



Mycotoxins and Poultry
A Review for Poultry Producers

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INTRODUCTION

Mycotoxins are produced by several fungi, particularly by many species of *Aspergillus*, *Fusarium*, and *Penicillium* etc. They are secondary metabolites from fungi with unclear functions. While over 400 mycotoxins have been identified, aflatoxin, fumonisin, vomitoxin, and zearalenone have captured the most attention. An estimated quarter of the world's food crops are contaminated by one or several mycotoxins. It is therefore important to understand how mycotoxins affect animals in order to properly control them and prevent economic loss.

Among these common mycotoxins, aflatoxin is one of the most toxic to poultry. Aflatoxin is mainly produced by *Aspergillus* species of fungi and is thought to primarily occur during storage, but can occur in the field during drought conditions

and high temperatures. Trace amounts (few ppb) of aflatoxin toxicity in ducks is commonly seen in the field, and research has shown that feeding only 1 ppm (part per million) of aflatoxin to young ducks results in mortality under a controlled environment (Muller et al., 1970). Fumonisin, zearalenone, and vomitoxin are mainly produced from *Fusarium* fungi. *Fusarium* mycotoxins are primarily produced in the field rather than during storage and can occur in a wide range of climates. Good ventilation and added preservatives, such as organic acids, are useful in controlling aflatoxin production but not *Fusarium* mycotoxins during storage or ocean transportation.

Five major classes of mycotoxins are described below in terms of their primary origins and biological effects on poultry:

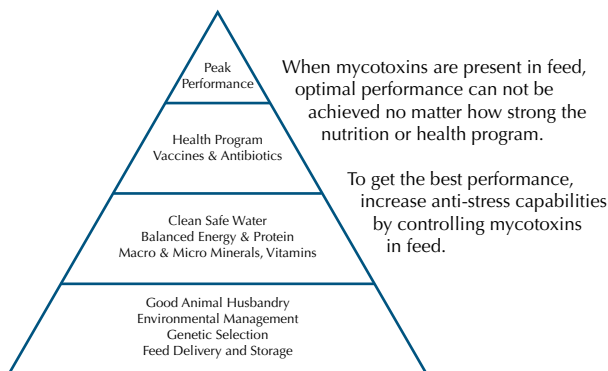
Mycotoxins	Primary Origins*	Effects on Poultry
Aflatoxin	<i>A. flavus</i> <i>A. paracitrus</i> <i>A. nomius</i> <i>A. pseudotamarii</i>	<ul style="list-style-type: none"> • Reduces feed intake and weight gain • Reduces feed efficiency • Reduces immunity • Increases mortality • Results in liver damage, such as fatty liver • Causes hemorrhaging of the kidney and intestine • Causes carcinogenesis and teratogenesis
Fumonisin	<i>F. moniliforme</i> <i>F. verticillioides</i>	<ul style="list-style-type: none"> • Little effects on growth performance of moderate levels • Reduces immunity and promotes poor growth at high levels (200~400 ppm)
Ochratoxin	<i>A. ochraceus</i> <i>P. verrucosum</i> <i>P. palitans</i>	<ul style="list-style-type: none"> • Reduces growth performance • Reduces hatchability • Kidney and liver damage • Carcinogenesis and teratogenesis
Trichothecenes Deoxynivalenol	<i>F. graminearum</i>	<ul style="list-style-type: none"> • Reduces feed intake and feed efficiency • Reduces immunity • Decreases egg production
T-2 Toxin	<i>F. sporotrichioides</i>	<ul style="list-style-type: none"> • Oral lesions and gizzard erosion • Regression of the bursa
Zearalenone	<i>F. graminearum</i>	<ul style="list-style-type: none"> • Little effects on growth and reproduction as compared to mammals

*A. – *Aspergillus*; F. – *Fusarium*; P. – *Penicillium*

Mycotoxins are stable toxic compounds; once they have been produced, it is difficult to destroy them (even with high pelleting temperatures). Several methods of controlling mycotoxins in feed and grain have been introduced, such as irradiation, ammoniation, ozone degradation, and fermentation (CAST, 2003). These methods are either expensive, can reduce nutrient quality, or can produce hazardous compounds. The most common and safest method used today is including a mycotoxin binder in the feed to adsorb the toxins, resulting in the mycotoxin passing harmlessly through the animal. Huwig et al. (2001) has published a comprehensive review of mycotoxin adsorbents/binders available in the market. It is a useful reference for nutritionists and producers.

