Mycotoxins in dairy
Facts, figures & solutions

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Dairy cows are known to have some capacity to protect themselves against the harmful effects of mycotoxins. This capacity depends on the cow’s ability to efficiently deactivate mycotoxins in the rumen, which in turn depends on having feedstuffs retained in this rumen “compartment” long enough to allow rumen microorganisms to work properly.
With large amounts of feed comes the risk of increased mycotoxin exposure, higher passage rates and less time available for proper feed digestion. As animals are fed increasing quantities of feed to increase milk yields, it becomes more difficult to guarantee that mycotoxins can be effectively deactivated in the rumen. Complete mycotoxin degradation in the rumen is therefore not possible.

Various mycotoxins are able to modify the rumen microflora as they exert antimicrobial, antiprotzoal and antifungal activity. In practical terms, this means that mycotoxins escape detoxification and are absorbed by the intestine. In other words, mycotoxins disrupt the rumen function before impacting the animal itself.

Drastic changes in feed composition and a high percentage of protein-rich concentrates in the daily diet also impair the cleavage capacity of rumen microorganisms. Reduced ruminal motility, decreased dry matter intake, acid detergent fiber and starch digestion are some negative impacts reported due to the ingestion of mycotoxin-contaminated feed.
Because fertility and milk yield are parameters that are closely related, all factors disrupting fertility have a negative economic impact on herds. Zearalenone (ZEN), an estrogenic metabolite, shows a structural similarity to the female hormone estradiol, and is able to activate specific estrogen receptors. Thus, ZEN causes abnormal estrous cycles which ultimately impair fertility.

Reduced fertility in dairy cattle has also been reported as a result of ergot alkaloids and aflatoxins.

### Case study 2

**Dairy farm Europe, 110 Holstein dairy cows**

**Background**

Feed intake of dairy cows decreased overnight (55% lower) accompanied by lowered milk production, diarrhea and reproduction failure. Mycotoxins were detected in the corn silage (600 ppb DON, 50 ppb ZEN; based on fresh matter).

**Feedback**

The farm started using Mycofix® at 30 g/cow/day. After just 4 days, feed intake was completely re-established. Mycofix® was used for one month (2 weeks at 30 g/cow/day; thereafter at 20 g/cow/day) and parameters such as feed intake and milk production were back to normal. After a month the farmer decided to stop using Mycofix®. Within 2 days, the same problems resurfaced with a rapid decrease of feed intake. Milk characteristics during the affected period were as follows: SCC: 400,000; Fat: 3.95%; Protein: 3.35%; Lactose: 5.00%; Urea: 24 mg/dl.

Mycofix® was again added to the ration. After a few days, feed intake and milk production were again stabilized. Milk characteristics also returned to normal as follows: SCC: 160,000; Fat: 3.75%; Protein: 3.30%; Lactose: 5.00%; Urea: 24.5 mg/dl.
• Toxic residues in milk

In the case of aflatoxins, the most worrying effect is their carry-over ranging from 1.8 to 6.2% into milk as aflatoxin M₁ (AfM₁). Aflatoxins are considered carcinogenic by the Institute of International Agency for Research on Cancer (IARC).

• Mycotoxins increase the incidence of metabolic problems in dairy animals

Figure 1 provides an overview of the effects of mycotoxins in dairy cattle.

The most common and difficult challenges to identify occur when rations contain low levels of mycotoxins. Subclinical mycotoxicoses decrease profitability by lowering milk production and quality, and increasing veterinary expenses, sometimes with inappropriate therapies. The presence of mycotoxins in feed is very often connected with increased incidences of metabolic disorders such as ketosis, retained placenta, displaced abomasum, mastitis, metritis, lameness, elevated somatic cell count and consequently, decreased milk production (refer to page 6 for case studies 4 and 5).

Multi-mycotoxin strategies

Avoiding mycotoxin formation must begin on the field, should continue in the silage production process and end with correct management of the open silo and feedstuffs.

Most grains and feedstuffs are afflicted by a wide variety of mycotoxin types. The Mycofix® product line of Biomin combines three modes of action—adsorption, biological degradation of non-adsorbable mycotoxins, and protection of the liver and immune system. Accurate feeding of dairy cows in combination with continuous mycotoxin risk management is the key to managing the optimal performance of the livestock business.
Avoiding mycotoxin formation

1. Begin in the field
2. Continue in silage production
3. End with correct management of the open silo and feedstuffs.

Case study 4

- Dairy farm in the Americas, 1,100 Holstein dairy cows

Background
The farm had difficulties with elevated somatic cell counts (SCC) and mycotoxins were suspected to be the causative agent. Mycofix® (30 g/cow/day) was fed and data recorded for one year.

Feedback
Comparing average SCC for the first 2 months of the trial period (most reflective of pre-treatment) with average SCC for the last 2 months, a reduction of approximately 40% can be observed.
Along with the reduction in SCC, the farm found that they had fewer reasons to cull cows from the herd: better production, less mastitis and less breeding issues. This allowed the herd to retain older cows with greater production, and sell younger heifers as an additional source of income.

Figure 3. Somatic cell count reduction over a year with Mycofix®.

Case study 5

- Dairy farm in Asia, 600 Holstein dairy cows

Background
Mycotoxins (mainly ZEN, 200 ppb and DON, 1,200 ppb) were detected in the total mixed ration (TMR).

Feedback
Data were collected over a period of 3 months and compared 2 different treatments (control group without Mycofix® versus trial group with Mycofix® at 2 kg/tonne feed).
After administering Mycofix® in TMR, the health status of animals in the trial group improved with fewer disease incidences.

Figure 4. Disease incidences in the control and trial groups.

Data collected worldwide. Special thanks to Doug Taylor, Bryan Miller, Luis Cardo and Shu Guan.

References are available on request.