Common nutritional deficiencies and how they affect bovine reproduction

Dry matter intake (DMI) is probably the most essential factor for managing reproduction in postpartum cows.¹ The following reviews several critical components of DMI management that require close attention:

Rumen acidosis

Undoubtedly the most common health problem for most dairy herds, rumen acidosis stems from multiple factors and is characterized by a rumen pH of less than 6.0. The low pH results from excessive lactic and propionic acid produced by the rumen microflora, relative to buffering by bicarbonate-containing saliva produced during cud chewing. The acids damage papillae that line the rumen wall, allowing bacteria to escape into the bloodstream. These bacteria settle in vascular areas where they become trapped. They cause significant pathology, forming abscesses in the liver, lungs, hoof lamina, and subcutaneous tissue.

Due to histamine-induced laminitis and sole abscesses, the affected cows become lame, compounding their health problems. Lame cows often eat poorly, lose body condition, and become more susceptible to other conditions. They may also experience a delayed estrous cycle, which may cause anestrus and delayed conception.

In the event a cow with rumen acidosis exhibits estrus and conceives, the blastocyst is often smaller than normal at nine to 12 days, resulting in early attrition, often referred to as early embryonic death.

Several management tools can help prevent rumen acidosis. These include:

- Ascertaining moisture content of the total ration to prevent sorting by the cows
- Determining particle size of the ration with the Penn State Particle Separator Box
- Providing optimum cow comfort in the heat of summer

For complete information on rumen acidosis prevention, dairy producers should consult with a nutritionist or veterinarian who provides nutritional services.
Energy management and/or high body condition score

Excessive condition during the dry period often results in fatty infiltration of the liver and other internal organs, which become surrounded by adipose tissue – even when a cow’s body condition score may be considered optimum, at 3.5. Fatty infiltration of the liver, which can also occur rapidly in the last few days prior to calving, may result in development of fewer pre-freshening follicles. Transition dry cows should be supplied a dietary energy maintenance level of 1.3 MegCal/kg, in contrast to a lactating cow receiving 1.65 MegCal/kg of energy.

A herd with an ineffective transition dry cow program is easy to identify. The cows close to calving often have high body condition scores and are commonly diagnosed with “fat cow syndrome” after calving. Cows that survive being too fat at calving often develop extremely low body condition scores a few weeks later. This results in low milk production, subsequent anestrus, and lower fertility.

Rumen-protected choline and niacin are useful products to add to the transition TMR and the early lactation TMR to aid in metabolizing excess abdominal fat. Positive ramifications of choline supplementation will be discussed further in this paper.

Dietary cation-anion difference (DCAD)

Dietary cation-anion difference in transition dairy cows’ diets during the last three weeks before calving is one of the most recently developed tools to help cows calve and lactate successfully. The DCAD formula for determination is \((K^+ + Na^+ – Cl^- + SO_4^-)\). This is a method for creating metabolic acidosis in a cow by causing the DCAD to stay in a range between -5 and -15.

Cows normally have a blood pH of 7.4. Excessive anionic chlorides without cations and sulfates drops the blood pH, creating acidosis. The cow’s metabolic system responds by pulling calcium from the bones to stabilize blood pH. Increasing the blood calcium level to 8.0-10.0 mg/dl has many benefits. It prevents clinical and subclinical hypocalcemia. If either of these conditions occurs, a cow is susceptible to retained placenta, ketosis, and displaced abomasum. In addition, her immune response is depressed, resulting in increased susceptibility to metritis and mastitis immediately after calving. Calcium is integral to maintaining a strong immune response. It is also essential for muscle function and bone integrity.

A cow near calving requires 0.8% calcium in her diet, when no modifications are made for an anionic diet. Close-up cows (last 21 days prior to calving) should receive 1.0% calcium, provided that an anionic salt is included for a calculated DCAD of -5 to -15. Weekly monitoring of urine pH is essential to
ensure acidosis occurs with a urine pH of 5.6-6.8. Be aware that a pH outside this range may lead to metabolic disorders.

