlighted additional differences in metabolism of FA. Selection of fat supplements should consider the basal diet, rumen available fat sources, and the goal of using the fat supplement. Our understanding of FA continues to grow and provides tools to increase production, efficiency, and health of dairy cows.

INTRODUCTION

Lipids are a broad group of compounds that are soluble in organic solvent including waxes, sterols, and compounds that include fatty acids (**FA**) including triglycerides, phospholipids, and glycolipids. Dietary FA are the nutritionally important compoenent of lipids and serve a number of functions in animal nutrition. Fatty acids are a concentrated source of energy, but also serve as integral structural components of cellular membranes and regulatory molecules. Over the past 25 yr we have come to appreciate that some FA are bioactive compounds that modify physiology and metabolism. The dairy cow experiences very different metabolic demands and physiological conditions across lactation and it is reasonable to expect that the role of FA differ during these states.

Palmquist and Jenkins (1980) reviewed the history of fat research in dairy cows starting from a 1907 review of the effect of fat on milk and milk fat yield (Kellner, 1907). It is interesting that over 100 yr later we still are asking some of the same questions, but in the context of a cow with much higher metabolic demands. Interest in fat supplementation has traditionally centered around increasing dietary energy density without increasing dietary fermentability to support energy requirements of *high producing cows*. More recently, interest in fat supplementation has broadened to increasing milk or milk fat yield, increasing reproductive efficiency (Staples et al., 1998), and modifying FA profile of milk (Glasser et al., 2008a; Shingfield et al., 2013). The field of ruminant FA metabolism underwent tremendous growth with the Biohydrogenation Theory of milk fat depression (**MFD**) and the identifaction of bioactive conjugated linoleic acid (**CLA**) isomers. Most recently, availability of enriched palmitic acid supplements provides additional options.

Fat Digestion and Metabolism

Fatty acids are not fermented in the rumen and normally duodenal flow of FA is similar to intake. Rumen microbes synthesize some FA, resulting in ruminal outflow of odd and branch-chain FA. There currently is growing interest in the positive human health attributes of these FA and ruminant meat and milk is the predominant source in the human diet. Ruminal synthesis of FA is increased when feeding low fat diets as the microbes require FA for synthesis of their cellular membranes. The majority of FA in forage and grain feedstuffs are unsaturated and the rumen microbes will biohydrogenate these unsaturated FA forming *trans-FA* intermediates. Complete biohydrogenation results in saturated FA, but biohydrogenation is commonly incomplete. Rumen microbes biohydrogenate unsaturated FA because they are toxic and because they prefer saturated and *trans-FA* for their cellular membranes. The pathways of biohydrogenation are dynamic and responsive to nutritional factors and rumen

environment. Specific *trans-FA* formed in alternate biohydrogenation pathways can cause diet-induced MFD and limits the amount of unsaturated FA that can be fed to dairy cows [see Harvatine et al. (2009) and below]. Biohydrogenation also severely limits absorption of the essential polyunsaturated FA by the cow.

Rumen Availability of FA

Increasing unsaturated FA increases the toxic effect on microbial populations and also increases the substrate required for biohydrogenation. The first step in balancing any nutrient is considering diet concentration and Jenkins (2011) developed the concept of Rumen Unsaturated Fatty Acid Load (**RUFAL**), which is the sum of unsaturated FA in the diet and provides insight into the risk of altering fermentation. Traditionally, the second step in balancing nutrients is consideration of rates. Although considerable effort has been put towards determining rates of fiber and starch digestion in the rumen, little has been directed towards understanding the rate of FA availability. The rate of rumen availability is drastically different between some feeds. For example, unsaturated FA in distiller's grains with solubles (**DGS**) is rapidly available and has a large impact in the rumen compared to whole cottonseed that is slowly released. Previous work has demonstrated that increased grinding of oilseeds increases their risk of diet-induced MFD. Jenkins et al. (2016) recently presented the initial development of a laboratory method to estimate FA availability and future analytical progress in this area is expected.

