

Endotoxins in the rumen – should we care?

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In recent years, much attention has focused on the topic of increasing endotoxin values in the rumen during rumen acidosis. High carbohydrate diets change the microflora in the rumen. Gram negative bacteria die and the amount of Gram positive bacteria increases.

This effect leads to a dysbiosis, which in turn results in ruminitis and acidosis. Ruminitis consequently leads to an increased rumen permeability, which allows endotox-

ins to enter the organism. But what does this mean for your cow?

Endotoxins in general

Endotoxins have been known since the early 1960s because of their pyrogenic (fever inducing) effect. In general, endotoxins are parts of the cell wall of all Gram negative bacteria (Fig. 1). They are of great interest because of their effect on the immune system. Endotoxins are also called lipopolysaccharides (LPS, see Fig. 2) because of their structure, consisting of a lipid (lipid A, lowest variability) and a polysaccharide (high variability of chain length).

Fig. 1. Comparison of Gram positive and Gram negative bacterial cell wall. Location of lipopolysaccharide in the cell wall is circled.

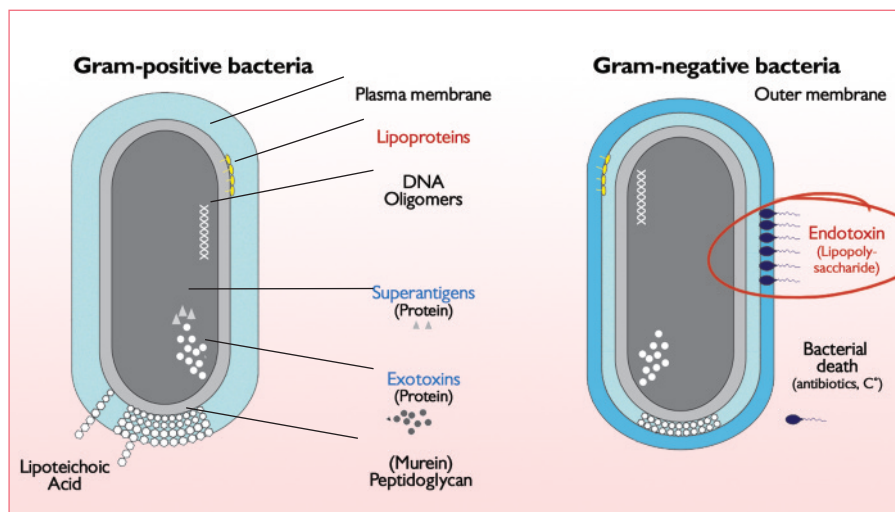
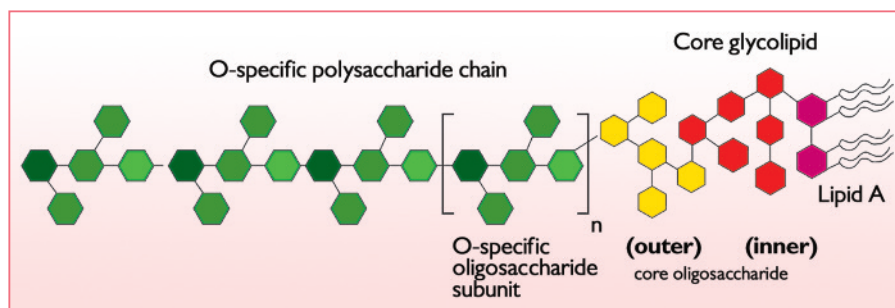


Fig. 2. Structure of LPS (endotoxin).



The structure of the LPS is decisive in the uptake and detoxification of the molecule. Endotoxins are released by bacteria during death or overwhelming proliferation.

The administration of special kinds of antibiotics (for example beta-lactam) with bactericidal activity may increase the liberation of endotoxins. This fact should be taken into consideration when treating a cow with antibiotics.

Endotoxins in ruminant feed

Wieding (2001) recommended that the endotoxin load of silage should be lower than 15µg endotoxin/g feed. Compared to Krüger (2000) (700µg/g feed) this is quite a low maximum contamination level. Different feed matrices can contain varying endotoxin contamination levels. For example, maize silage can comprise 12-46µg/g and rapeseed expeller 39-48µg/g, while wheat can contain up to 90µg/g feed.