When untreated mycotoxin contaminated feeds are fed to birds, it may reduce growth and alter the immune system. No matter how strong and how solid the nutrition and health program, if mycotoxins are not under control, producers can experience reduced profitability. Therefore, controlling mycotoxins are a key in managing birds' peak performance (Figure 1).

FIGURE 1: Controlling mycotoxins is the key in managing animal peak performance



Many papers have been published on the effects of mycotoxins since the 1960s when aflatoxin was first identified. Since then, poultry genetics have improved and updated studies are required on how modern genetics respond to mycotoxins in feeds. This review highlights current published data on the effects of feeding various mycotoxins to poultry. The abbreviated names for common mycotoxins used in the paper are:

- AFL – aflatoxin
- FUM – fumonisin
- FUA – fusaric acid
- ZEA – zearalenone
- DON – vomitoxin
- OTA – ochratoxin
- CPA – cyclopiazonic acid

FUSARIUM MYCOTOXINS

Fumonisin, vomitoxin, and zearalenone are common toxins found in corn and are produced by several species of *Fusarium* fungi. The mode of action is unique for each toxin and toxic effects can vary by animal species and animal sex. For instance, zearalenone has been found to have estrogenic effects and has a greater impact on female pigs than male pigs and poultry.

In a joint research project between the University of Georgia and North Carolina State University, (Henry et al., 2000) various levels of purified FUM were fed to 120 day-old Arbor Acres chicks. Chicks were fed four levels of fumonisin from 0 to 80 ppm for 3 weeks, (Table 1).

TABLE 1: Effects of fumonisin on broiler growth performance

Dietary FUM, ppm	Weight, g			Feed : Gain	
	d 7	d 14	d 21	d 8-14	d 15-21
0	129	354	660	1.34 ^a	1.44 ^a
20	124	343	627	1.35 ^a	1.36 ^a
40	135	356	659	1.20 ^{ab}	1.24 ^{ab}
80	135	361	667	1.09 ^b	1.19 ^b

^{ab} Significant difference (P < 0.05)

In the study, no differences in body weight were observed in chicks fed various levels of fumonisin ranging from 20 ppm to 80 ppm compared to birds fed the control diet. However, feed conversion rates (F:G) were improved linearly (P<0.05) as dietary fumonisin increased during weeks 2 and 3 of the study. It is not unusual to see positive, nutritive effects in broilers when feeding low levels of a *Fusarium* mycotoxin (below the toxic threshold; Swamy et al., 2002). It has been repeatedly proven that broilers have a high tolerance to fumonisin when fed the toxin in academic research conditions.

Researchers at the University of Missouri (Li et al, 1999) conducted studies and fed broilers fumonisin B₁ contaminated diets as high as 200 ppm. The group was not able to show any detrimental performance effects of feeding fumonisin to broilers (Table 2). However, birds fed 200 ppm fumonisin B₁ showed lower secondary antibody response (P<0.05) after being injected with the Newcastle disease vaccine. The studies suggest that 200 ppm fumonisin B₁ causes a reduction of immune response although no difference of growth performance was observed.

TABLE 2: Effects of Fumonisin on broiler growth.

Dietary FUM, ppm	Exp I – 3 wks			Exp II – 4 wks		
	Intake, g	Weight, g	FCR	Intake, g	Weight, g	FCR
0	876	718	1.22	1663	1145	1.45
50	921	733	1.26	1733	1196	1.45
100	954	759	1.26	1663	1167	1.42
200	878	693	1.26	1709	1131	1.51

Tardieu et al. (2007) reported that turkeys can tolerate fumonisin as high as 20 ppm in feed. In the study, a total of 300 BUT poults were fed diets containing 0, 5, 10, or 20 ppm fumonisin for 9 weeks (Table 3). No differences in average daily gain (ADG) and feed conversion rate (FCR) were observed between turkeys fed any dietary level of fumonisin. The only performance effect observed in the study was from the group fed 20 ppm fumonisin. They had greater feed intake ($P < 0.05$), when calculated as mean daily feed consumption, than other treatments. There was no difference between treatments on the size of liver, kidney and breast muscle; however sphingolipid ratios in kidneys and livers were affected at 10 or 20 ppm dietary fumonisin. Changes in sphingolipid concentrations have been shown previously (Broomhead et al., 2002) as an early biomarker for fumonisin exposure. Sphingolipid is a major class of lipids found in cell membranes and involved in cell-to-cell communication, cell growth, and multiple other cell functions.

