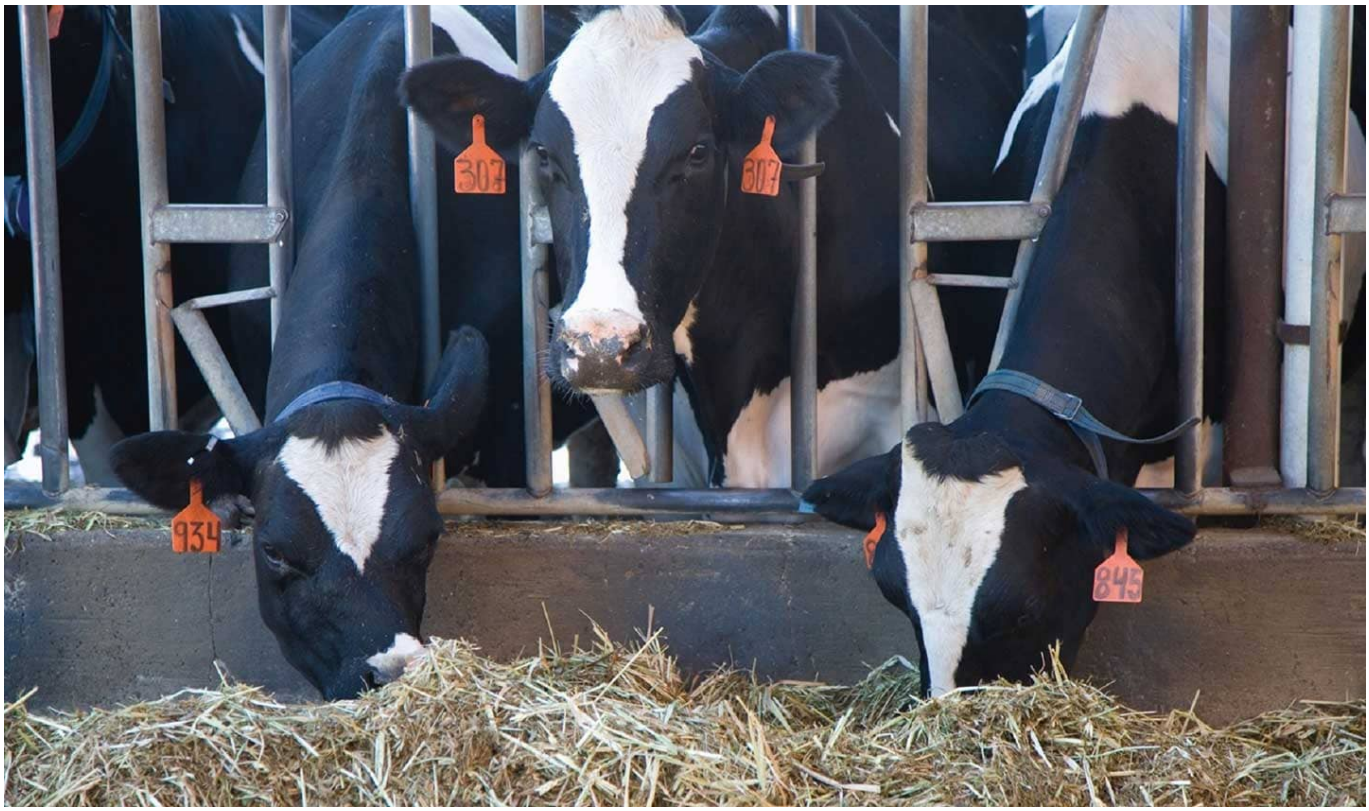
Ketosis: the most critical metabolic disease in dairy cows



***Judith Schmidt**, Product Manager On-Farm Solutions*

Improvements in genetics, nutrition, and management continue to enhance dairy cows' performance. However, being high-performance athletes comes at a cost, putting an extremely high burden on the animals' energy metabolism. Especially around calving and during the first eight weeks of lactation, dairy cows can experience many stress factors: subclinical hypocalcemia, abomasum displacements, herd composition changes, or lameness. The more stress factors put the cows' organism under pressure, the more likely they will become sick. A common consequence of stress is the occurrence of metabolic diseases, especially ketosis.

