Undesirable substances in ruminant nutrition – The bright and the dark side of the rumen

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According to the feed law a feed ingredient or a substance in feed is considered as undesirable if it is suited to adversely affect the health of the animals, to induce residues of human health concern in edible tissues or to have a negative impact on the environment.

Some of these substances are listed with upper limits which must not be exceeded (e.g. aflatoxin B1, polychlorinated dibenzo-para-dioxins [PCDDs] and polychlorinated dibenzofurans [PCDFs]), while for others guiding values exist for a harmonized management (e.g. deoxynivalenol [DON], ochratoxin A [OTA], fumonisins). In addition, some substances are not regulated at all but might become undesirable depending on a certain feeding situation (e.g. nitrate, tryptophan, sulfur).

Some substances might become undesirable not before being altered in their structure due to chemical modification mediated by endogenous enzymes especially of the intestinal mucosa and the liver or by the action of the microbiota of the digestive tract. The latter occurs post-gastric and mostly post-small intestinally in monogastric animals while the rumen serves as a pre-gastric and thus pre-systemic metabolization chamber. Consequently, substances which become more toxic by microbial metabolization might induce more serious adverse effects in ruminants compared to monogastrics while the opposite might be expected from potentially toxic feed substances which underlie microbial inactivation. The order of magnitude of both aspects depends in many cases on an adequate adaptation of the microbes to the respective substrates. In this regard, the absence or presence of metabolizing capacity for certain substances was shown to be dependent on the geographical region where ruminants were kept and on ruminant species.

Examples for the dark side: Nitrate might become critical when excessive amounts are ingested whereby the toxic nitrite is accumulated due to a higher capacity of the nitrate reductase compared to the activity of the nitrite reductase which converts nitrite to ammonia. Feedstuffs containing higher amounts of sulfur might induce excessive ruminal formation of hydrogen sulfide which is inhaled via ructus. Subsequently it might affect the lungs and reach the brain where it causes lesions known as polioencephalomalacia. The tryptophan degradation substance 3-methylindole was shown as the causative agent of the acute bovine pulmonary edema and emphysema which might occur after a sudden change from a poor quality to a green and rapidly growing pasture.

Examples for the bright side: The mycotoxin DON bears an epoxid group which is responsible for its toxic effects. This epoxid group is efficiently cleaved by ruminal microbes whereby the generated metabolite de-epoxy-DON lacks the toxicity of the mother compound. OTA as an example for another mycotoxin which is also cleaved by rumen microbes and the resulting OT-alpha can be considered as the non-toxic metabolite of OTA. Oxalates might be degraded to carbon dioxide and formic acid which consequently prevents the nephrotoxic effects of the oxalates.

This strong differentiation between a dark and a bright side of the rumen does certainly not apply for all situations as discussed above and rather neutral or nil effects also occur. For example, PCDDs and PCDFs are not significantly modified by rumen bacteria and although aflatoxin B1 is metabolized to aflatoxicol this reaction does not eliminate the toxicity of the initial toxin. Fumonisins as another group of mycotoxins appear not to be metabolized at all in the rumen.

In conclusion, the rumen bears the potential for inactivation of a number of substances while others might become undesirable by ruminal action. Adaptation to the respective substrates and the surrounding milieu which can largely be modified by feeding might contribute to the overall effects.