



**Mycotoxins and Dairy Cattle**  
*A Review for Dairy Producers*

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## INTRODUCTION

Mycotoxins are ubiquitous and produced by several fungi, particularly by many species of *Aspergillus*, *Fusarium*, *Penicillium*, *Claviceps*, and *Alternaria* etc. They are secondary metabolites from fungi with unclear functions. Over 400 known mycotoxins have been identified today with a potential of 30,000 different metabolites. Among which aflatoxin, fumonisin, ochratoxin, T-2 toxin, vomitoxin, and zearalenone have the most attention by industry and the most research by academy.

In the early 1950's, death losses of cattle consuming moldy corn was reported in the United States (Sippel et al., 1953). Toxic substances from *Aspergillus* and *Penicillium* fungi were identified to cause the problem in later years (Burnside et al., 1957). The toxin responsible for the mortality was first purified by British scientists (Allcroft et al., 1961) from peanut meal originating in Brazil and was then named aflatoxin. Since then, many acute and chronic toxicity studies of aflatoxin and other mycotoxins have been demonstrated and reported.

With the ability of detoxification by rumen microorganisms, mycotoxins are considered less toxic in ruminants as compared to simple stomach animals. Among all of the mycotoxins, aflatoxin is the most problematic in dairy due to its derivative aflatoxin M<sub>1</sub> (AFM<sub>1</sub>), present in milk, and its potential health hazard for human consumption. Aflatoxin can be present in several forms in feedstuffs, aflatoxin B<sub>1</sub>, B<sub>2</sub>, G<sub>1</sub>, and G<sub>2</sub>, with aflatoxin B<sub>1</sub> (AFB<sub>1</sub>) being the most biologically active and toxic to animals (and humans).

## MYCOTOXINS AND MILK

### Aflatoxin B<sub>1</sub> and Aflatoxin M<sub>1</sub>

Dairy cattle, in general, can tolerate a greater mycotoxin challenge. It has been hypothesized that the microbial population in the rumen is able to metabolize most mycotoxins. However, some of the toxic metabolites can be excreted in milk and cause public health concern. Among all known mycotoxins present in feed, aflatoxin (AFB<sub>1</sub>) has the most significant impact to the dairy industry. Because little of the AFB<sub>1</sub> consumed is degraded by rumen and the resulting metabolite (aflatoxicol) is as toxic as AFB<sub>1</sub>, it was suggested by Jouany and Diaz (2005) that ruminants have little protection against this toxin.

AFB<sub>1</sub> and AFM<sub>1</sub> (metabolite) are found in feeds and milk, respectively. Dairy cattle will produce milk contaminated with AFM<sub>1</sub> after consuming feeds contaminated with AFB<sub>1</sub>.

The AFB<sub>1</sub> is rapidly absorbed in the digestive tract and primarily metabolized by liver enzymes, converting it to AFM<sub>1</sub>, which is then excreted in milk and urine. AFM<sub>1</sub> is less toxic than AFB<sub>1</sub>; however, it has been demonstrated to be a carcinogen in rainbow trout (Sinnhuber et al., 1970) and causes morphological changes in rat liver (Pong and Wogan., 1971). The carcinogenic and highly toxic effects of aflatoxin and its metabolites has resulted in aflatoxin being highly regulated by most countries in the world (Table 1). Once it exceeds the regulatory limits, the AFM<sub>1</sub> contaminated milk, by law, has to be discarded to prevent it from getting back into the food chain. AFM<sub>1</sub> contamination in milk occurs often (not always above regulatory limits) because AFB<sub>1</sub> often occurs naturally in grains, by-products, and roughage.

Jouany and Diaz (2005) reported that the average transfer of AFB<sub>1</sub> in diet to AFM<sub>1</sub> in milk is 1.7%. With this figure, the authors calculated that only 30 ppb of AFB<sub>1</sub> in feed will result in 0.5 ppb AFM<sub>1</sub> (above regulatory limits in USA). Using the same calculations, in the EU, only 3 ppb dietary AFB<sub>1</sub> would result in milk being over the regulatory limits (0.05 ppb AFM<sub>1</sub>). It is not practical to completely eliminate the use of AFB<sub>1</sub> contaminated feed ingredients; however, it is possible to control the toxin, preventing or at least reducing the concentration of AFM<sub>1</sub> in milk.