TABLE 3: Effects of fumonisin on turkey growth performance

	Dietary FUM, ppm			
	0	5	10	20
ADFI, g	178 ^a	181 ^a	178 ^a	189 ^a
ADG, g	98	99	99	103
FCR	1.82	1.83	1.81	1.84
Liver, g	77	86	81	83
Kidney, g	35	35	36	37
Breast, g	813	832	857	822

^{a,b} Significant difference ($P < 0.05$)

Swany et al. in 2002 used a total of 360 day-old chicks and fed *Fusarium* mycotoxin contaminated diets for 8 weeks with 3-week starter feed, 3-week grower feed, and 2-week finisher feed. The chicks were divided into 4 treatments with a toxin-free control diet, a low mycotoxins diet (DON 4.7 ppm, FUA 20.7 ppm, ZEA 0.2 ppm), a high mycotoxins diet (DON 8.2 ppm, FUA 20.3 ppm, ZEA 0.6 ppm), and a high mycotoxins diet (DON 9.7 ppm, FUA 21.6 ppm, ZEA 0.8 ppm) with the

addition of 0.2% yeast cell wall. All diets were formulated to an isocaloric and isonitrogenous for each feeding phase (Table 4).

TABLE 4: Effects of Fumonisin on broiler growth performance

Treatments	Feed Intake, g	Weight Gain, g	Gain : Feed
Control	5723	3139	0.548
Low	5786	3218	0.556
High	5514	3010	0.546
High + 0.2% YCW	5543	2957	0.533

The authors reported no significant weight gain, feed intake, or feed efficiency differences between feeding high levels of mycotoxins alone or with the inclusion of 0.2% yeast cell wall for each individual growth phase of the study. During the finisher phase of the study a quadratic effect was observed in intake and gain. This resulted from the low mycotoxin inclusion birds having increased feed intake and weight gain compared to the control and the high mycotoxin inclusion broilers. The authors suggested that feeding low levels of DON and/or ZEA could have nutritive, growth promoting effects in broilers. Results would suggest that broilers can tolerate an average combination of 9.7 ppm DON, 21.6 ppm FUA, 0.8 ppm ZEA for a full grow-out cycle without showing detrimental effects. Adding 0.2% yeast cell wall to contaminated diets had no effect in improving overall broiler performance.

Researchers at University of Guelph completed three different studies to investigate the effects of DON on poultry (Yegani et al., 2006). In the studies, mycotoxin contaminated feed, averaging 12.6 ppm DON and 575 ppb ZEA, was fed to layers and broiler breeders.

- In the first study, a total of thirty-six, 45 week-old, layers were fed either a control diet, *Fusarium* mycotoxins contaminated diet, or the contaminated diet plus 0.2% yeast cell wall for 4 weeks.
- In the second study, a total of forty-two, 26 week-old, broiler breeders were fed test diets for 15 weeks. They found no difference on neurotransmitter concentration in the hypothalamus in layers or broiler breeders. The authors did report an increase in serotonin (5-HT) in the cortex of layers when feeding the *Fusarium* contaminated diet; however, no reduction of egg production or decreased feed intake was reported. In pigs (more sensitive to DON) it is suggested that DON decreases hepatic protein synthesis and results in a hyperaminoacidemia, which increases tryptophan accumulation in the brain. Tryptophan is the precursor of 5-HT and once 5-HT concentration increases in the brain, it activates the satiety center in hypothalamus

and results in decrease feed intake in pigs (Riley and Pestka, 2005).

- In the third study, the group used thirty-six, day-old turkey poults and fed the same 3 diets as the previous studies for 4 weeks, however the diets contained 7 ppm DON and 400 ppb ZEA. Again, there was no difference on neurotransmitter concentration in the hypothalamus and brain cortex (similar to the broiler breeders) between treatments including epinephrine, norepinephrine, dopamine, serotonin and other neurotransmitters. Apparently the response of poultry, including broilers and turkeys, to DON is different than in pigs. With layers, there was an increase in 5-HT in the cortex only but it is unknown if feed intake was affected. It is obvious that layers are not as sensitive as pigs because only the cortex was affected.

Ducks are considered one of the most sensitive avian species to mycotoxins. Davis et al. (1994) reported a high mortality in Pekin ducklings after 2 flocks of ducks were fed contaminated feed (0.3-1.2 ppm DON, 4.5 ppm FUM and 10 ppb AFL). The mortality rate of young ducklings was 20% by day 3 and 50% by day 7. However, two years later, Boston et al. (1996) found no negative effect on growth performance in adult Mallard ducks by feeding DON at level of 5.8 ppm, which would suggest that DON may have not been the cause of death in the previously mentioned field toxicity.