Of special interest are feeding materials with high fat content. Enteral available lipids can lead to an increase in endotoxins in the serum and milk. Because of high lipid levels, the amount of free fatty acids increases.

These fatty acids have a toxic effect on other micro-organisms, especially on protozoan and Gram negative bacteria, with the latter leading to an increase in endotoxins.

Effects in ruminants

Ruminants are constantly in contact with endotoxins via feed, air and the environment. In healthy animals, only small quantities are adsorbed through the intestine into the blood. They are then transported and detoxified in the liver.

Due to their structure, endotoxins can also be stored in the fat tissue. In the case of an energy deficiency or feed imbalances (low fibre, high concentrate), the gut wall becomes more permeable and therefore, more endotoxins can enter the bloodstream.

In addition, if the animal lacks sufficient energy, fat is degraded and even more endotoxins enter the organism. As a result,

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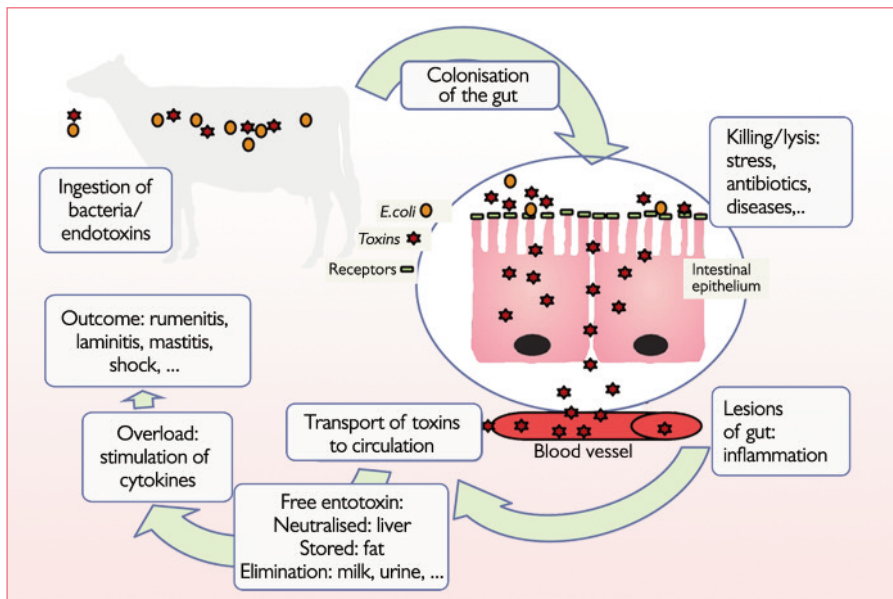


Fig. 3. Pathway of endotoxins: After ingestion by the cow, endotoxins enter the gut and reach the blood circulation. This results in a series of diseases.

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this may trigger different diseases such as mastitis, endometritis, laminitis, dermatitis digitalis, endotoxic shock and others.

Rumen simulation model

The rumen simulation model is an important in vitro model to test the influence of feed additives on rumen physiology. This model, adapted at the Biomin Research Center, uses 'natural' rumen fluid to determine the influence of additives on the rumen pH, bacterial number and concentration of fatty acids, which are important parameters of rumen physiology.

In addition, the influence on endotoxin concentration in the rumen can be tested.

Preliminary results with the rumen simulations model confirmed that antibiotics have a negative effect on endotoxin production in the rumen.

After a two-week long incubation, the endotoxin concentration of the reactors treated with antibiotics increased significantly compared to an untreated reactor (Fig. 4).

This shows the need for alternative treatment strategies to positively influence the rumen physiology and control the endotoxin load in the rumen.

Conclusion

The damages caused by endotoxins are fact and no fiction. They are ubiquitous in the

environment and are permanently released. A healthy cow can cope with the normal load of endotoxins by detoxification in the liver.

In the case of an increase in endotoxins, or a liver failure, endotoxins overwhelm the organism of the cow. Inflammation cascades are triggered and result in different diseases which, in the worst cases, may lead to shock and death.

As endotoxins are ever present in the ruminants' environment, control strategies to prevent endotoxin-related diseases among cows are essential, and recommended. ■

References are available from the authors on request

Fig. 4. Comparison of mean endotoxin values of two reactors from the rumen simulation model. The antibiotic treated reactor (red) showed significant increased endotoxin values after long term incubation.