Ozonization and ammoniation have been shown as promising treatments for AFB<sub>1</sub> contaminated corn and cottonseed meal because it can be used in large batches of product (CAST, 2003). However, both methods are time consuming and economically impractical. Besides, ammonia treated grains are not currently allowed for interstate shipments in the U.S. Therefore, use of adsorbent materials in feeds to prevent toxin absorption by dairy cattle becomes a more feasible strategy. A comprehensive review of different types of mycotoxin adsorbents is reported by Huwig et al. (2001). Dairy producers are encouraged to read the paper for better understanding of mycotoxin binders including natural vs. synthetic adsorbents and biological methods, such as enzymes and yeasts.

TABLE 1: Global regulation of aflatoxin M<sub>1</sub> (AFM<sub>1</sub>) in milk.

Countries	AFM <sub>1</sub> Concentration in Milk
EU Countries	Less than 0.05 ppb
China, Japan, Mexico, Thailand, USA etc.	Less than 0.5 ppb
Nigeria	Less than 1.0 ppb

To control the amount of AFB<sub>1</sub> present in animal feeds and human foods, the U.S. Food and Drug Administration (FDA) has established ‘action levels’ for aflatoxin in feed ingredients (Table 2). The action level is the maximum amount of AFB<sub>1</sub> that can be present in animal feedstuffs to avoid toxins in meat, egg, and milk products intended for human consumption. The maximum allowance of aflatoxin in feed ingredients used in dairy feeds is 20 ppb, which is the same for young pigs and chicks. Lactating cows are as sensitive to aflatoxin as young animals; not for the toxicity of AFB<sub>1</sub> to the cow but because of the resulting AFM<sub>1</sub> in the milk.

TABLE 2: U.S. FDA action levels for aflatoxin in animal feeds.

Commodity	Action Levels, ppb
Corn, peanut meal, cottonseed meal, and other animal feeds ingredients intended for dairy animals, or when the intended use is not known	20
Corn, peanut meal, and other animal feeds and feed ingredients but excluding cottonseed meal, intended for immature animals	20
Corn and peanut meal intended for breeding beef cattle, breeding swine, or mature poultry	100
Corn and peanut meal intended for finishing swine of 100 pounds or greater	200
Corn and peanut meal intended for finishing (i.e. feedlot) beef cattle	300
Cottonseed meal intended for beef cattle, swine, or poultry (regardless of age or breeding status)	300

The appearance of AFM<sub>1</sub> levels, greater than 0.5 ppb, in milk can be found in as quickly as 4 hours after placing 13 mg AFB<sub>1</sub> directly into the rumen of a cow (Applebaum et al., 1982; Figure 1). Therefore, speed or rate of AFB<sub>1</sub> binding is a critically important attribute for dairy producers to consider when selecting a mycotoxin binder. A natural hydrated sodium calcium aluminosilicate (HSCAS) is superior to competitive products, such as enzymes and yeast cell wall, because of its binding efficiency speed.

Once AFB<sub>1</sub> is absorbed into the cow’s body, the clearance of AFM<sub>1</sub> in milk may take 5 to 7 days depending on the amount and duration of the AFB<sub>1</sub> consumption (Whitlow and Hagler, 2005). Diaz et al. (2004) conducted a trial and fed aflatoxin to lactating cattle with or without the addition of clay products (HSCAS) in feed (Figure 2). A surge of AFM<sub>1</sub> in milk was observed after AFB<sub>1</sub> added to feed; it declined gradually after AFB<sub>1</sub> removed from feed. Without a toxin absorbent in feed, the concentration of AFM<sub>1</sub> in milk was higher than the current U.S. regulation (0.5 ppb). On the contrary, with the addition of 1% clay product, AFM<sub>1</sub> in all sampled milk was under the U.S. regulation, whether AFB<sub>1</sub> was present or not.

FIGURE 1: Milk aflatoxin M<sub>1</sub> concentrations after placing aflatoxin B<sub>1</sub> in rumen.