In 2005, Chowdhury et al. used a total of 464 day-old Pekin ducks and fed them different levels of DON and ZEA for 6 weeks (Table 5). The mycotoxins used in the study were not purified but obtained from a natural occurrence in grain. The group reported no difference on weight gain, feed intake, or feed efficiency from feeding high levels of DON and ZEA; and there was no advantage of yeast cell wall addition in the diet. The researchers also did not observe overall differences in plasma calcium, uric acid, white blood cell, lymphocyte, or webfoot thickness between treatments. Minor differences were seen in some 2 or 4-week parameters.

TABLE 5: Effects of *Fusarium* mycotoxins on young ducklings

Treatments	Feed Intake, g	Weight Gain, g	Gain : Feed
Control	7092	2889	0.407
Low ¹	7066	2814	0.398
High ²	6930	2842	0.410
High + 0.2% YCW	7130	2947	0.413

¹ Low contaminated diet contained DON 7 ppm and ZEA 0.6 ppm (average of 3 phases)
² High contaminated diet contained DON 16 ppm and ZEA 1.2 ppm (average of 3 phases)

A group of French researchers (Tardien et al., 2004) force-fed seventy-five 12-week old ducks to reach a total consumption of 0 mg, 100 mg (10 kg of 10 mg/kg FUM corn) or 200 mg (10 kg of 20 mg/kg FUM corn) of fumonisin within 12 days. The body weight and breast muscle were not different between treatments. Numerically, liver size was decreased and the feed conversion rate was increased as fumonisin consumptions increased (Table 6). However, both the liver size and FCR were not statistically different.

TABLE 6: Force fed fumonisin in ducks

Treatment	FUM Intake, mg	Weight, kg	FCR	Breat Mucle, g	Liver, g	Mortality
1	0	5.64	6.32	480	532	0/25
2	100	5.58	7.04	456	526	0/25
3	200	5.54	7.54	470	472	2/25

Based upon the above data, poultry can tolerate high levels of dietary *Fusarium* mycotoxins FUM, FUA, DON and ZEA. Why did Davis and his colleagues observe a high mortality in young ducklings a decade ago? One possible reason may be due to the combination of *Fusarium* mycotoxins and aflatoxin. The contaminated feed contained 10 ppb aflatoxin and moderate levels of DON and FUM. The high duck mortality rate may have resulted from the aflatoxin contamination in the feed, even at such a low level, or another mycotoxin that had not yet been characterized. As previously mentioned and detailed later, aflatoxin is one of the most toxic mycotoxins in poultry.

ASPERGILLUS AND PENICILLIUM MYCOTOXINS

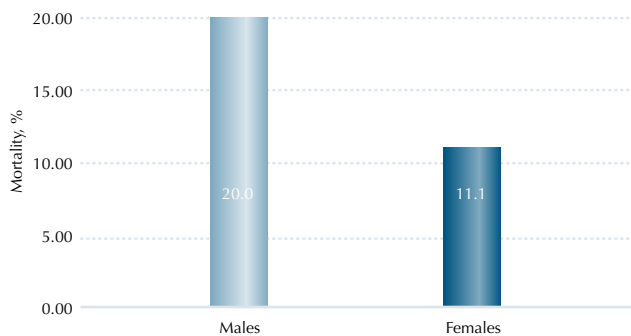
Aflatoxin is only produced by *Aspergillus* species of fungi, where OTA can be produced by both *Aspergillus* and *Penicillium* species of fungi. Both of these species of fungi are primarily considered as storage fungi that are found when grains are inappropriately stored. Even though AFL is carcinogenic, which is more of a concern for human consumption, OTA is more toxic to poultry. Devegowda and Murthy (2005) reported LD50 (dose at which 50% of the animals die) for common mycotoxins in poultry in which OTA was reported at 3.6 ppm and AFL was reported at 6.5 ppm. Huff et al. (1974) reported LD50 for OTA in day-old and 3 week-old broilers to be 2.14 and 3.6 ppm, respectively. Turkeys are less sensitive to OTA with LD50 of 4.63 and 5.9 ppm for day-old and 3 week-old turkeys, respectively (Change et al., 1981). In regards to AFL, the sensitivity of broilers and turkeys to the toxin is opposite from that of OTA, in which broilers are more tolerant than

turkeys.

Aflatoxin is a known carcinogen and is primarily hepatotoxic in poultry, resulting in pale and swollen (fatty) livers. Absorbed aflatoxin is transported to liver and other peripheral cells, where it is metabolized to its active form aflatoxin-8,9-epoxide. The epoxide form is presumed to be responsible for the carcinogenicity, due to its reactivity with nucleophilic site (Leeson et al., 1995). It generates peroxides, damaging cell lipid bilayer membranes, mitochondria membranes, and chromosomes. It further alters protein synthesis, energy utilization, fatty acid metabolism, immune response, and can eventually cause death.

