Optimising rumen function to manage heat stress and milk fat depression

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Cows under heat stress often suffer milk fat depression (MFD) and scientific understanding of this phenomenon is far from complete. However, it appears possible that interaction between heat stress and the ration impacts MFD and optimising rumen function may help reduce MFD effects.

To understand how heat stress can lead to MFD, it helps to review milk fat synthesis in the cow and potential mechanisms for MFD during heat stress.

Milk fat synthesis

Fatty acids for milk fat synthesis originate from two sources. Long-chain fatty acids (greater than 16 carbon atoms per molecule) derive from the uptake of circulating preformed fatty acids, dietary fat absorbed from the digestive tract, and non-esterified fatty acids (NEFA) from the mobilisation of body fat reserves. Short-chain (4-8 carbons) and medium-chain (10-14 carbons) fatty acids originate in the mammary gland from de novo synthesis. The 16-carbon fatty acids can originate from both sources.

For a well fed cow, an estimated 4-8% of milk fatty acids originate from the breakdown of body fat (NEFA). However, contribution from this source could increase progressively as the net energy balance decreases, which happens in cows under heat stress. There are two potential mechanisms for MFD during heat stress:

- Rumen fatty acid biohydrogenation – inhibiting de novo milk fat synthesis.
- Rumen lipopolysaccharide – limiting substrate supply and de novo milk fat synthesis.

Biohydrogenation

Biohydrogenation of unsaturated fatty acids in the rumen produces saturated fatty acids.

According to the well accepted 'biohydrogenation theory', MFD results

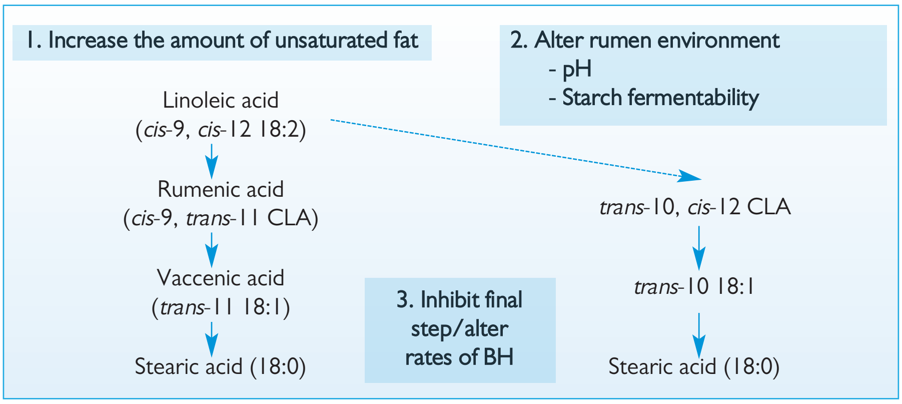

Fig. 1. Dietary components can impact the risk of milk fat depression in three ways through the rumen biohydrogenation (BH) pathway. From Lock and Bauman (2007).

from changes in rumen biohydrogenation of unsaturated fatty acids and the passage of specific intermediates of biohydrogenation out of the rumen (ie. trans-10, cis-12 CLA).

These biohydrogenation intermediates subsequently interfere with the expression of genes involved in fat synthesis thereby reducing milk fat synthesis in the mammary gland. Following the biohydrogenation theory, MFD requires:

- Substrates for the formation of potent inhibitors of milk fat synthesis (linoleic acid and other polyunsaturated fatty acids).
- Altered rumen environment impacting biohydrogenation (high concentrate-low fibre diet which is low in effective fibre).
- Altered rate of biohydrogenation caused by certain feedstuffs that increase the passage of biohydrogenation intermediates out of the rumen.

Furthermore, the increased rate of feedstuffs outflow from the rumen may increase the likelihood of biohydrogenation intermediates passing through the rumen. The theory thus identifies how certain feedstuffs can represent risk factors for MFD (Fig. 1).