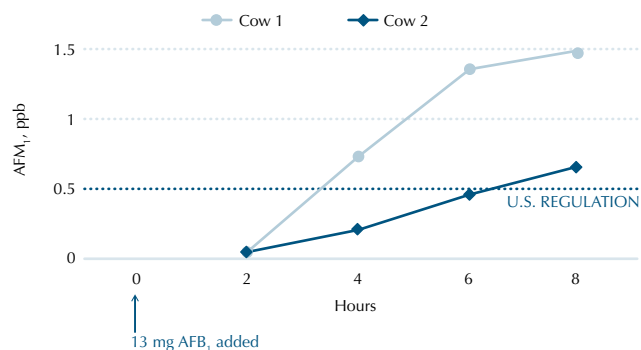
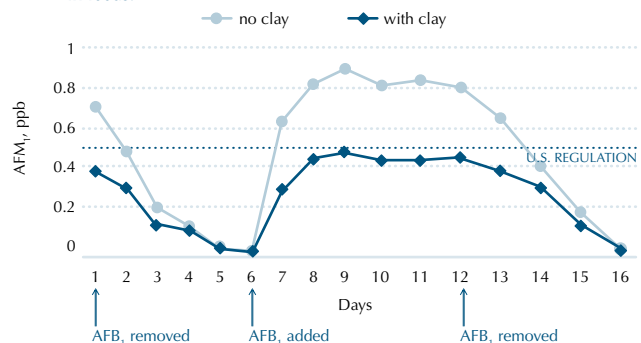


FIGURE 2: Concentration of aflatoxin M<sub>1</sub> in milk with or without 1% clay product in feeds.



Similar results were also found in dairy ewe (Battacone et al., 2003 and 2005). AFM<sub>1</sub> appeared in the sheep milk 6 hours after single AFB<sub>1</sub> dosage (2 mg), and then the concentration decreased with time. Trace amounts of AFM<sub>1</sub> was detectable until 78 hours after the dosage. In a separate study, the appearance of AFM<sub>1</sub> in sheep milk was shown to be AFB<sub>1</sub> dose dependent. When ewes were fed a diet containing 128 ppb AFB<sub>1</sub>, the level of AFM<sub>1</sub> exceeded EU regulation (0.05 ppb) as early as 12 h post-treatment. When the ewes diet contained 64 ppb or 32 ppb AFB<sub>1</sub>, the level of AFM<sub>1</sub> exceeded EU regulation at 24 hours and 144 hours post-treatment, respectively.

The stability of AFM<sub>1</sub> in milk, upon storage, has been studied; however contradicting results were reported depending on the method of processing. A report from U.S. FDA indicated that AFM<sub>1</sub> was stable for 18 days when milk was pasteurized and for 120 days when milk was frozen at -18o C (Stoloff et al., 1975). In the study, 2 ppb synthetic AFM<sub>1</sub> was added to raw milk, the raw milk was then divided into 2 halves. One half served as control and the other half pasteurized at 63o C for 30 min. Both halves were then stored at 4o C for 18 days. After 18 days, 100% of AFM<sub>1</sub> was recovered from samples with or without pasteurization (Table 3).

TABLE 3: Effect of pasteurization (63°C, 30 min) and refrigeration (4°C) on AFM<sub>1</sub> stability.

Treatments	AFM <sub>1</sub> , µg/L	
	D-1	D-18
Raw milk	1.8	1.8
Pasteurized milk	1.7	1.8

In the second study, highly AFM<sub>1</sub> contaminated milk was obtained from a university research farm and was stored at -18o C (Stoloff et al., 1975). The concentration of AFM<sub>1</sub> in the samples was measured as storage time increased (Table 4). AFM<sub>1</sub> concentration in milk was stable for up to 2 months and started to decline after 68 days of storage. However, the concentration was still above 60% of initial concentration after 120 days of storage and considered not safe for consumption.

TABLE 4: Effect of frozen storage (-18°C) on AFM<sub>1</sub> stability.

	D-1	D-12	D-53	D-68	D-100	D-120
AFM <sub>1</sub> , µg/kg	5.4	6.2	7.0	4.9	4.0	3.4

The presence of AFM<sub>1</sub> in milk is not the only problem resulting from dairy cows consuming aflatoxin contaminated feeds. Studies

have shown decreased milk production by feeding aflatoxin contaminated feed to dairy cows (Applebaum et al., 1981). In the study, 13 mg of unpurified aflatoxin was placed directly into the rumen of fistulated Holsteins for 7 days. Milk production was reduced significantly after impure AFB<sub>1</sub> was dosed directly into the rumen (Table 5). Reduced milk production was not a result of feed intake because total feed intake was not affected by AFB<sub>1</sub> presence. While feeding pure aflatoxin did not reduce milk production in the study, AFM<sub>1</sub> concentration in milk was significantly increased (P<0.05) with pure or impure aflatoxin.

