

Ketosis

Definition

Ketosis or acetonemia is a disorder associated with carbohydrate fat metabolism. The disease is characterized by increased blood ketones, which eventually spill into urine and milk. Blood glucose levels also drop below normal levels.

Ketosis affects high producing cows during the first 6-8 weeks of lactation when cows are in negative energy balance. Ketosis occurs when energy intake fails to meet the requirements for high glucose production, necessary for maintenance and milk lactose production. In clinical ketosis, excess ketone bodies are produced from incomplete fatty acid breakdown of body tissue. Fat is also deposited in the liver. The disease takes two forms:

1. Primary - No apparent predisposing disease condition.
2. Secondary - Accompanied by some other complicating disease. When an elevated temperature accompanies a ketosis, complicating diseases are implicated. Approximately one-third of ketosis cases are of the secondary type.

Symptoms

1. Loss of appetite. Grain is refused first, followed by silage. Hay continues to be consumed.
2. Rapid decrease in milk production.
3. Rapid body weight loss.
4. General depression, rough hair coat, sunken, glazed eyes. Some cows may become nervous or excitable (termed Nervous Ketosis).
5. Odor of acetone on breath.
6. Elevated ketone bodies in blood, urine, and milk.
7. Decrease in blood glucose. Normal value = 50 mg. glucose/100 mg. blood. Values below 40 mg/100 mg blood are subnormal.

Below are excerpts from a paper recently written by Dr Gary Oetzel, UW School of Veterinary Medicine.

Ketosis is often poorly defined in dairy herds which makes it difficult to compare the incidence and prevalence. Sub-clinical ketosis (SCK) can be defined objectively using what Dr Oetzel calls the gold standard. That being blood betahydroxybutyrate (BHB). The threshold concentration of 1400 umol/l (14.4 mg/dl) defines SCK.

Prevalence should be determined by sampling 12 or more early lactation cows (between 5 and 50 days). Published studies report a range of 8 to 34% prevalence. Dr Oetzel's data over the last three years has shown a prevalence of 12.4%. His recommendation is to set the alarm for a problem at greater than 10%. The incidence of ketosis in dairy herds is about 2 to 4 times the measured prevalence because the duration of SCK can be short, and many cows that have or will have SCK have normal BHB concentrations when the herd is tested. Blood BHB concentrations of clinical ketosis will range from 2600 to 6000 umol/l.



Dr Oetzel has classified ketosis in two types:

Type I is when energy output is greater than energy input. These cows typically did not have difficulty in the pre fresh period, calve normally, and start their lactation by milking well. The limiting factor is the supply of glucose precursors (mostly propionate and partially amino acids). Cows generally respond well to treatment. The key to prevention is to maximize energy intake in early lactation without causing subacute ruminal acidosis (SARA). Fat supplementation does increase energy but does little for ketosis prevention because fat is not a glucose precursor. In fact, fat may have a negative effect by adding more fatty acids to a struggling liver.

Over crowding and lack of bunk space can be another cause of insufficient energy intake. Overfeeding protein and underfeeding energy in post-fresh groups is another cause. Low fiber or sortable diets lead to SARA that lowers DMI and feed efficiency.

Type II ketosis encompasses any cow that develops negative energy balance pre calving due to a reduction in DMI. Fat cows are at the highest risk. The primary cause is the normal reduction in DMI prior to calving. The key to success in prevention of type II ketosis is to minimize the drop in intake during this time. (This appears to be the type most producers are dealing with.)

The literature is showing that NEFA levels are correlated with the degree of energy decline pre calving. For example - If one cow is on a high energy pre fresh ration and another cow is on a moderate energy pre fresh ration and they both drop in intake at the same rate, cows on the high energy ration will have higher NEFA levels because the degree of energy drop is greater.

The goal is to provide a diet that cows readily consume to maintain DMI as long as possible. That means the best possible forages available on the farm and prevent feeding ingredients that are unpalatable. Having said that, now you need to evaluate whether anionic products are needed based on the type of diet fed and their impact on DMI. We have had great success with DCAD balancing however, its not going to work 100% of the time.

Maternity area separate from the pre fresh area can also increase type II ketosis. Simply moving a cow to a different pen depresses DMI for several days. Again, overcrowding, lack of bunk space, poor cow comfort, cow handling, etc. all play a role. The quality of the post fresh management has little bearing on the risk for type II ketosis. Affected cows were programmed to get ketosis by the time they calve.

Blood ketone concentrations are not as high in type II versus type I, yet the prognosis for recovery in type II cases following treatment is poor, because treatment does little to improve the cow's underlying lesion of fatty liver infiltration and loss of the livers ability to produce glucose. Not only does this lead to persistent ketosis, but also immune suppression causing secondary problems.

Excellent pre fresh management is key. Maximize DMI pre fresh. Balance energy according to our current guidelines. Optimum body condition at freshening is 3.5. Use the best quality feeds available. Consider using glucose precursors, niacin (minimizes body fat mobilization), Reashure (exports fat from the liver), Yeast Fortifier, Microlac, MicroPrime (stimulate DMI).



One of the challenges on farms is producers who test for ketosis using keto test strips, or keto powder. Most agree that urine testing is more accurate than milk. Most cows will show signs of ketosis after calving, many will recover on their own. Overreacting and treating every cow that shows positive is problematic. As mentioned, some cows will recover on their own. In fact the stress of treatment may have a negative impact. In addition the test will show false positives (cows that really don't have ketosis but the test shows that they do). Because of this producers may think they have a serious problem when it may not exist. How do you determine if the problem is real. If there are secondary problems then it is real. Also use other cow observations. Monitor feed intake, cud chewing, manure consistency, temps and general thriftiness in conjunction with keto testing to determine if treatment is necessary.

Treatment and Recovery

Most cows will recover when milk production drops to a point where glucose requirements are met by glucose formation from feed. Intravenous injection of 50% glucose solution will increase blood glucose. Glucocorticoid injections will enable production of blood glucose from amino acids. Propylene glycol may also be fed at 8-16 oz per head daily.

Nutritional Prevention

1. Monitor cow condition. Excessive body fat inhibits appetite after freshening, impairs liver function and prolongs fatty acid mobilization. In contrast, thin cows do not have enough condition to support milk production, exasperating glucose demands.
2. Begin transitioning dry cows to the milking ration 2-3 weeks prior to freshening. Grain feeding should be gradually increased to about .5% of body weight on a dry matter basis. After freshening, increase grain feeding as rapidly as possible without causing off-feed problems (typically 24-30 days). Feed small amounts of grain several times throughout the day. Maintain forage intake at a minimum of 40% of dry matter.
3. Provide adequate amounts of protein to stimulate intake and provide amino acids for milk protein synthesis. Excessive protein will increase energy requirements and needs to be avoided.
4. Feed top quality forages to stimulate intake and provide energy. Avoid silages high in butyric acid - this discourages intake and also contributes to ketone production.
5. Feed sufficient quantities of minerals and vitamins in both dry and lactating diets.
6. Research suggests that 6-12 grams of niacin, starting 14 days before and continuing for 45-70 days after freshening, may help prevent ketosis. Overconditioned cows and cows predisposed to ketosis appear to benefit the most.
7. Propylene glycol or sodium propionate can be administered orally. These glucose procurers may need to be drenched due to their unpalatable nature. Recommended doses are 4-8 ounces starting 1-2 weeks before calving and continuing 6-8 weeks postpartum. Propylene glycol is the preferred choice because it is used directly by the liver for glucose synthesis and tends to be more palatable.

