Avoid adipose tissue inflammation

[Dairy Physiology Series – Schothorst Feed Research]

Adipose (fat) tissue not only serves as energy storage in the body, it can also modulate metabolism and immune function. Inflammation of adipose tissue may aggravate negative energy balance, loss in body condition score, and risk of metabolic disorders in transition phase dairy cows over subsequent parities.

Adipose tissue

Adipose tissue depots are highly diverse and composed of adipocytes (fat cells) embedded in an extracellular matrix, immune cells, fibroblasts, vascular cells, and stem cells and adipocytes progenitors. Besides, the adipose tissue also serves as an endocrine organ. Adipose tissue depots are found adjacent to many organs, with evidence of regional crosstalk. An example is the periovarian fat, which exerts paracrine control of folliculogenesis and, in monogastric animals, the excessive inflammation of this adipose tissue depot can lead to reproductive failure.

In case of metabolic disease, where pro-inflammatory processes are involved, immune cells promote a shift in adipocyte function and signalling. This results in increased lipolysis (breakdown of triglycerides), reduced insulin sensitivity, altered adipokines profiles, and changes in signalling profiles of released lipids. Moreover, the metabolic and inflammatory roles of adipose tissue overlap.

In healthy dairy cows, macrophages are about 6-13% of the cells in visceral depots. In cows with metabolic diseases such as clinical ketosis, macrophages can make up 20% of adipose tissue and are drivers of the inflammatory response observed in these cases.

Adipose tissue and energy metabolism

The role of adipose tissue in maintaining basic energy requirements during fasting periods is clear, through lipolysis of triglycerides and release of free fatty acids. Another product of lipolysis, glycerol, can be used as substrate for liver gluconeogenesis in such fasting states. Adipose tissue is also important for nutrient buffering in a fed state. Whenever excess nutrients are available, insulin triggers anabolic processes in the adipose tissue, driving the uptake of metabolites for de novo fatty acid and triglycerides synthesis. An example of such a situation in a dairy cow's productive life would be towards the end of the lactation period, when the nutrient requirements for milk production are decreasing and the intake of nutrients is increasing, resulting in increased body condition.

Transition period

Around calving, dairy cows go through a period of negative energy balance: Energy requirements rise sharply after parturition because of the increasing milk production, and dry matter intake cannot keep up. Therefore, the cow needs to mobilize fat from her body reserves to fulfil her energy requirement. This intense lipid mobilization triggers an inflammatory response in the adipose tissue, doubling the number of macrophages in adipose tissue depots. In mice models, it was shown that the number of immune cells accumulated during the lipid mobilization phase does not fully return to basal values during the lipid repletion phase. In dairy cows, this implies that the population of macrophages will still be higher than 'normal' in late lactation and during the dry period. This may result in a more exacerbated recruitment of immune cells during the next period of rapid lipolysis (the subsequent parturition and early lactation).

Adipose tissue inflammation

The consequences of inflamed adipose tissue are systemic, with reports of impairment of liver function and systemic glucose homeostasis. Therefore, in a disease state, the inflammatory state of the adipose tissue limits the metabolic flexibility of the animal, as it can impact the flow of nutrient availability and buffering capacity.

In dairy cattle, adipose tissue mobilization occurs in the early lactation period and tissue repletion recovers body condition score and weight during late lactation and dry period. As this cycle repeats over parities, the proportion of immune cells in the adipose tissue increases and decreases concomitantly, however, the number of macrophages in the adipose tissue does not completely return to pre-parturition levels. The higher proportion of immune cells at the next calving turns the adipose tissue more immune reactive, exacerbating the immune response and cell recruitment in the following cycles of tissue mobilization and repletion, reducing the cow's resilience to disease challenge over time.

Practical relevance

From practical dairy farm experiences, it is known that the magnitude of body weight loss in the first month of lactation increases with each parity, and so does the risk of developing subclinical and clinical metabolic diseases. In addition, cows that consistently lose more body weight than others in the early lactation period have signs of glucose intolerance as well poorer fertility. A proposed explanation for the link between body condition score losses and disease occurrence is that metabolites from the lipolysis could mediate immune cell function, thus resulting in poor pathogen clearance and increasing the conditions to development of chronic inflammatory processes. Adipose tissue inflammation itself can also have negative effects on immune function. But the net accumulation of macrophages in adipose tissue during the lifespan of a dairy cow could be associated with the greater risk of metabolic diseases in postpartum dairy cows of increasing parities.

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