Inadequate calcium in the diet, irrespective of the DCAD, results in reduced immunity and poor muscle tone, especially in the smooth muscles of the abomasum and uterus. Uterine immunity is as essential as smooth muscle tone.

Subclinical low blood calcium of 7.5 or lower causes a reduced defense mechanism in the uterus. This leads to more profound issues with puerperal metritis. Treatment of sub-hypocalcemia may be corrected by intravenous administration of calcium borogluconate or calcium boluses. Acute hypocalcemia is most effectively remedied by intravenous calcium. When subclinical hypocalcemia is diagnosed, an evaluation of the diet is advised to determine changes made in forage calcium values to adjust the DCAD. The ration DCAD should be modified to -5 to -15, using an anionic salt such as BioChlor™, Animate™, or SoyChlor™.

To summarize the overall nutritional strategy for managing hypocalcemia and immune responses: Lower the energy level of the ration by incorporating 3-5 kg of wheat straw/cow/day, chopped at 2.0-3.5 cm and blended with the remaining ingredients. Provide no more than a kilogram of corn and minerals to supply a DCAD of -5 to -15, a vitamin-mineral mix to fulfill the nutritional requirements, 0.1 kg of rumen bypass fat and 14% crude protein for a total mix ration (TMR). No high potassium legume haylage should be fed, unless the DCAD can be maintained within the optimum negative DCAD range. Eight to ten kilograms of corn silage should be included. The TMR should be blended and fed fresh each day to promote full consumption. Large breed dairy cows often consume 13 kg of the transition cow TMR daily. In contrast, cows consume 9-10 kg of feed daily when not on a low energy anionic diet. The major contrast between the transition diet and a lactating diet is reduced energy (close-up cows on TMR consume 1.30 MegCal/kg as compared to lactating cows consuming 1.65 MegCal/kg). This encourages more dry matter consumption by late pregnancy cows.

Phosphorus deficiency and reproduction

Phosphorus (P) is intricately related to many metabolic mechanisms in the cow, such as the workings of the calcium-to-phosphorus ratio.

Historically, soils were low in P, but not so much today. Fertilization practices and advanced crop production methods have eliminated low P levels in forages fed to cows. This issue is referred to as “legacy phosphorus.” Many farms have an abundance of soil P. This results in winter run-off into streams and lakes, promoting algae blooms that create oxygen “dead spots” in bodies of water.

Phosphorus is a key element in many enzymatic and endocrine processes including trace mineral absorption and utilization. It plays an integral role with calcium in bone growth and maintenance.

The bovine requirement for dietary P is 0.35-0.40%. Many ruminant diets have a surplus of P. Phosphorus deficiency is rarely diagnosed today. If P deficiency occurs, it manifests as anestrus or at least “silent” estrus. In rare instances, P deficiency plays a role in down cows, appearing as clinical hypocalcemia. A relatively rare condition of acute P deficiency is described as post-parturient hemoglobinuria.

A diagnosis of P deficiency is made with forage and concentrate analyses and a calculation of the cow’s dietary intake. Blood tests are mostly ineffectual for diagnosing P deficiency. Correcting the ration formulations generally resolves issues related to low dietary P.

Copper deficiency

Copper deficiency and toxicity present an enigma. The best illustration of this is copper’s relationship to molybdenum when fed to sheep. When seven mg/kg of dietary copper are fed to sheep with an inherent level of molybdenum under 1.5 mg/kg, toxicity results. This leads to a hemolytic crisis and potential death. In contrast, feeding seven mg/kg of dietary copper to sheep with dietary molybdenum of more than 1.5 mg/kg will cause sheep to manifest copper deficiency signs. While cattle aren’t as sensitive to toxicity or deficiency as sheep, a similar paradox exists. Jersey cattle are similar to
sheep in regard to dietary copper and molybdenum. Adult Jerseys should not be fed more than 15 mg/kg of dietary copper unless dietary molybdenum levels are elevated.