Calcium salts of FA were developed to reduce the inhibitory effects of unsaturated FA on fiber digestion as they are insoluble salts that block FA metabolism by microbes (Palmquist and Jenkins, 1980). It appears that a main mechanism of calcium salts is slowing rumen availability, rather than true protection, as bypass rates of unsaturated FA fed as calcium salts are rather low. The dissociation of the calcium salt in the rumen is dependent on the dissociation constant of the FA and rumen pH and increasing unsaturation decreases the strength of the calcium salt (Sukhija and Palmquist, 1990). However, calcium salts are far less disruptive to rumen fermentation than feeding a free oil.

Digestibility of FA

Intestinal absorption of FA is quite different in the ruminant as duodenal flow is predominantly saturated free FA. Non-ruminants depend on monoglycerides and unsaturated FA for formation of miscelles, while the ruminant lysolecithin aids formation of micelles as a very potent emulsifier (Doreau and Chilliard, 1997). In the ruminant, there is a large decrease in total tract digestibility when saturated triglycerides (**TG**) are fed as they are more resistant to ruminal and intestinal lipolysis than unsaturated TG (Elliott et al., 1994; Elliott et al., 1999). There is significant variation in total tract digestibility in the literature that reflects both variation between diets and the technical challenges of digestion studies. Total tract FA absorption is roughly between 70 and 80 % in dairy cows. Differences in digestibility of individual FA is controversial and is difficult to investigate because of rumen and hindgut biohydrogenation. Meta-analysis using different approaches have observed little difference in digestibility between FA, although FA digestibility decreases with increasing fat intake (Glasser et al., 2008b; Schmidely et al., 2008; Boerman et al., 2015). More attention should be paid to FA digestibility, but will require a dedicated effort to conduct well-controlled experiments.

Metabolic Fate of FA

Fatty acids can be oxidized to provide energy for maintenance and production and provide 2.5 times more energy than carbohydrate. Fatty acids can also be used for body storage and milk fat production and are energetically efficient processes as FA can be directly deposited and do not have any energy loss. The metabolic fate of absorbed FA is expected to depend on the physiological state of the cow and the FA. During peak lactation FA are directed towards meeting energy requirements for milk production. In some cases, fat supplementation increases milk fat yield and the response appears to be dependent on FA profile. Kadegowda et al. (2008) observed a 243 g/d increase in milk fat yield with abomasal infusion of 400 g/d of butter oil and the increase was predominantly short and medium chain FA. More recently, milk fat responses have been commonly reported when feeding enriched palmitic acid [C16:0; (Mosley et al., 2007)], but also have been observed with oilseeds and other FA supplements (Weiss and Pinos-Rodriguez, 2009). After peak lactation, dietary FA will be increasingly partitioned towards body reserves. Importantly, oxidization of FA spares other nutrients from oxidation, which creates a complicated discussion of the metabolic impact of dietary FA.

Essential FA

Fatty acids can be categorized as essential or nonessential based on the animals capacity to synthesize or conserve the required amounts and

linoleic (C18:2 n-6) and linolenic (C18:3 n-3) acid are traditionally considered the two essential FA (Cunnane, 2000). Some consider the very long chain omega-3 FA (e.g. eicosapentaeonic acid (**EPA)** and docosahexaenoic (**DHA**)) to be conditionally essential as they can be synthesized by elongation and desaturation, but the capacity of their synthesis is highly limited in most production animals. There is overlap in the ability to utilize omega-3 and omega-6 FA as substrate in some pathways. However, signaling molecules originating from omega-3 are more anti-inflammatory and omega-6 FA are more pro-inflammatory. This overlap and competition for elongation and desaturation has led to the concept of omega-3 to omega-6 ratios, although the importance of these measures is still uncertain.