TABLE 5: Milk production by feeding impure aflatoxin in cow.

Lactation (Month)	Milk Production,kg		Total Intake, (Concentrate + Hay, kg)	
	Before	After	Before	After
8	21.2	18.4	21.9	17.0
3	24.3	22.7	30.0	30.6
10	20.8	18.1	17.7	19.2
Ave	21.1 <sup>a</sup>	19.7 <sup>b</sup>	23.7	22.3

<sup>a,b</sup> - significant difference between treatments (P<0.05)

Clinical symptoms of feeding AFB<sub>1</sub> contaminated feed to calves had been well documented (Lynch et al., 1970; CAST, 2003). Calves fed contaminated diets ranging from 0.8 to 8 ppm of AFB<sub>1</sub> for 6 weeks showed no difference in weight gain and feed intake. However, serum alkaline phosphatase increased when calves were fed diets that contained more than 2 ppm AFB<sub>1</sub>. Histological examination confirmed the livers were enlarged with pale color in those treatments containing 2 ppm (0.02 mg/kg BW) and higher in feeds.

## OTHER MYCOTOXINS AND MILK

Unlike AFM<sub>1</sub>, no other mycotoxins are tightly regulated in milk. However, some toxins can be transferred into milk. In a study by Robinson et al. (1979), sows were fed 12 ppm T-2 toxin for 220 days and six days after parturition. A milk sample contained 76 ppb T-2 toxin. In a separate study, 182 mg of T-2 toxin was intubated into pregnant Holstein cow for 15 days. T-2 toxin was then found ranging from 10 to 160 ppb in milk (Robinson et al., 1979).

Unlike T-2 toxin, vomitoxin (deoxynivalenol, DON) was not detectable in milk when cows were fed 66 mg/kg for 5 days (Cote et al., 1986) or a single dose of 920 mg (Prelusky et al., 1984) in dairy cattle. However, a less toxicity metabolite,

de-epoxydeoxynivalenol (DOM-1; produced from DON by rumen microbes) was found at concentrations up to 26 ppb ( $\mu\text{g}/\text{L}$ ) in milk. Although the significance of the presence of DOM-1 to public health is unknown, dairy producers should be more proactive in controlling mycotoxins to prevent unnecessary public concerns.

Non-lactating cows fed 6.4 mg/kg DON for 6 weeks showed no noticeable symptoms or illness as compared to the control (Trenholm et al., 1985; conducted by Canadian Agriculture Research Stations). The researchers also investigated the impact of DON on milk production. Results showed that feeding medium or high levels of DON (6 or 12 ppm, respectively) did not alter dry matter intake, milk production, or protein and lactose contents in the milk. However, lower milk fat content ( $P < 0.05$ ) was found in medium DON fed cows, but not in high DON fed cows (Table 6). The authors could not explain why lower fat was observed in cows fed 6 ppm DON.

TABLE 6: Milk production and fat content after feeding different levels of DON.

Items	DON- 0 ppm	DON- 6 ppm	DON-12 ppm
DON intake, mg/d	0.6	42.7	104.2
DON intake, kg/d	16.3	15.9	16.3
Milk yield, kg/d	22.8	21.4	21.5
Milk fat, %	3.92 <sup>a</sup>	2.77 <sup>b</sup>	3.30 <sup>a</sup>
4% FCM, kg/d*	21.6	18.1	19.4

\* Fat correct milk yield.

<sup>a, b</sup> - significant difference between treatments ( $P < 0.05$ )

However, in an earlier study Noller et al. (1979) showed no influence of feeding DON on milk production and milk composition. In the study, a total of 54 lactating Holsteins were used and divided into 3 groups. Group one was fed a control diet with clean corn (20% of diet); group two fed diet with corn (20% of diet) contaminated with trichothecenes (DON 12-13 ppm and zearalenone 500 ppb in corn), and group three fed a diet with normal and contaminated corn at 50:50 mixture (10% of each in the diet). Results clearly indicated no difference of milk production or milk fat by feeding DON-ZEA contaminated feed, but the cows' weight gain was significantly reduced by consuming molded corn (Table 7).