Copper status in the bovine affects several enzyme systems. Copper-containing ceruloplasmin is a key enzyme for the synthesis of reticulocytes. Low levels of ceruloplasmin result in anemia. Copper is essential for the enzyme system lysyl oxidase, also known as amine oxidase. Lysyl oxidase is essential for the cartilaginous matrix in which bone osteoblasts lay calcium to provide structural rigidity to bone. A lack of copper causes a loss of integrity in the cartilaginous matrix. This makes young calves susceptible to long bone fractures. This enzymatic system is also necessary for the cartilaginous matrix in the hoof. Deficiency causes heel cracks to develop, resulting in lameness.

Lysyl oxidase plays a key role in maintaining myocardial fibrils. Deficiency during cardiac muscle fiber maintenance results in fibrotic tissue replacing myofibrils, causing what appear to be heart attacks. This malady is often referred to as “falling disease,” because dairymen report perfectly healthy cows falling over dead. Observed more often in lambs than calves, amine oxidase is essential for the formation of elastin in the arterial intima of the great vessels such as the aorta and pulmonary arteries. These animals develop aortic or pulmonary aneurysms because of defective elastin in the arterial walls and are diagnosed at necropsy with a blown aneurysm.

Copper is essential to the pigment enzyme tyrosinase, which converts tyrosine to melanin in the black hair of cattle. Copper deficiency causes the hair to fade and appear reddish, as though it will soon shed.

Another copper-containing enzyme is cytochrome oxidase, which is essential for myelination of the brain stem and spinal cord. Termed “a-myelination,” it is similar to demyelinating diseases, but in this case, affected calves are born of copper-deficient dams. Such calves are unable to nurse and often have a “star-gazing” look. In my experience these calves never learn to nurse, and in the case of beef calves, never manage to find the dam’s teat. This is often a herd problem.

Finally, animals affected by copper deficiency often have a reduced defense mechanism. The mechanism at work here has not been fully described, but one biological system involved relates to Cu,Zn superoxide dismutase. Copper-zinc superoxide dismutase is an intracellular system that is essential in reducing free oxygen radicals as they are metabolically produced. Copper-zinc superoxide dismutase reduces free oxidative radicals intracellularly to hydrogen peroxide. Hydrogen peroxide is then further reduced by selenium-dependent glutathione peroxidase to water and oxygen. Selenium will be addressed further later in this paper. Vitamin E also can and does reduce hydrogen peroxide, similar to Cu,Zn superoxide dismutase.

Several interactions with copper can interfere with copper availability. These include iron, sulfates/molybdenum, and zinc. (See Nutritional toxicities and reproduction, Sanders, SFT Annual Conference 2017.)

The copper nutritional requirement depends upon many other interactions, varying with the presence and level of other minerals. As mentioned previously, sulfate and molybdenum nutritional levels greatly impact the requirement for dietary copper. Requirements: sheep, ~7 ppm; Jerseys, 10-15 mg/kg; and other dairy breeds, 15-25 mg/kg, depending on Mo status when above 1.5 mg/kg and sulfates in water when above 350 mg/kg. Treatment of deficiency includes a Multimin™90 injection and correction of dietary levels with supplementation of chelated copper sources or copper sulfate. Copper oxide is not an acceptable source of copper because of its low biological availability.

Manganese deficiency

Manganese (Mn) typically is not included in a differential diagnosis list because Mn deficiency is considered rare. However, glyphosate herbicide has been reported to reduce the uptake of Mn by row crops. In a couple of instances, this author has confirmed Mn deficiency in swine fed GMO corn, although the GMO corn was not validated to be the cause of the Mn deficiency.

Manganese has also been identified as an element that may be incorporated into the superoxide dismutase intracellular system for scavenging. Diagnosis of Mn deficiency, in this author’s opinion, is
difficult because confirmed serum deficiency levels of 0.005 mg/kg⁴ are at the low end of the spectrum detectable in a laboratory.