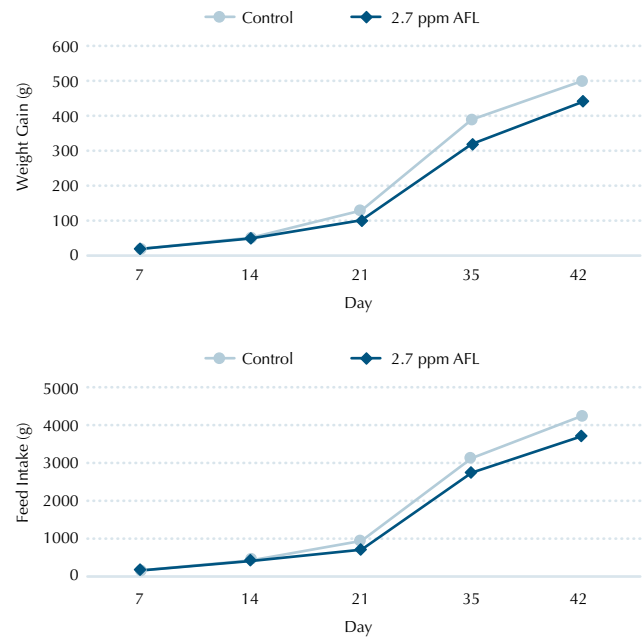
In an unpublished study conducted at LAMIC in Brazil in 2000, a high mortality rate was observed in male and female broilers after feeding 3 ppm AFL for 3 weeks. No mortality was observed in the control birds. The male broilers appeared to be more susceptible to aflatoxicosis because the mortality in males was greater (Figure 2). In the 2003 CAST report, it was stated that the susceptibility of aflatoxins can vary depending on diet, stress, sex, immune state, and drug treatment.

FIGURE 2: High mortality in broilers fed 3 ppm aflatoxin



In another unpublished study conducted at LAMIC in Brazil in 2005, a total of 480 day-old Cobb male birds were fed diets containing either 0 or 2.7 ppm AFL for 41 days. Results showed that birds fed AFL decreased body weight ($P < 0.05$) as early as 14 days (Figure 3). The reduced body weight was mainly due to significantly decreased feed intake ($P < 0.05$; Figure 4) and the feed conversion was not different between treatments. The relative liver weight also increased in broilers fed 2.7 ppm at both 20 and 41 days of the study.

FIGURE 3: Effects of 2.7 ppm aflatoxin on weight gain and feed intake of broilers



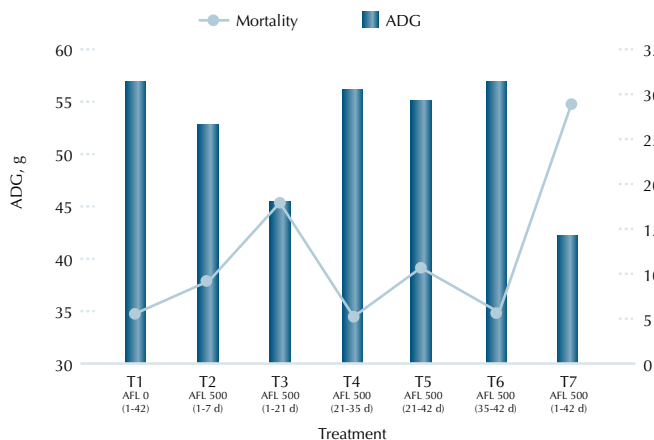
Mariani et al. (1998) conducted a unique trial to demonstrate the impact of AFL on broiler growth when fed at different times during grow-out (Table 7). Birds receiving the control diet (0 ppb AFL, Treatment 1) had the best average daily weight gain (ADG; 57 grams a day) and low mortality (Figure 4). As expected, the birds fed 500 ppb AFL for the complete 42 days (Treatment 7) showed the lowest ADG (42 grams a day) and highest mortality. Birds fed 500 ppb AFL starting day one for 21 days (Treatment 3) showed a significant decrease on weight gain (46 grams a day) and increased mortality, compared to the controls, but were better than birds fed AFL for the entire 42 day study. Birds fed AFL for the first 7 days of the study (Treatment 2) had slightly lower ADG and higher mortality than the control birds.