The requirement for essential FA is different based on what is required for maintenance and sustained production vs. the amount that may stimulate maximum production through changing physiology and metabolism. The first definition is easier to define based on metabolic use, but the second demands an understanding of the physiological and metabolic effect of individual FA. This includes their effect on hard to research processes such as immunology and reproduction. Essential FA have been a subject of conversation in many species, including ruminants, for many years. Absorption of essential FA is very limited in ruminants, but there are no reports of classical FA deficiency in adult ruminants and Mattos and Palmquist (1977) determined that linoleic acid was available to the cow at twice the requirement for female weanling rats on a metabolic body weight basis. In addition, ruminants may be adapted to conserving essential FA as they are less available for oxidation (Drackley, 2000). It appears that essential FA are normally available in adequate concentrations based on production requirements, however there may be benefits to FA supplementation to health including improving reproductive efficiency and immunology.

Effect on Intake

A main goal of fat supplementation is to increase energy intake and depression of dry matter intake (**DMI**) can limit the benefits of fat supplements. Intake is highly regulated by animal nutrient requirements and metabolic state, and also by the type and temporal pattern of fuels absorbed (Allen, 2000). Fat source, form, and FA profile are significant predictors of intake response. In a meta-analysis, Allen (2000) reported a linear decrease in intake with calcium salts of palm distillate, while saturated FA had no effect on intake. Benson et al. (2001) summarized 11 infusion studies and observed a negative relationship between infused C18:1 and C18:2 FA concentration and intake, with C18:2 creating greater intake depression. Abomasal infusions of unsaturated FA with a lower C16:C18 FA ratio decreased DMI and energy intake (Drackley et al., 1992, Christensen et al., 1994). Finally, 4-d continuous intravenous infusion of both palmitic and oleic acid significantly decreased intake, while stearic acid only numerically decreased intake (Vandermeerschen-Doize and Paquay, 1984). Recent work with enriched palmitic acid supplements have observed decreased intake compared to no fat controls (Lock et al., 2013; Rico et al., 2014a), although the overall decrease in DMI was not significant and energy intake was increased in a recent meta-analysis (deSouza et al., 2016)

Fats in the Basal Diet

Nearly all feed ingredients contain FA and it is best to think about diet FA starting with the base diet through high fat feeds and then fat supplements. Feeds vary in type and FA profile and have different effects in the rumen. Forages and cereal grains have a low concentration of fat, but their high feeding rates make them a major dietary source of FA. Oilseeds, high fat byproduct feeds, and liquid fats are economical sources of FA, but care must be taken to not disrupt rumen fermentation. Lastly, dry fats are convenient to add on farm and provide the opportunity to customize absorbed FA profile, but are expensive and differ greatly in FA profile, risks, and benefits.

Forages

Lipids in forages are predominantly in the plant leaf in the form of glycolipids. Total FA concentration in forages is only around 50 % of the ether extract value because of the large non-FA content of glycolipids. Fatty acids in forages are highly unsaturated and normally contain more than 50 % α-linolenic acid (C18:3 n3, reviewed by Glasser et al., 2013). Forages would be a great source of essential FA, but they are readily available in the rumen and extensively biohydrogenated. Grasses contain higher levels of FA in the early growth stages (can exceed 5 %) and are a common culprit in diet-induced MFD with intensive grazing. Lastly, wilting and drying before harvest decreases the availability of unsaturated FA in forages because of the formation of indigestible resins.

Cereal Grains

Corn, wheat, barley, and oats all have rather similar FA profiles and contain approximately 55 % linoleic acid (C18:2 n-6) and less than 1 % omega-3 FA. Corn grain is higher in total fat than small grains and varies with variety including specially bred high-oil corn. In a recent characterization of a field test of 36 commercial hybrids we observed a range of 3.3 to 3.9 % total FA (10^{th} to 90^{th} percentile) and 55.7 to 60.0 % linoleic acid (C18:2 n-6). In corn, the majority of the FA is in the germ and it is expected that processing methods that increase rate of digestion will increase the rate of rumen availability of the unsaturated FA.