Both in terms of animal health and economic aspects, ketosis is probably the most critical dairy cow disease when also considering the correlated diseases. In this article, we explore the causes and consequences of ketosis and highlight prevention strategies that keep this issue under control.

Ketosis: causes and consequences

How ketosis develops

A restricted feed intake capacity and/or reduced energy concentration in the ration lead to a deficit in the animal's energy balance. This situation occurs, for instance, at calving when the mother animal focuses her resources on the calf and its care. To compensate for the energy deficit, body fat is broken down for energy production. This process creates free fatty acids that accumulate in the liver and are partially converted into ketone bodies. These ketone bodies are a "transport medium" for energy, which various organs can use as an alternative energy source.

The problem arises when the deficiency lasts too long: more and more body fat is broken down, more and

more fatty acids reach the liver, which leads to a fatty liver, and too high an amount of ketone bodies is formed and released into the blood. The ketone bodies in the blood inhibit appetite, resulting in less feed consumption and an energy deficit – the vicious cycle of ketosis begins.

Subclinical ketosis

Subclinical ketosis is defined as the stage of the disease at which an increased level of ketone bodies can be detected in the blood, urine, and milk. Furthermore, signs of hypoglycemia, increased levels of non-esterified fatty acid, and decreased hepatic gluconeogenesis can be seen in the blood. These conditions are typically not detected because there are no clinical signs.

Subclinical ketosis is a problem as it does not cause visible symptoms but leads to an increased incidence of subsequent diseases such as lab stomach displacement, clinical ketosis, and uterine inflammation. In addition, there may be loss of milk and fertility problems. Subclinically ill animals cannot be identified by the farmer by observation alone. Therefore, subclinical ketosis must be detected at an early stage to be able to act at the right time: prophylaxis instead of therapy.

There are several test possibilities to find out if an animal suffers from ketosis:

1. **Milk**: Milk test for ketosis detection has been available for many years. The results are to be obtained based on a color gamut. In contrast to blood analysis, the milk test does not evaluate exact values but shows a color change of the contained indicator. However, an increased milk cell content of the feeding of poorly fermented silages with a high butyric acid content significantly influences the result. The test often does not adequately reflect the actual conditions.
2. **Urine**: Another possibility is the examination of urine samples. Urine can be obtained spontaneously or with the help of a catheter. The results can also be read on a color scale of the urine test stripes. Like the milk test, the urine test only distinguishes different concentration ranges, but these are more finely graded than in the milk tests.
3. **Blood**: The most accurate but also most complex and expensive method is a blood test. It has the advantage that not only ketone bodies but also other parameters such as free fatty acids, minerals, and liver enzymes can be analyzed. In addition, the blood analysis results are evaluated in numbers and are more comparable than the color changes of test stripes. A good alternative is a rapid test by using a rapid test device, which is also used for measuring human blood sugar. A result is displayed with a drop of blood on a test strip within a few seconds.

Clinical ketosis

Depending on why there are elevated ketone body levels in the blood, we distinguish between primary and secondary clinical ketosis. For the primary form of clinical ketosis, the energy deficit itself (due to high performance and/or incorrect feeding) causes the condition. This form mainly occurs in susceptible, high-yielding dairy cows between the second and seventh weeks of lactation (Vicente et al., 2014). Secondary ketosis is caused indirectly by other diseases disease. A cow suffering from, for example, a claw disease might no longer consume a performance-based feed ration, leading to an energy deficit.

Typical symptoms

Typical of metabolic diseases, ketosis leads to a broad spectrum of symptoms. The classic symptoms at the beginning of the disease are a loss of appetite and decreased milk performance. As the disease develops, motor skills may be affected, and the excrement’s consistency becomes firmer and darker in color. The respiratory rate of sick animals increases, and they show dyspnea. Dyspnea is the medical description for breathing difficulties. Affected animals suffer from air shortage, which can occur in different situations. Due to the excretion of ketone bodies via the mucous membranes, the animals’ breath smells more or less strongly of acetone (Robinson and Williamson, 1977).

In addition, the animals undergo rapid and severe weight loss, and their general body conditions deteriorate noticeably. Furthermore, cows suffering from ketosis show increased milk fat content or an increased milk fat/protein quotient. Clinical symptoms include reduced general well-being, apathy, blindness, staggering, persistent “absent-minded” licking of the environment or overexcitability, muscle tremors, and aggressiveness (Andersson, 1984).