Feeding supplemental fat is one of the feeding strategies suggested to maintain energy intake during heat stress. However, it is important that the source of fat is rumen inert. Otherwise, low rumen pH, a predisposed condition of heat stressed

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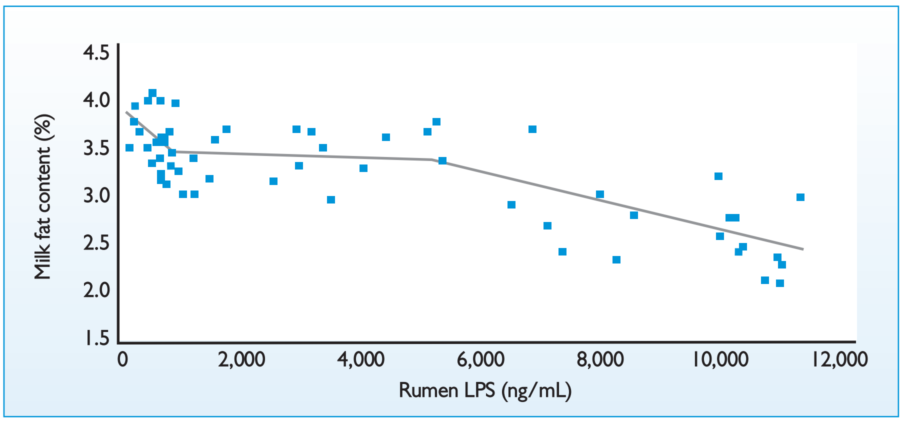
Fig. 2. Correlation between rumen lipopolysaccharide (LPS) and milk fat content. From Zebeli and Ametaj (2009).



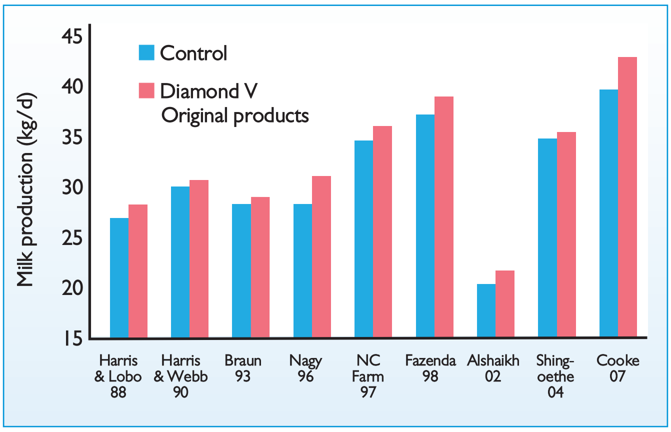


Fig. 3. Effect on milk production during heat stress.

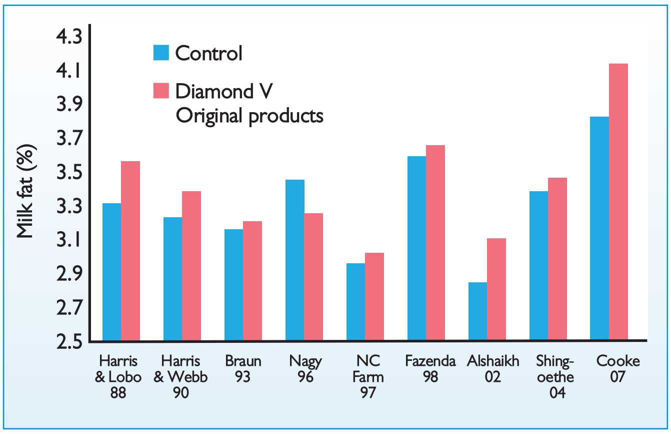


Fig. 4. Effect on milk fat content during heat stress.

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cows, could generate more intermediates of biohydrogenation and increase the risk of MFD.

Rumen lipopolysaccharide

The other potential mechanism for MFD during heat stress involves the concentration of rumen lipopolysaccharide (LPS), a component of the outer membrane of Gram negative bacteria that is released when these bacteria die. Research shows that when rumen pH decreases, the rumen concentration of LPS increases and milk fat concentration decreases.

Zebeli and Ametaj (2009) showed markedly greater concentrations of rumen LPS with increasing dietary grain level and found a strong negative relationship between rumen LPS and milk fat content (Fig. 2). This correlation could be due to the ability of LPS to induce insulin production in the pancreas.

Increased circulating insulin and increased insulin sensitivity of heat stressed cows could reduce body fat mobilisation. This condition could occur even though heat stressed cows are under negative energy balance due to reduced feed intake and increased maintenance demands. The lack of plasma NEFA, potentially an important precursor for milk fat synthesis under heat stress, may also contribute to MFD.

