Session 07: Metabolic status and risk of disease

Ketosis in dairy cows: Prevention and therapy
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Introduction Ketosis in dairy cows is characterized by increased levels of circulating ketone bodies, particularly of beta-hydroxybutyrate (BHB), as a result of the inability of the liver to metabolize an excess of non-esterified fatty acids (NEFA) which is typically observed shortly after calving. In this period the energy intake of the cow increases more slowly than the milk energy output does ultimately resulting in a negative energy balance (NEB) and a stimulated lipolysis. Thus, the p.p. lipomobilization syndrome is accompanied by hyperketonaemia and hepatolipidosis (e.g. [1]). Based on the absence or presence of clinical symptoms in combination with the levels of BHB in blood circulation the hyperketonaemia is classified as either subclinical or clinical ketosis irrespective of pathogenesis. Most authors define a subclinical ketosis at BHB concentrations between ≥1.2 and ≤2.0 mMol/L although these thresholds should be considered a convention (e.g. [2-4]). Clinical signs are often paralleled by BHB concentrations of >2.0 mMol/L although they might be absent even at higher BHB levels. Considering this classification it becomes clear that a medical treatment is mostly confined to clinical cases while subclinical cases usually are not discovered and consequently not treated. Preventive measures need to be implemented in view of the significant incidence of subclinical ketosis, its adverse impact on dry matter intake and milk yield, and its association with clinical ketosis and other productions diseases, such as displaced abomasum, metritis and mastitis [2] but also with vaccination success ([5]). Based on the fact that a subclinical ketosis is at high risk to derail into a clinical ketosis prevention of the subclinical form concurrently reduces the incidence of clinical cases and should be the method of choice.

Prevention Preventive measures can be derived from factors putting cows at risk of development of a ketosis. Thus, energy intake shortly after calving should be stimulated while milk energy output could be decreased ultimately resulting in a less pronounced NEB. Energy intake is usually stimulated by gradual increase of the energy concentration of the ration within a period of approximately 2 weeks by means of increasing concentrate feed proportions. At the same time, high-quality roughage needs to be offered ensuring both a high energy concentration [6] and sufficient physically effective neutral detergent fibre (peNDF) (e.g. [7]). The latter is particularly important to stimulate rumination and to avoid ruminal disorders such as subacute ruminal acidosis (SARA). Other measures to improve the energy supply include optimization of the dietary starch content resistant to rumen degradation in a way that such an amount of ingested starch reaches the small intestine which can be digested and absorbed without flooding the hindgut by undegraded starch. As bovine gluconeogenesis relies on rumen originating propionate feedstuffs consisting of gluconeogenesis its precursors such as propylene glycol are widely used around calving, either as a part of the ration or as drench, in order to provide substrates for gluconeogenesis whereby ketogenic pathways of metabolic intermediates are downregulated. Not only an increased energy supply has the potential to decrease the magnitude of the NEB but also a decreased milk energy output. However, a decreased milk energy output during the first days after parturition by milking the cows only once a day is less popular because of long-persisting adverse effects on milk yield although this method is the most effective way to minimize p.p. NEB. Therefore, alternatives for decreasing the milk energy output are aimed at decreasing the energy concentration of the milk and at the same time maintaining the milk yield. The milk fat content can effectively be reduced by feeding conjugated linoleic acids (CLA) which has a marked effect on the energy content of milk due to the high gross energy concentration of fat compared to carbohydrates and proteins. However, literature results are inconsistent regarding the net effect of feeding CLA on NEB showing in some cases a parallel increase in milk yield or a decrease in dry matter intake (DMI). Besides energy intake and milk energy output, the extent of mobilization of body reserves also influences the magnitude of NEB. However, particularly an excessive lipomobilization is known to trigger ketogenesis. Cows overconditioned at parturition are at higher risk for such a condition. Body condition score (BCS) is a suitable indicator for production, reproduction and health of cows. It is simple to determine and particularly the loss of body condition after calving, expressed in BCS points, is related to NEB [8]. It was suggested that for many production and health traits the association to BCS was non-linear [8] which might hinder its usefulness as a predictor for all nutritional and metabolic states. Nevertheless, the optimal BCS according to a 5-point-scale ranges between 3.0 to 3.25; a lower BCS is associated to a compromised production and reproduction whereas a BCS higher than 3.5 is related to a reduced dry matter intake shortly after parturition and to an increased susceptibility to metabolic diseases, most notably to ketosis [8]. Thus, avoiding a too high BCS prior to calving is one of the key elements in preventive feeding strategies. Based on the common practice to offer diets as total mixed rations (TMR) for ad libitum consumption through the whole production cycle it is not always feasible to feed individual cows or groups of cows according to a target BCS. Grouping might even become questionable when the herd is too small. An alternative might be offering partial mixed rations consisting of roughage and a small portion of concentrate feed while most of the concentrate feed is assigned individually according to actual performance and/or BCS through transponder regulated automatic feeders. Although such a way of feeding is close to precision feeding the technical and personal expenditure still needs to be optimized.

Therapy Therapeutic measures that are used to treat a clinical ketosis might include dextrose infusion, treatment with dexamethasone, insulin, a butaphophan-cyanocobalamin combination product, and further measures discussed above; particularly oral propylene glycol treatment (for review see [9]).

References