Corn Silage

Corn silage is a mixture of grain and forage and thus has a combination of the forage and grain attributes discussed above. We recently found that 80 % of the total fat and over 90 % of the oleic (C18:1 n-9) and linoleic (C18:2 n-6) was found in the kernel and over 70 % of the α-linolenic acid (18:3 n-3) was in the leaves. Therefore, grain concentration is going to impact the FA concentration and profile. Additionally, we expect that unsaturated FA in the kernel are rapidly available in well processed and ensiled silage. We also observed moderate variation in FA concentrations and profiles of corn silage, which was raised in test plots, with C18:2 ranging from 0.94 and 1.60 % of DM (10^{th} and 90^{th} percentile). Fatty acid profile of corn silage is going to be highly dependent on genetics. Routine analysis is probably not needed, but it is advisable to determine each crop's profile or when trouble-shooting diet-induced MFD.

Oilseeds

Oilseeds are commonly an economical and convenient method to increase FA intake. The FA are highly unsaturated and are mostly found in triglycerides in the fruit contained inside the seed coat. The seed coat and processing method dictates the rate of rumen availability, which has a large impact on the associative effect of the FA on the rumen. Although the release rate of FA in the rumen can be decreased by less aggressive processing, oilseed unsaturated FA are normally extensively biohydrogenated and it is difficult to bypass unsaturated FA in oilseeds.

Expeller oilseed meals are normally higher in fat (~ 9 %) than solvent extracted meals (< 3 %), but depends on the seed, processing plant, and batch. Some facilities may also add phospholipids and free recovered oil back to the meal, which may change rumen availability and risk for oxidative rancidity.

Oilseed FA profile has and continues to undergo strong genetic selection to modify FA profile for human health and processing characteristic. For example, canola is a low erucic acid (C22:1) rapeseed. To aid the movement away from partially hydrogenated frying oils, low α-linolenic acid (18:3 n-3) soybean oil was developed in the mid-2000s, which allows increased oil frying life and oxidative stability. The next step has been development of high-oleic acid soybeans (> 70 % C18:1) that are currently under limited commercial production in the US. Once final approvals in key export countries are received it is expected to become a major part of the US soybean acreage. These specialty oilseeds are commonly processed in specific facilities allowing identification, but as the market grows they may become mixed within the commodity market.

Byproducts

Many high fat byproduct feeds are available at a reasonable cost, including DGS and bakery byproducts. These byproducts vary considerable in amount and profile of FA. The FA in many of these byproducts is rapidly available. For example, condensed corn solubles is a high fat stream in distillers plants that is added back to distillers grains in making DGS. This fat is thus adhered to the outside of the grain and rapidly available in the rumen. Arguably, many of the issues with diet-induced MFD when DGS is fed may be due to the rapid availability of the unsaturated FA and may not be the amount of unsaturated FA. Many ethanol plants are now recovering some of the lipid to be sold as oil, which has decreased fat concentration of DGS. The key element to any byproduct feed is knowing the source and managing the variation to take maximal advantage of their value.

Liquid Fats

Liquid fats can be an economical source of FA, but require specialized equipment. Liquid fats adhere to feed particles and are expected to be rapidly available in the rumen. Liquid fats vary in their FA profile depending on their source. Importantly, the FA profile of lard and tallow changes as feeding practices change and the recent increase in DGS feeding has increase unsaturated FA in these oils. Changes in oilseed FA profile also impacts vegetable oil streams. Quality can be an issue in liquid fats as unsaturated FA are more susceptible to oxidation

once extracted and some processing streams include heating. Antioxidants are commonly added to liquid fats, especially when they are highly unsaturated. Measuring unsaponifiable matter can also provide some indication of the quality of the fat.

Dry Fat Supplements

Dry fat supplements are convenient because they are concentrated sources of FA that are easy to handle on farm. They differ greatly in their source, FA profile, and metabolic effects. Some dry fat supplements may melt in extreme temperature conditions.