TABLE 7: Effects of moldy corn (DON 12 ppm and ZEA 500 ppb) on lactating cows.

	Group I clean corn	Group II moldy corn 2.5 ppm DON 100 ppb ZEA	Group III 50 : 50 clean : moldy corn 1.25 ppm DON 50 ppb ZEA
DM intake, % live weight	2.90	2.79	2.85
Milk production, kg/d	22.7	23.2	22.9
Fat, %	3.81	3.82	3.81
Body weight gain, g/d	872 <sup>a</sup>	486 <sup>b</sup>	595 <sup>b</sup>

<sup>a, b</sup> - significant difference between treatments ( $P < 0.05$ )

Moderate levels of fumonisin show no influence on milk yield; however, a high dosage of fumonisin for long period has detrimental effects on milk production. Dairy cattle (Holsteins and Jerseys) fed diets containing 100 ppm fumonisin from approximately 7 days prior to freshening through 70 days postpartum demonstrated lower milk production (approximately 9 kg a day at d-56) with an average of 6 kg difference per cow per day (Diaz et al., 2000).

## TEST METHODS

There are simple, fast, semi-quantitative tests that can be used for AFM<sub>1</sub>. Kits using ELISA (enzyme-linked immunosorbent assay) technology are available commercially for farm use. AFM<sub>1</sub> can also be analyzed using HPLC (high performance liquid chromatography), which is more of a quantitative analysis, but requires very expensive equipment, a clean analytical lab and an experienced technician. On a typical farm, samples would have to be sent out to an analytical lab for quantitative AFM<sub>1</sub> analysis.

## CONCLUSION

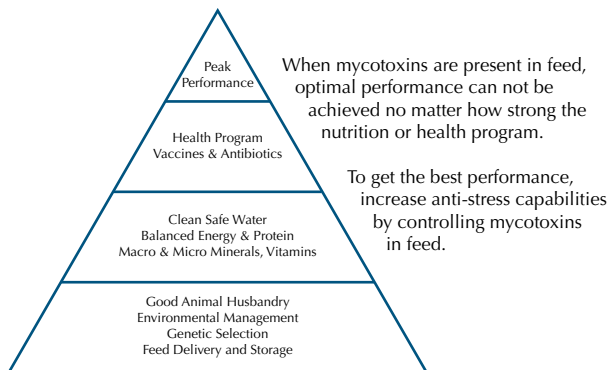
Dairy cattle are less sensitive to mycotoxins as compared to poultry, swine, equine, and aquaculture species. However, the concentration of AFM<sub>1</sub> in milk is highly dependent upon dietary aflatoxin and the threat to humans makes aflatoxin in dairy feeds a constant concern. Because of the efficiency of AFB<sub>1</sub> conversion to AFM<sub>1</sub> in milk, only 30 ppb (3 ppb in EU) of aflatoxin contaminated feed is required to increase AFM<sub>1</sub> in milk, which could result in discarded milk and lost profit. Tolerance to fumonisin, ochratoxin, vomitoxin and T-2 toxin are generally higher in dairy than simple stomach animals. Major toxins and their sensitivities in dairy cattle are summarized in the Table 8.

TABLE 8: Summary of mycotoxins sensitivities in dairy.

Mycotoxins	AFB <sub>1</sub>	DON	FUM	OTA	T-2	ZEA
Sensitivity	+++++	+++	+++	+	++	+/-
Toxin Tolerance	20~100s ppb	100s ppm	100s ppm	1000s ppm	500s ppm	Gender dependent

It is important to keep in mind that untreated mycotoxin contaminated feeds fed to dairy cattle may reduce milk production, alter milk compositions, or produce toxins in milk. No matter how strong the nutrition and health program, if dairies are not able to control mycotoxins, they will never achieve the greatest genetic potential from the animal and make the greatest profit. Therefore, controlling mycotoxins is the key in managing the peak performance of the dairy business (Figure 3).

FIGURE 3: Controlling mycotoxins is the key in managing animal peak performance.



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