From science to practice 1

Mycotoxins in brief

This new series will give an over view on the effects of different mycotoxins in ruminants, helping to identify possible risks – from science to practice.

The omnipresence of mycotoxins is an undeniable fact. Fusarium, Aspergillus, and Penicillium sp. are the most abundant moulds that produce these secondary metabolites and contaminate animal feeds through fungal growth prior to and during harvest, or through (improper) storage.

Types of mycotoxins such as aflatoxins, trichothecenes, fumonisins, zearalenone, ochratoxins and ergot alkaloids seem to be the most relevant in regards to feed contamination and economic impact in the animal industry.

Mycotoxins are invisible, odourless and tasteless. If certain characteristics appear like low bushel weight, discoloured kernels, musty odour or great amount of broken kernels one might suspect mould contamination. However, it is important to keep in mind that the presence or absence of fungi does not necessarily correspond to the occurrence or lack of mycotoxins.

These fungal toxins may cause millions of dollars worth of damage but these impacts are not easy to diagnose. The economic losses, more often than not, result from mycotoxins exacerbating common dairy problems rather than causing overt clinical symptoms. Lower milk production, higher SCC (somatic cell count) and incidence of mastitis, more metabolic problems, reproductive problems (retained placentas, irregular heats and abortions) and decreased feed intake are common consequences.

Moreover, exposure of farm animals can result in undesirable residues in animal derived food products. Many questions are often posed in the case of mycotoxicoses. Analysis reports indicate low mycotoxin concentrations and still, animals show severe symptoms. Many times mycotoxins interact amongst each other and their individual effects are therefore increased.

Other interacting factors like environmental condition and age of the animal, may enhance the susceptibility of animals to mycotoxins. ■

From science to practice 2

Aflatoxins in brief

Aflatoxins in cattle feeds and carry-over to dairy milk

Did you know that up to 6% of the consumed aflatoxin present in feedstuffs is carried-over as aflatoxin M1 into the milk of dairy cows which can cause significant economic impacts through loss of milk sales?

Among mycotoxins, aflatoxins are the most intensively studied group of toxins. Aflatoxins were already identified more than 50 years ago and comprise of four known toxins (AFB1, AFB2, AFG1 and AFG2) mainly produced by Aspergillus parasiticus and A. flavus strains.

In consideration of the carcinogenic properties of aflatoxins (especially AFB1), legislation exists on a worldwide bases to limit the exposure of theses toxins.

Early indication of aflatoxin contamination include reduced feed intake followed by weight loss and a slower growth rate as well as reduced milk production.

Even low levels may lead to liver damage with increased susceptibility to diseases and reduced resistance to environmental stress factors.

Calves and high yielding cows are considered to be more sensitive to aflatoxins. It is reported that aflatoxins are partly degraded by the ruminal flora into aflatoxicol which leads, in a minor extent, to reduction of biological potency.

The remaining part is converted to aflatoxin M1, which is excreted to a certain amount into the milk. This carry-over rate can vary depending on different factors including, among others, individual animal health status, biotransformation capacity and, finally, also by the actual milk production.

It is stated that milk yield is the major aspect affecting the total excretion of AFM1.

Aflatoxin M1 appears in the milk within hours after consumption and returns to baseline levels within two or three days after removal of con-taminated feed from the diet.

Monitoring aflatoxin B1 in feedstuffs and aflatoxin M1 is an important part in a correct mycotoxin risk management. ■

From science to practice 3

Do trichothecenes increase the incidences of mastitis and laminitis?

Since their first discovery, more than 200 different trichothecenes (type A e.g., T-2 toxin and type B e.g., deoxynivalenol) have been documented. Even though dairy cows have some capacity to protect themselves against the harmful effects of mycotoxins, these substance are still a menace to ruminants. As feed passes more quickly through the rumen of modern dairy cows due to higher feed intake, rumen microbes have less time to detoxify mycotoxins.

The combined factors of intensive farming of dairy cattle, incompetent rumen microflora action, nutritional imbalance and mycotoxins in the feed are key factors that allow mycotoxins to escape detoxification and be absorbed by the intestine.

The occurrence of mycotoxins in feed is very often connected to increased incidences of metabolic disorders such as ketosis, retained placentas, displaced abomasum, mastitis, metritis, lameness, elevated somatic cell counts and consequently, slight decreases in milk production. Subclinical mycotoxicoses decrease profitability by lowering milk production and quality while increasing expenses from inappropriate veterinary therapies.

The presence of T-2 toxin in cattle feed can result in gastroenteritis and intestinal haemorrhages. T-2 toxin has also been related to feed refusal, gastrointestinal lesions, bloody diarrhoea, low feed consumption and decreased milk production. The impact of deoxynivalenol in dairy cattle is controversial, but data have shown an association between deoxynivalenol intake and poor performance. Besides the effects mentioned above, trichothe-cenes are also known to trigger metabolic disorders.

Exposure to deoxynivalenol can increase inflammatory reactions in the form of mastitis and laminitis. In addition, it was observed that deoxynivalenol may also reduce phagocytic and neutrophil activity, leading to severe symptoms such as the occurrence of mastitis and lameness.

Accurate feeding of dairy cows in combination with continuous mycotoxin risk management is the key to managing the optimal performance of the livestock business. ■

From science to practice 4

Are ruminants affected by ochratoxin A?