The remaining treatments (4, 5, and 6) were fed AFL starting day 21 or day 35, for 7 or 21 days, had similar ADG and mortality as compared to control group (with the exception of a slight increase in mortality, Treatment 5). It demonstrated that the young broiler is more sensitive to aflatoxin feeding than older broilers. Also, feeding aflatoxin for a longer period of time resulted in higher mortality, but was less detrimental in older birds (Treatment 5 vs. 3).

TABLE 7: AFL fed to broilers at different times during grow-out

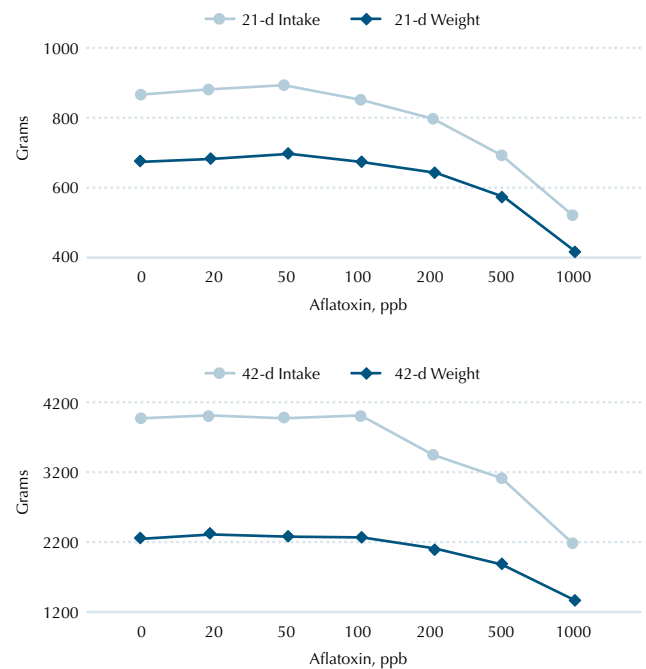
Treatments	Description
1	0 ppb AFL from day 1 to 42 (Control)
2	500 ppb AFL from day 1 to 7
3	500 ppb AFL from day 1 to 21
4	500 ppb AFL from day 21 to 35
5	500 ppb AFL from day 21 to 42
6	500 ppb AFL from day 35 to 42
7	500 ppb AFL from day 1 to 42

FIGURE 4: Average daily gain (ADG) and mortality of broilers fed aflatoxin



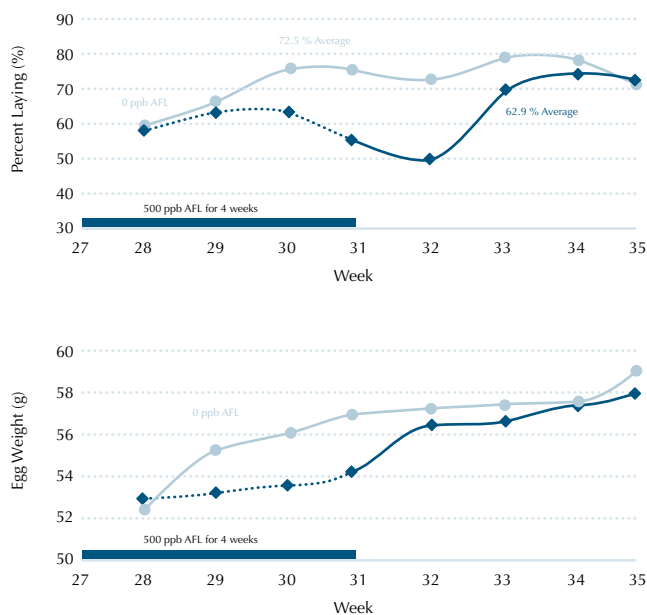
Rauber et al. (2007) recently conducted a trial in which turkeys were fed increasing levels of aflatoxin ranging from 0 to 1,000 ppb. Three hundred and thirty-six day-old turkey poults were divided into 7 treatments and fed diets containing 0, 20, 50, 100, 200, 500, or 1,000 ppb of aflatoxin for 6 weeks. Both weight gain and feed intake significantly decreased as dietary AFL increased above 100 ppb (Figure 5). Poults fed greater than 200 ppb AFL showed a significant growth impact through day 21. However, decreased body weight ($P < 0.05$) was observed when poults were fed greater than 100 ppb AFL through day 42. Results indicate that aflatoxin has more of an accumulative toxic affect in turkeys (increased growth depression at lower levels at day 42 vs. 21). However, it is apparent that the reduced weight gain was mainly due to decreased feed intake. The results are similar to the observations in the previously mentioned 2005 LAMIC broiler study.