Prilled Saturated Fats

Saturated FA are naturally ruminal inert as they are not toxic to microbes and do not require biohydrogenation. The first major difference in prilled fats is their free FA concentration. Saturated TG are poorly digested as they are not hydrolyzed in the rumen and the cow has poor lipase activity in the intestine. Most prilled supplements on the market are high free FA products (80 to 99 %) and decreased digestibility would be expected in products that are high in TG. The second major difference is FA profile. Traditionally, prilled products were a mixture of palmitic and stearic with a lower concentration of oleic. More recently, enriched palmitic acid (> 80 % C16:0) products have become available as a byproduct of palm oil manufacturing. Additional differences exist in FA source and manufacturing. For example, saturated FA can be enriched by separation from unsaturated FA or unsaturated oils or made by partial or full hydrogenation of unsaturated FA. Hydrogenation adds the risk of presence of bioactive *trans*-FA, if the process is not complete, and some methods may result in addition of small amounts of metals used as a catalyst. Also, some plant-based sources have an increased risk for contamination of residues including dioxins. Prill size can also differ between manufacturing processes and the impact on digestibility has not been extensively investigated, but appears to be minor.

Prilled free FA blends of palmitic and stearic acid have the longest history in the literature and generally do not decrease DMI and are well digested. Enriched palmitic acid products (80 to 90 % C16:0) have been extensively investigated in the past 6 yr and generally result in a small increase in milk fat (~ 0.2-unit increase when fed at 1.5 to 2 % of diet) and are also well digested. Limited research has been done with highly enriched palmitic and stearic acid products [> 95 %; (Piantoni et al., 2013; Rico et al., 2014b; Piantoni et al., 2015)]. The highly enriched product used in these experiments decreased diet FA digestibility considerably, although it is unclear if this is attributed to specific attributes of the product fed, such as prill size, or the high enrichment.

Calcium Salts of FA

Calcium salts of palm FA was developed in the 1980's to allow feeding unsaturated FA without negative effects on fiber digestion. Traditionally, calcium salts were made from palm oil distillate, but specialty blends that include n-6 and n-3 FA are now available. More recently, there has been interest in using calcium salts to protect unsaturated FA in the rumen and increase essential FA absorption. Calcium salts are the only method currently available to increase rumen bypass of unsaturated FA, however their effectiveness is limited. The release of highly unsaturated FA in the rumen increases the risk of diet-induced MFD when feeding calcium salts enriched in polyunsaturated FA compared to feeding a prilled saturated FA supplement.

Recommendation on Fat Feeding

The strategy for balancing FA in a diet should be based on your goal and the feeds available. It is best if total dietary FA does not exceed 7 % of DM. First consideration should be given to the FA entering from forages and cereal grains and note the rate of availability of these FA. Next, use economical sources of rumen available FA including high fat byproducts, liquid fats, and oilseeds. It is especially important to note the profile and rate of availability of these sources and their interaction with the basal diet. The amount of these that can be incorporated will be limited as to not disrupt rumen fermentation and cause diet-induced MFD. Lastly, dry fat supplements should be selected based on your goal. For example, supplements highly enriched in palmitic acid provide the most consistent increase in milk fat yield, prilled saturated blends of palmitic and stearic may increase energy intake most effectively, and calcium salts have the potential to increase unsaturated FA flow to the duodenum, although the level of protection is low.

CONCLUSIONS

Fat supplementation continues to evolve with changes in oilseed FA profile through selection and new dry fat supplements available from palm oil processing. Fatty acids have been appreciated as

bioactive FA for some time with great interest in CLA-induced MFD and essential FA, but there also appears to be differences between saturated FA. We expect that we will continue to move towards balancing for specific FA as our knowledge of ruminal biohydrogenation, specific roles of individual FA, and strategies to protect unsaturated FA improve.

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Update on Regional Extension Programs

Ellen Jordan, Ph.D. and Robert Hagevoort, Ph.D.
Texas A&M AgriLife Extension Service and New Mexico State University Extension
Email: e-jordan2@tamu.edu and dairydoc@ad.nmsu.edu

METABOLIC PROFILE RESEARCH UPDATE

Research was conducted to evaluate the relationship between dietary intake and various parameters of the metabolic profile. Blood samples were collected at the morning feeding during 3 wk before and 3 wk after calving (N=4129 dairy cows) in 8 Holstein herds in the summer and winter, as well as in 8 Jersey herds in the summer. The samples were refrigerated, processed, and stored at -20 °C until laboratory analysis. On the day of sampling, total mixed ration samples were collected for subsequent analysis. Associations were tested by logistic regression (pregnancy at 90 [**P90**] and 150 days in milk (**DIM**) [**P150**]), correlation analysis (dietary and metabolic profile concentrations), and analysis of variance (days to first service [**DFS**] and days open [**DO**]). Herd records were collected and health events were extracted from herd records including dystocia, still births, twins, retained placenta, hypocalcemia, ketosis, and mastitis. Cows that were over-

Table 1. Mean and SD ration composition during the prepartum period.