Effects on animal health and performance

Even in its subclinical form – if untreated – ketosis will engender health risks and reduced performance, negatively impacting milk yield and cows' fertility. For clinical cases, typical effects include infertility, udder and hoof problems, and a fatty liver. Ketosis during early lactation is usually associated with fatty liver disease. In severe cases, the liver becomes enlarged and more fragile. It then no longer performs its detoxification function, toxic compounds increase, and the central nervous system is damaged. Anorexia or even a total loss of consciousness, the so-called hepatic coma, might ensue, ending in a complete liver function failure.

Direct economic costs range from high veterinary costs to the total loss of the dairy cow, i.e., approximately € 600 to € 1.000 per cow. Moreover, producers face indirect costs from secondary diseases such as fatty liver disease, increased postpartum behavior such as uterine infections, abomasum dislocations, or claw diseases.

Ketosis prevention: feeding and targeted supplementation

Feeding strategy

As part of the preparatory feeding, both dry and pregnant cows should receive rations that lead to an optimal (and not maximum) body condition at the time of calving. Animals with a poorer nutritional status do not have enough body fat reserves to compensate for lack of energy in the first phase of lactation. In more cases, animals have a too high BCS, leading to a risk of difficult births, and the cows have too little appetite at the beginning of lactation. These cows tend to show an excessive mobilization of fat reserves and develop a fatty liver. So prevention of ketosis of the current lactation starts with preventing a too-high BCS in the middle of the previous lactation.

The aim of feeding measures is to keep the lactating cow's discrepancy between nutrient requirements and nutrient uptake as low as possible when the genetically determined performance potential is exhausted. For this reason, the ration must have a certain minimum energy density (high-quality forage and appropriate concentrate supplements). Also, anything that prevents the cows from ingesting the maximum amount of dry matter should be avoided.

Ket-o-Vital bolus for metabolic support

Another important preventive measure is the specific support of the calving cow's liver, rumen, and immune system. EW Nutrition's Ket-o-Vital Bolus was explicitly designed to reduce the risk of ketosis. It contains fast-available glucogenic substances, positively influencing the cow's energy metabolism. Another advantage the bolus offers is the slow release of the contained cobalt, selenium, niacin, and active yeast:

- Cobalt is a trace element important to form cobalamin, the so-called vitamin B12. It is essential for blood formation and the functioning of the nervous system.
- Selenium protects cells from oxidative damage and ensures an intact immune defense;
- Niacin is a B vitamin that intervenes in energy metabolism and prevents fatty liver syndrome;
- And active yeast supports rumen health, preventing rumen acidosis and increasing feed intake.

The application of the Ket-o-Vital Bolus is profitable and straightforward. Only one bolus per application is required.

Ketosis control: be one step ahead

High-performance dairy cows are at risk of ketosis, which results in involuntary culling, poor health, and performance losses. Advanced feed management practices combined with the targeted use of the Ket-o-Vital bolus offer a solution for preventing this debilitating disease. The bolus protects the cows from clinical and subclinical ketosis, reduces metabolic disorders, increases appetite, and improves health – leading to a quick recovery and ensuring profitable production.

References

Vicente, Fernando, María Luisa Rodríguez, Adela Martínez-Fernández, Ana Soldado, Alejandro Argamentería, Mario Peláez, and Begoña de la Roza-Delgado. “Subclinical ketosis on dairy cows in transition period in farms with contrasting butyric acid contents in silages.” *The Scientific World Journal* 2014 (November 25, 2014): 1–4. https://doi.org/10.1155/2014/279614.

Andersson, L. “Concentrations of blood and milk ketone bodies, blood isopropanol and plasma glucose in dairy cows in relation to the degree of hyperketonaemia and clinical signs*.” Zentralblatt für Veterinärmedizin Reihe A 31, no. 1-10 (1984): 683–93. https://doi.org/10.1111/j.1439-0442.1984.tb01327.x.

Robinson, A. M., and D. H. Williamson. “Effects of acetoacetate administration on glucose metabolism in mammary gland of fed lactating rats.” Biochemical Journal 164, no. 3 (1977): 749–52. https://doi.org/10.1042/bj1640749.