FIGURE 5: Feed intake and weight of turkeys fed aflatoxin at 21 or 42 days of age



Rosa (2001) conducted a feeding study in layers to investigate AFL effects on egg production and egg weight. Layers at 28 weeks of age were used in the study and were divided into two groups. Group A was fed a control diet that contained 0 ppb aflatoxin for 8 weeks. Group B was fed a diet that contained 500 ppb AFL for 4 weeks and then shifted to a 0 ppb AFL control diet for another 4 weeks. Results clearly indicated that layers receiving the diet contaminated with AFL significantly decreased egg-laying compared to the control layers (Figure 6). The percentage of egg-laying started to decrease about 2 weeks after AFL feeding began and decreased linearly thereafter. At week 4 the difference between the two groups was 25% (75% vs. 50%), which is a 33% reduction of egg production and is a significant loss for egg producers. After AFL contaminated feed was removed, it took 4 weeks for these layers to recover to the production level of the control birds. Besides reduced egg production, egg weights also decreased when AFL was present in feed (Figure 6). Unlike the percent of egg-laying, the egg weights recovered (to control level) only one week after AFL contaminated feed was removed.

FIGURE 6: Effects on layer production when aflatoxin was fed and then removed



Where AFL is considered to be more hepatotoxic, the primary target organ of OTA toxicity is the kidneys. Phenylalanine is one of the metabolites of OTA and is believed to be the cause of OTA interference with DNA, RNA, and protein synthesis. Ochratoxin interferes with phosphoenolpyruvate carboxykinase (PEPCK) production in the kidneys, which is required for glucose and glycogen production (Leeson et al., 1995).

In a study conducted in Brazil by Santin et al. (2002), 288 broilers were fed 2 ppm OTA from 1 to 42 days of age. Animal performance was significantly reduced when OTA was fed to both 21 and 42 days of age (42 day performance can be found in Table 8). Body weight gain was reduced by 36% when OTA was included at 2 ppm. The relative liver and kidney weights were significantly higher at both 21 and 42 days of age when OTA was included in the diets (42 day organ weights are in Table 8). Kidney as well as liver lesions are common in OTA exposure. The kidneys were pale and swollen and the livers were a yellowish color and enlarged as is commonly seen in OTA exposure. The significantly reduced serum total proteins (Table 8), observed with OTA intake, probably results from OTA inhibiting protein synthesis.

TABLE 8: Final performance and relative organ weight and 18 day serum total protein results in broilers exposed to ochratoxin

Treatment	42-d Feed Intake, g	42-d Weight Gain, g	42-d Feed Conversion, g:g	42-d Liver Weight, g/100g BW	42-d Kidney Weight, g/100g BW	18-d Serum Protein, g/dL
0 ppm OTA	3,893	2,328	1.672	2.17	0.70	3.73
2 ppm OTA	2,751	1,483	1.856	4.43	1.24	3.00

In a study conducted by Gentles et al. (1999), day-old broilers were fed either 2.5 ppm OTA, 34 ppm cyclopiazonic acid (CPA; can also be produced by *Aspergillus* or *Penicillium* species of fungi), or a combination of both for 21 days. Both 14 and 21 day body weights were reduced by both OTA and CPA (Table 9); however, feed conversion was not affected by either toxin in this study. The relative kidney weights were increased by OTA (typically observed with the toxin; Table 9) but were not affected by CPA alone. Unlike the study by Santin et al. (2002), relative liver weights were not significantly affected by OTA alone (Table 9). However, serum total protein (including albumin; Table 9) was significantly lower in the birds fed OTA alone (Santin et al., 2002). A synergistic effect was observed in this study as shown by the significant change (increase or decrease) in each parameter in Table 9 when OTA and CPA are fed in combination compared to results of each toxin fed alone. For example, liver weight was not significantly affected by either OTA or CPA alone in this study, but when toxins are combined the liver is overwhelmed and enlargement occurred.

TABLE 9: Body weight, relative organ weight, serum protein results in broilers exposed to ochratoxin (OTA) and cyclopiazonic acid (CPA)

Treatment	14-d Body Weight, g	21-d Body Weight, g	Liver Weight, g/100g BW	Kidney Weight, g/100g BW	Serum Protein, g/dL	Serum Albumin, g/dL
Control	372 ^a	675 ^a	3.30 ^b	0.55 ^c	2.69 ^a	1.21 ^a
2.5 ppm OTA	300 ^b	521 ^b	3.71 ^b	0.73 ^b	2.23 ^b	1.03 ^b
34 ppm CPA	307 ^b	556 ^b	3.32 ^b	0.51 ^c	2.64 ^a	1.11 ^{ab}
2.5 ppm OTA, 34 ppm CPA	258 ^c	441 ^c	4.79 ^a	0.89 ^a	1.88 ^c	0.80 ^c