Variable	Winter Holsteins		Summer Holsteins		Summer Jerseys	
	Mean	SD	Mean	SD	Mean	SD
P	0.38625	0.06589	0.347778	0.04868	0.445	0.12286
DM	56.625	6.97705	54.85556	5.13423	53.61	7.89774
CP	15.15	1.18442	14.6	1.63018	15.26	1.45082
Adj. Prot	15.025	1.28702	14.28889	2.04539	15.18	1.60333
Sol. Prot.	38.5125	7.257	36.61111	4.65657	39.72	6.31379
RDP	69.275	3.62127	68.3	2.32648	69.87	3.15667
ADF	27.875	2.51779	27.54444	25.7	25.89	6.14373
NDF	41.425	3.46894	40.4	5.54256	38.32	6.85578
Ash	10.2625	1.55833	10.85556	2.52543	10.58	1.24704
Ca	1.29875	0.27247	1.296667	0.35408	1.58	0.44021
Mg	0.38625	0.06323	0.411111	0.07737	0.44	0.18233
K	1.54125	0.3943	1.4288889	0.23872	1.732	0.51166
Na	0.189125	0.07249	0.134778	0.06699	0.1535	0.06668
Fe	443	268.44845	553.1111	241.4014	402.9	79.13905
Mn	91.875	36.56867	98.7778	36.43754	94.7	45.44851
Zn	93.125	35.89046	99.55556	27.31351	114	53.15596
Cu	23	6.78233	27.33333	12.22702	24.5	8.33
TDN	63.9	4.20912	63.87778	5.479	65.6	4.4292
NEL	0.664286	0.05192	0.663333	0.06403	0.685	0.05017
NEM	0.651429	0.0623	0.65	0.08109	0.678	0.06546
NEG	0.385714	0.05855	0.385556	0.0735	0.408	0.05808
NFC	29.87143	3.35147	31.47778	4.77566	32.79	6.1209

conditioned or under-conditioned and evidence of lameness was noted at sampling.

The mean and standard deviation (**SD**) for the ration composition by season for the Holsteins and for summer sampling for the Jerseys are shown in Table 1 for the prepartum period and Table 2 for the postpartum period.

The time of sample collection was categorized into weeks relative to calving (D 0) with wk -3 from D -24 to D – 18; wk -2 from D -17 to D -11; wk -1 from D -10 to D -4; wk 0 from D -3 to D 3; wk 1 from D 4 to D 10; wk 2 from D 11 to D 17; and wk 3 from D 18 to D 24. Pearson correlation coefficients were determined between serum values for magnesium (Mg), potassium (K), calcium (Ca), sodium (Na), and phosphorus (P) and the mineral level in the prepartum or postpartum ration associated with the herd of origin, season, and day relative to calving. Pearson correlation coefficients between serum urea and albumin concentration and ration concentration of adjusted protein, soluble protein (**SP**), rumen degradable protein (**RDP**), and crude protein (**CP**) were determined. Correlations were calculated for the individual weeks of wk -3, -2, -1, 1, 2, and 3; for the combined prepartum period of wk -3, -2 and -1; for the combined postpartum period of wk 1, 2, and 3; and over the entire period except week 0.

For serum urea, the highest correlation coefficients were with ration CP ($P < 0.0001$), particularly during the prepartum period. Although serum albumin was significantly correlated with SP, RDP, and CP; the correlations were not strong, ranging from 0.15 to -0.06. Serum Ca and ration Ca,

Table 2. Mean and SD ration analyses during the postpartum period.