Ochratoxin A is a nephrotoxic, hepatotoxic and teratogenic mycotoxin formed by Aspergillus and Penicillium sp, which have been shown to occur in various grains and other products throughout the world.

The intake of ochratoxin A contaminated feed affects animal health and performance and can result in the presence of these toxins in animal products.

Although it has been reported that ochratoxin A affect cattle, it is degraded in the rumen which explains the higher tolerance of ochratoxin exposure via contaminated feeds.

Ruminal microbes, especially protozoa, are active in converting ochratoxin A to the less toxic compound ochratoxin α.

The capacity of cattle to degrade ochratoxin A has been reported in several in vitro and in vivo studies. Nevertheless, ochratoxin A is not completely degraded by an active rumen population and it is estimated that a maximum rumen bypass of ochratoxin A is 10%.

The transfer of ochratoxin A into dairy milk is limited but it can occur under certain circumstances.

The combined factors like high concentrate as well as feeding levels are factors observed to promote the likelihood of systemic occurrence due to factors like changes in the rumen microflora.

In the case of pre-ruminants, experimental examinations of 30 day old calves which received 0.1-0.5mg ochratoxin A/kg live mass daily over four weeks showed polyuria, depression, decreased weight gain, low specific gravity of urine and dehydration.

At necropsy, greyish coloured kidneys and mild enteritis were observed. Ochratoxin A was also found combined with citrinin, a metabolic product produced by the same fungi.

Co-occurrence of these two toxins may cause haemorrhagic syndrome in cattle.

Cases of ochratoxicosis have been seldom reported in the field; however, the chronic intake of contaminated feeds should be avoided as long term exposure of ochratoxin A could represent a potential risk arising from the adverse effects of these toxins on cattle. ■

From science to practice 5

Are ergot alkaloids and endophyte toxins responsible for vasoconstriction?

Did you know that estimated losses due to fescue toxicosis are likely to exceed $1 billion annually in the US ruminant industry alone?

Ergotism, also named 'St Anthony's Fire', is one of the oldest known mycotoxicoses. The first documented epidemic occurred in the Middle Ages when thousands of people died. The term ergot alkaloid refers to a diverse group of about 40 different toxins which are formed by Claviceps spp. on grains (rye, triticale, wheat, barley) and by fungal endophytes such as Neotyphodium spp. in grasses, particularly tall fescue and perennial ryegrass.

Animals can be exposed to complex mixtures of ergot alkaloids. The kinds of alkaloids present and their levels can vary widely, depending on the fungal strain, the host plant and environmental conditions.

The chemically diverse group of ergot alkaloids has been shown in vasoconstriction symptoms, and is responsible for heat intolerance as well as necrosis of ears, tails and often hooves.

Other symptoms, such as gangrenous changes, neurotoxic signs including convulsions, abortions and death, reduced prolactin secretion and consequently agalactia have been reported as major adverse effects in animals.

Endophyte toxins in grasses include ergot alkaloids in tall fescue and tremorgens (for example lolitrem B) in perennial ryegrass. Perennial ryegrass endophyte (Neotyphodium lolii) alkaloids are frequently present in pastures containing the neurotoxin lolitrem B, together with the vasoconstrictor ergovaline, at levels associated with ryegrass staggers, impaired heat stress recognition, ill-thrift and lowered milk production.

As ergot alkaloids and endophyte toxins can cause significant losses in the ruminant industry, accurate feeding of dairy cows in combination with continuous mycotoxin risk management are the keys to maintaining optimal performance. ■

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From science to practice 6

Fumonisins: serious destroyers of ruminant offspring?

Fumonisins are produced by Fusarium verticillioides (formerly = F. moniliforme), F. proliferatum, and other Fusarium species. While these mycotoxins are found in other commodities, animal and human health problems related to these mycotoxins are almost exclusively associated with the consumption of contaminated maize or maize products.

Cattle, with the exception of calves, lamb and goat kids are considerably less sensitive to fumonisins than horses, pigs, rabbits, or laboratory rodents.

Maize screenings are part of a basal diet for ruminants such as cattle in the USA, making fumonisin toxicity and excretion in milk a concern in ruminants.

Fumonisins are hepatoxic and nephrotoxic to calves. Major clinical signs of fumonisin poisoning in calves are lethargy and decreased appetite accompanied by serum biochemical and histologic evidence of hepatic damage.

The accumulation of sphinganine (Sa) and sphingosine (So) in the serum and urine is a useful biomarker for the exposure of fumonisins. These free sphingoid bases are toxic to most cells by affecting cell proliferation and inducing apoptosis or necrotic cell death and are associated with hepato- and nephrotoxic effects.

Increased concentrations of Sa and So were found in the liver, kidney, lung, heart and skeletal muscle of calves fed diets contaminated with fumonisins.

Lambs and goat kids exposed to fumonisins expressed similar biochemical indices and histologic findings than calves and suffered from renal and mild hepatic toxicity. Similar to calves, goats also showed increased Sa and So concentrations in the liver, kidney and heart, which is clear evidence that all young ruminants with non-fully developed rumens and weak immune systems can be heavily affected by this group of mycotoxins. Only accurate feeding in combination with continuous and effective mycotoxin risk management offer the keys to maintaining optimal health and improving the future performance of young ruminants affected by fumonisins. ■