Other reported negative LPS effects on fatty acid production include:

- Decrease in activity of lipoprotein lipase.
- Decrease in expression of lipoprotein lipase and fatty acid transport protein 1.
- Suppressive effect on enzymes related to de novo fatty acid synthesis in the mammary tissue.

Maintaining rumen health

Research to date helps explain how MFD during heat stress relates to depressed rumen health. Given this relationship, optimising rumen function could help maintain milk fat content and production efficiency of dairy cows under heat stress.

Studies with Diamond V Original products show they can minimise the negative effect of heat stress by optimising rumen function. The results of milk production from nine studies in four countries (Israel, Portugal, Saudi Arabia, and USA) during the summer months show a consistent increase in milk production when cows were supplemented with Diamond V Original products (Fig. 3).

In these same studies, milk fat content was higher or trended higher in eight out of nine studies (Fig. 4), indicating no dilution of milk fat occurred due to increased milk volume.

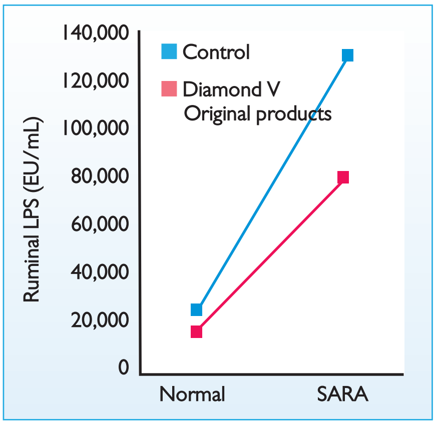
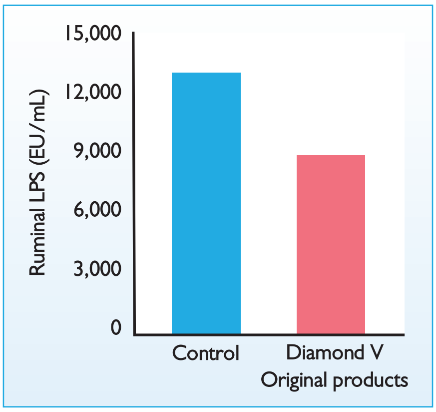


Fig. 5. Effect of on rumen lipopolysaccharide (LPS) concentration (Endotoxin unit/ml) under sub-acute ruminal acidosis (SARA) challenge. From Li et al. (2012).

Fig. 6. Effect on rumen lipopolysaccharide (LPS) concentration (Endotoxin unit/ml) under heat stress challenge.



The products optimised rumen function, helping to achieve improved production benefits under conditions of heat stress.

Subacute ruminal acidosis (SARA) is a risk for cows under heat stress. SARA induction studies have been used to produce rumen conditions similar to what heat stressed cows experience.

In such a study, Li et al. (2012) reported that cows supplemented with Diamond V Original product maintained lower rumen LPS (Fig. 5), which was associated with the stabilised rumen bacterial community.

Another trial performed at the Diamond V Research and Innovation Center in Cedar Rapids, Iowa during the summer months of 2013 (June to September) used eight non-lactating Jersey cows with rumen cannula.

This trial found that Diamond V Original products can maintain lower rumen LPS when cows were exposed to elevated ambient temperature and relative humidity (Fig. 6, unpublished data).

Keeping the rumen LPS low could help maintain lower circulating insulin concentrations and allow body fat mobilisation during heat stress. It could also allow a supply of NEFA as a precursor for milk fat synthesis in the mammary gland.

Low rumen LPS could also minimise the production of LPS induced mediators that inhibit the activity of key enzymes related to de novo fatty acids synthesis.

Preparing for heat stress

Heat stress causes physiological and behavioural changes in dairy cows. These changes can lead to suboptimal rumen conditions, resulting in production of fatty acid biohydrogenation intermediates and LPS that inhibit milk fat synthesis in the mammary gland. Optimising the rumen condition with Original XP or Original XPC can minimise the negative impact of heat stress on milk fat and maintain production efficiency of dairy cows. ■

References are available from iyoon@diamondv.com on request