Variable	Winter Holsteins		Summer Holsteins		Summer Jerseys	
	Mean	SD	Mean	SD	Mean	SD
P	0.4225	0.03196	0.42125	0.03314	0.467	0.07319
DM	54.225	3.39779	55.125	5.89619	56.31	6.38356
CP	17.8	0.53436	16.8	0.65027	17.53	0.80422
Adj. Prot	17.0375	0.53436	16.8	0.65027	17.53	0.80422
Sol. Prot.	36.5125	3.61956	35.5875	1.72746	36.29	2.18604
RDP	68.25	1.81265	67.775	0.86644	68.14	1.07311
ADF	23.475	2.59216	22.775	1.56821	21.72	1.47784
NDF	34.45	2.53659	33.6	33.05	32.61	2.36711
Ash	8.875	1.02085	9.325	1.61842	8.64	0.95242
Ca	1.22375	0.27959	1.16375	0.38497	1.148	0.22885
Mg	0.40625	0.05097	0.37125	0.02416	0.39	0.04216
K	1.5575	0.1353	1.57	0.19864	1.699	0.17673
Na	0.346125	0.11877	0.4	0.13517	0.3436	0.07435
Fe	308.875	68.36757	339	129.1798	311.2	69.08578
Mn	77.25	30.79773	66.125	13.81963	60.6	22.13695
Zn	88.125	20.08153	78.875	13.3142	75.1	21.60478
Cu	23.875	7.98995	21.375	6.04595	22.2	7.05219
TDN	71	2.69921	71.175	2.03523	72.54	1.42299
NEL	0.74625	0.03159	0.74875	0.02475	0.765	0.0178
NEM	0.75375	0.03815	0.7575	0.02765	0.778	0.02044
NEG	0.47875	0.03357	0.48	0.02449	0.496	0.01713
NFC	34.9875	3.06288	35.575	3.386	36.21	2.99683

were correlated over all weeks (excluding wk 0), for wk -2, wk -1, wk 3, prepartum and postpartum with the highest correlation of 0.155 occurring in wk -2. Ration and serum K concentrations were correlated over all weeks (excluding wk 0), wk 1, wk 2, and postpartum. Over all Mg was correlated; however, serum Mg in the postpartum period, combined or by individual week, was not correlated. The correlation was approaching significance ($P < 0.102$) by week 3, although the correlation was low (0.086). In the prepartum period, Na tended to have a slightly negative correlation (-0.04, $P < 0.097$) and overall the correlation was negative ($P < 0.0001$); however postpartum as a group or by individual week correlations between ration and serum concentrations were not significant.

Ration and serum P concentrations were correlated for all time periods evaluated, although the highest correlation was 0.166. Previously we reported to this group that when serum from Holstein cows within the transition period were evaluated for P on a week by week basis (week zero = -3 d prepartum to 3 d post calving), week 1 was the only week where serum P values differed (Lager et al., 2011). This would mean that P levels decrease around 10 d postpartum. There was not an effect of number of lactation. Further, our group collected samples from summer and winter to provide seasonal analysis and it was discovered that P levels are impacted by season.

CONCLUSION

The metabolic profile is a useful tool that has evolved over time. This evolution or adaptation is necessary to account for changes in feeding management and animal genetics. The key is ensuring that the reference values match the stage of lactation. From this evaluation, it is apparent that correlations do exist between ration nutrients and some parameters analyzed as part of the metabolic profile; however, during the transition period evaluated these correlations although significant aren't strong, which may be related to the changes in nutrient composition which occurs during this 6 wk period. Because of the prevalence of disease within the periparturient period, a reference profile based on mid-lactation cows limits the interpretation of data from cows within the transition period. Due to the number of ration manipulations frequently occurring during this period some analytes may not be reflective of the current nutrition program and over interpretation should be avoided. There is certainly a need to understand the fluctuations that occur in serum biochemical analytes over the course of a lactation, especially within the transition period. With recent data displaying an impact of breed and season on the metabolic profile as well as the documented variability within the transition period, it may be necessary to account for each factor as well as be cognizant of when a sample is collected to be sure that the results received will be of value.

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