Signs of Mn deficiency include silent estrus, reduced conception, abortions, reduced birth weight, skeletal changes in growing calves, weak calves, and cystic ovaries. Calcium, cadmium, cobalt, iron, and phosphorus are antagonistic to Mn. High Mn causes calcium retention in the fetus. Mn deficiency is rarely diagnosed, yet when manganese assays are performed, manganese frequently is in the range considered deficient.⁶

Manganese uptake naturally occurs from the soil, but is reduced by soil liming. Manganese sulfate is 100% available. The requirement in cattle is 40-200 mg/kg. The treatment of Mn deficiency is readily resolved with Multimin™90 injections and increased dietary MnSO₄ to 40-200 mg/kg.

Zinc deficiency
Zinc is linked closely with Cu in superoxide dismutase, to capture free oxygen radicals for conversion to hydrogen peroxide. Zinc is also important for hoof and skin integrity and integral to more than 200 proteins and zinc metallothionein enzyme systems.⁵ Parakeratosis is common in zinc-deficient calves and causes stiffness and shortening of bones, increased pododermatitis, reduced conception rate, severely impaired spermatozoan maturation, reduced feed intake, and growth rate, apparently related to reduced insulin levels. A genetic defect in Holsteins (A46) causes inability to absorb Zn from the intestine

Diagnosis of deficiency is readily performed through blood serum tests. Levels of 0.20-0.40 mg/kg are deficient with test reports under 0.60mg/kg considered deficient. (Special blood tubes should be used for mineral assays, as the rubber stoppers of conventional tubes contain zinc, skewing the results.)

Excessive zinc reduces calcium metabolism and vice versa. Copper and iron are also antagonistic. In addition, low zinc inhibits vitamin A absorption, low P increases tissue Zn, high Al reduces Zn absorption, and monensin enhances Zn absorption.

Good sources of supplementation include Multimin™90 injectable, Zn oxide, Zn sulfate, and Zn methionine.

Vitamin E/selenium deficiency
Vitamin E, as alpha-Tocopherol, has long been noted to be closely interactive with selenium. While one can replace most of the other in muscle-related activities, this is not totally the case for other metabolic activity. Vitamin E and selenium perform related activities in the muscle action cycle. Selenium is a component of selenium-dependent glutathione peroxidase (GSH-Px), acting as an antioxidant during release of energy for muscle action. It reduces hydrogen peroxide radicals from Cu,Zn superoxide dismutase to water and oxygen and stimulates production of immunoglobulin M (IgM) antibody, producing cells that positively impact the immune system.

Selenium and vitamin E perform related functions in the muscles. One can replace the other, to an extent. Selenium is an essential element in production of an essential amino acid, cysteine. Selenium is mostly deficient in 44 states, but toxicity is possible in some areas in the western U.S. where soils and selenium accumulator plants contain high levels of selenium.

Injectable forms of vitamin E and selenium include Bo-Se™, Mu-Se™, and Multimin™90 (contains Se but no vitamin E). Dietary sources include inorganic sodium selenite-90, sodium selenite-200, selenomethionine, and sodium selenate. The requirement is 4-6 mg of Se per day for an adult dairy cow, 1,000-2,000 IU/day of vitamin E daily, and 4,000-6,000 IU of injectable vitamin E at calving is very useful.

Signs of deficiency include white muscle disease/cardiac failure in calves, abomasal ulcers, elevated SCC, cystic ovaries, reduced fertility, retained placenta, and oxidized flavor to milk.⁶

Selenium deficiency can be diagnosed through several tests including serum selenium (0.80.300 mg/kg, whole blood selenium 0.200-1.200 mg/kg, Se-GSH-Px 19.0-36.0 umoles/min. at 37°/mg Hb). A diagnosis of vitamin E deficiency can be confirmed with serum vitamin E. Normal values are 300-1,000 ūg/dl for adult cows.
Treatment of selenium deficiency in young calves is by injectable Bo-Se; in adults, with Mu-Se; or in the diet, with sodium selenite 90 mg/lb or selenium chelates. Adult dairy cows benefit from injectable vitamin E or 1,000-2,000 IU/day of dietary vitamin E supplementation.