^{a,b,c} Significant difference (P < 0.05)

MYCOTOXIN RESIDUES IN BROILER MEAT AND EGGS

There has been little research investigating mycotoxin residues in poultry meat and eggs during the past two decades. A USDA report from early 1970 indicated no aflatoxin residue found in broiler meat and eggs after feeding aflatoxin contaminated feeds to broilers and layers; however, a significant increase of AFLM₁ in cow milk was observed (Table 10). Danicke et al. (2002) fed diets contaminated with 17.6 ppm DON and 1.58 ppm ZEA to layers for 16 weeks and no ZEA residue was found in eggs. In a review by Galvano et al. (2005), AFL was reported to contaminate eggs at various transfer levels compared to feed levels. The greatest, most efficient, transfer rate was reported in research conducted by Jacobson and Wiseman (1974), in which between 250:1 and 430:1 (AFL in feed:AFL in eggs) was reported. However, Galvano et al. (2005) cited that much lower transfer rates, between 55000:1 and 125000:1, were reported by another group (Lotzsch and Leistner, 1976). Galvano et al. (2005) also cited a more recent study conducted by Pietri et al. (2001), where an approximate 48000:1 transfer ratio was calculated when feed contained 20 to 100 ppb AFL resulting in eggs averaging 0.002 ppb AFL. The strict USDA regulations for AFL in layer feed (20 ppb in United States; CAST 2003) would result in negligible contamination of AFL in eggs.

TABLE 10: Aflatoxin residues after feeding aflatoxin contaminated feeds

Species	AFL Dosage and Time	Results
Broiler	1600 ppb for 8 weeks	No AFL residue found in meat
Layer	2700 ppb	No AFL residue found in egg
Dairy	Total 67~200 mg a week	70~154 ppb AFL M ₁ in milk

Galvano et al. (2005) referenced a European food and agriculture organization report that lists various mycotoxin residues in animal proteins (the sources of mycotoxin contamination were not discussed in the report). The contamination is assumed to have come from grains and finished feeds. Ochratoxin contamination in meat has been a concern in European countries, however pork has been mentioned in several reports for this toxin (Galvano et al., 2005). Mycotoxins with longer residence times in the body, such as ochratoxin (due to strong binding to plasma proteins), are typically a greater concern for contamination of animal meat intended for human consumption. Veldman (2003) discussed that the concern for human mycotoxin consumption should be from plant-derived foods, with little originating from animal-derived foods.

TABLE 11: Examples of food from animal origin which may be naturally contaminated with mycotoxins

Mycotoxins	Occurrence	Reported Highest Levels
Aflatoxin	Eggs	0.4 ppb
	Pig liver	0.5 ppb
Ochratoxin A	Pig liver	98 ppb
	Sausages	3.4 ppb
Zearalenone	Pig liver and muscle	10 ppb

CONCLUSIONS

Poultry are generally very sensitive to aflatoxin and the effects vary by species and sex. Small quantities of aflatoxin in feed (tens to hundreds ppb) can cause increased mortality, reductions in weight gain, feed intake, egg production, egg weight, and profit. Among the species, duck is the most sensitive to aflatoxin followed by turkey. Broilers and layers are equally sensitive to aflatoxin but less sensitive than ducks and turkeys. Unlike with aflatoxin poultry are much more tolerant to fumonisin, fusaric acid, vomitoxin, and zearalenone (*Fusarium* mycotoxins) toxicity. For instance, day-old broiler chicks fed fumonisin, as high as 200 ppm for 4 weeks, did not show negative impacts on weight gain or feed intake (under controlled laboratory conditions; Li et al., 1999). The dietary levels of *Fusarium* mycotoxins (not including T-2 toxin) that could cause economical loss (decreased weight gain and poor feed conversion) is much higher than with *Aspergillus* or *Penicillium* mycotoxins (Table 12).

TABLE 12: Mycotoxin sensitivity in broiler, layer, turkey and duck.

Mycotoxins	Broiler	Layer	Turkey	Duck
Aflatoxin	+++	+++	++++	+++++
Ochratoxin	+++	++	+++	++
T-2 Toxin	++	++	+++	+++
Zearalenone	-	-	-	-
Fumonisin	-	-	-	+ (20 ppm)
Fusaric Acid	-	-	-	-
DON (Vomitoxin)	-	+ (18 ppm)	-	-

+ : sensitive; - : not sensitive

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