Ketosis: the most critical metabolic disease in dairy cows

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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.

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4 steps to improve dairy cow fertility through feeding

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The average pregnancy rate for dairy cows has declined over the past decades. But why is my cow not getting pregnant? Is it because of feeding? These are questions we ask ourselves when things do not quite work out with the offspring in the cowshed. Economic success in the cow barn is closely related to the successful reproduction of our cattle herd.

The maintenance and possible improvement of fertility are becoming increasingly important issues for farm productivity. Infertility is still one of the main reasons for culling on dairy farms. When farmers decide to cull a cow after a few unsuccessful inseminations, they often ask themselves whether this could not have been prevented. There is no “all-encompassing” solution for achieving an optimal fertility rate, which ultimately requires excellent management. Relevant factors include oestrus monitoring and insemination timing, genetic conditions, feeding, hygiene, and climate.

How can I tell if a cow is in heat?

A cow behaves differently than usual during oestrus. She is restless and walks around more. A cow in heat stands next to other cows – head to tail. Sie also quarrels with her herd mates or sniffs at the shame of the other cows. Fertility in cows decreases during late winter and spring; the resulting absence of clear signs of oestrus makes it difficult to recognize the right time for insemination. There are several possible causes which will be reviewed below.

Possible causes of fertility problems in dairy cows

Beta-carotene deficiency

A productive herd needs to receive an optimal mineral and trace element supply. Beta-carotene, in particular, is essential for herd fertility. But why?

Beta-carotene is an orange-yellow plant pigment whose name comes from “carrot” because of its appearance. It is also a precursor of vitamin A. Both as a precursor and as vitamin A itself, it is essential for the organism of humans and animals, particularly when it comes to the fertility of dairy cows. Besides its important function as provitamin A, beta-carotene also exerts an independent effect on the ovary. It influences the quality of the follicle and the corpus luteum. Beta-carotene also protects the corpus luteum. It promotes the synthesis of the pregnancy hormone progesterone and thus enables the fertilized egg to implant successfully in the uterine lining.

A beta-carotene deficiency can lead to the following problems:

- Smaller, not fully functional follicles
- Altered oestrus intervals
- Indistinct signs of oestrus
- Decreased corpus luteum quality

Scientific trials show how much a beta-carotene deficiency influences the fertility process. With a beta-carotene deficiency, the fertilization rate after the first insemination is only 40%, whereas with a normal beta-carotene supply, the fertilization rate is about 70%.

How do I know if my herd is deficient in beta-carotene?

The easiest way is to check the color of the fresh colostrum. If it is a deep yellow to an even orange, the cows are supplied with sufficient beta-carotene. If it looks more ivory, this is a sign of a deficiency. Of course, a poor herd fertilization rate can also indicate a deficiency. If you suspect a beta-carotene deficiency, it is best to test some blood samples from your animal or use a testing device such as a carotene photometer. With such a test kit, you can determine not only the levels in the blood but also in the colostrum and the milk.

Feeding deficiencies

Feeding plays a major role in fertility issues. Too low input rates often have a negative effect on the health of cows. Feed quality and herd management have an impact on how long the cow loses weight after calving and at what point she gains weight again. One must always keep in mind the cows’ feeding, energy balance, and nutrient supply because cows with a negative energy balance often do not show oestrus. It is also important that the silage is of high quality – poor silage inhibits fertility.

Follicle quality

The quality of the follicle is crucial for good fertility. The quality is influenced by the energy supply during the dry period and lactation during the first days. Since the follicles are already formed in the last days of gestation, a lack of energy during this period means that the maturation of the follicles – even with a better supply later on – can no longer proceed optimally and is ultimately inferior. This inevitably leads to a reduction of oestrus symptoms and minimizes the chances of successful insemination.

Prevention is key: 4 steps to improve fertility through feeding

1) Avoid stress in the feeding environment

Well-being and a high feed intake are the basis for high milk and fattening yields as well as healthy and fertile animals. Dry cows and transit cows particularly should only experience low stress. This means no overcrowding and generous feeding space, i.e., each animal should have its own feeding space. Feeding areas that are too narrow prevent the animals from eating, rank fights occur, and feed intake decreases.

Freshly lactating cows should be separated from the group. If the cows are in calving pens or calving stables, they should always have visual contact with the herd.

2) Optimize feed quality and rations

Feed quality and feeding management determine how long the cow loses weight after calving (negative energy balance) and at what point the cow gains weight again (positive energy balance). Optimal fertility performance can only happen when a positive energy balance is achieved.

The cow’s fertility performance is primarily determined by nutrient supply and feeding. At the beginning of the lactation, high-quality basic feed with a high energy concentration should be fed, as feed intake is slow to get going after calving. Nevertheless, this ration should have sufficient structure. The amounts of

concentrate should be divided into several individual portions and carefully increased. For high feed intakes, fresh water should be constantly available to the animals.

3) Treat diseases early to enable feeding

Diseases that lead to a reduced appetite should be treated as early as possible. In particular, attention should be paid to healthy hooves because a cow that has pain or difficulty getting up and walking is much less likely to go to the feed table.

4) Supplement vitamins, minerals, and trace elements

The needs-based supply of vitamins, minerals, and trace elements in every performance phase is a decisive success factor for good herd fertility. A sufficient supply of trace elements, especially selenium, manganese, zinc, as well as vitamin A and beta-carotene, are important for the formation of fertility hormones and optimal insemination success. At the same time, they ensure a high colostrum quality.

EW Nutrition’s Fertilgol Bolus is a long-term bolus to support fertility. The high content of beta-carotene has a positive influence on the formation of the corpus luteum, the oestrus cycle, the quality of colostrum and sperm. The release rate of the ingredients beta-carotene, selenium, vitamin A, and other trace elements takes place over at least twenty days. Fertilgol Bolus can be used for female and male breeding animals shortly before and during the breeding or insemination period.