Vitamin A deficiency

Vitamin A is a key element in colostrum, as the newborn has no reserve vitamin A stores at birth. Vitamin A is needed for growth, appetite, prevention of night blindness, follicular/testicular development, cell replication, and maintenance of epithelia integrity and mucous secretions. Several conditions affect vitamin A availability. Zinc deficiency and high dietary Ca negatively affect vitamin A utilization, as do increased nitrates in drought conditions and high ambient temperatures. Salt and urea reduce stability of vitamin A supplements, and long storage periods adversely affect vitamin A stability and utilization.

B-carotene from green leafy forages such as alfalfa is a precursor of vitamin A. One milligram of B-carotene is equivalent to 400 IU of vitamin A. Other sources of vitamin A include vitamin A retinyl palmitate and injectable vitamin A-D.

The daily requirement for vitamin A in dry cows is 60,000-100,000 IU, and for lactating cows, 53,000-173,000 IU. Supplying twice the recommended dietary level can help compensate for interfering factors and negative interactions.

Signs of vitamin A deficiency include abortions, still births, weak calves, retained placenta, reduced fertility in bulls, reduced conception in cows, poor appetite, reduced weight gains, rough hair coat, reduced defense mechanism, increased pneumonia rate, watery eyes, blindness or night blindness, diarrhea, and swollen joints or brisket.

Deficiency can be diagnosed with the incidence of night blindness, decreased appetite, poor growth, dermatosis, and pneumonia. Adult cattle may have anasarca, mastitis, infertility, and night blindness. Analysis of serum vitamin A is useful for a diagnosis, but must be conducted within one hour of a blood draw – which precludes this option for most diagnosticians.

Treatment of vitamin A deficiency is most easily managed with injectable vitamin A-D. Raising dietary vitamin A levels also is advantageous.

Choline

Little historic research supports the importance of choline for cows. However, this is changing. Rumen-protected choline (RPC) is believed to be liver and calcium sparing, playing a role in increasing fat metabolism.

Rumen-protected choline is highly recommended, as choline must be protected from rumen microbes. Cows can be supplemented during the transition and fresh periods with a commercially available feed additive such as ReaShure® from Balchem.

Data suggest that 15 gm/cow/day of RPC will provide a positive 2 kg/day response in milk production with less ketosis.

Heavy conditioned cows have lower milk production and higher incidence of ketosis. Fat cow syndrome is considered a classic symptom of choline deficiency. Energy should be fed at minimum maintenance levels of 1.3 MegCal/kg. During transition time and early lactation, cows should be fed a DCAD diet and RPC at 15 gm/cow/day.

Choline-supplemented cows have higher IgG levels in their colostrum. These cows also have a statistically significant higher level of blood calcium. Heifer calves born from RPC-supplemented cows were 31 lb. heavier at 12 months of age (even though they were smaller at birth (P <0.05).

Methionine

Methionine is known to interact with rumen-protected lysine with an ideal ratio for optimum performance of 3:1, rumen-protected lysine (RPL) to rumen-protected methionine (RPM). Methionine increases lipid content of blastocyst when fed three weeks prepartum to 30 days postpartum.
Methionine is closely related to choline. It has been observed that choline can spare methionine and methionine can spare choline in transition cows. However, feeding methionine without choline will not aid in the prevention of fatty liver degeneration. Two products that provide rumen-protected methionine are Smartamine™ and Megalac plus™.

Signs of methionine deficiency are subtle. They include a lower milk protein test and an increased rate of early embryonic death. Nutrition modeling software is used to diagnose deficiency. A deficiency is corrected by feeding methionine in the transition ration, continued for 60 days after calving.

References