MANAGING MOLDS AND MYCOTOXINS IN POULTRY FEEDS

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INTRODUCTION

The term "mycotoxin" is derived from myco meaning fungi and toxin, a poison; hence, the word literally means poisons from fungi. There are more than 300 known mycotoxins, but very little is known about the relative toxicity of most of these compounds. The fungi that produce mycotoxins can infect and produce mycotoxins in cereal grains such as corn and sorghum in the field during the growing stage, at harvest time, and during storage. As grains progress through harvesting, storage, feed manufacture, and delivery to the farm, the level of mycotoxin contamination generally increases (Table 1). Early suppression of mycotoxin levels, thus aids in reducing the final amount present in the poultry house.

Although problems related to mycotoxins have been observed in human and animal populations for centuries (NRC, 1979), concerns in commercial poultry production began to focus on aflatoxin in 1960 when more than 100,000 turkey poults in Great Britain died from what was called "Turkey X disease" (Blount, 1961). This was later confirmed to be the result of aflatoxin from a toxic groundnut meal. Since that time, problems with mycotoxins have been increasingly observed in all types of animal feeds. In part, this is due to changes in the methods used to harvest and store corn over the last 40 to 50 years. Fifty years ago, corn was harvested by hand or by machines that picked the whole ear, and was rather dry at harvest. This system was replaced by machines that removed the ears from the stalk and shelled them in the field. To reduce loss from shattering, corn is picked and shelled at a higher moisture content, and it molds quickly if rapid drying is not used in storage. Mechanical shelling of wet corn also causes more kernel breakage and allows greater entry of fungal spores into the kernels (Table 2). As yields have increased, drying facilities have often not kept pace and when coupled with escalated energy costs, this has resulted in more problems with high moisture corn.

Aflatoxin is the most commonly known mycotoxin in poultry feeding, but a number of other mycotoxins can result in adverse effects. For example, the mycotoxin ochratoxin A (OA) is approximately three times more toxic to poultry than is aflatoxin (Table 3), and when present in combination, the depressing effects can be even more severe. Mycotoxins generally affect the liver (hepatotoxins) or the kidney (nephrotoxins). As a result, a number of adverse effects arise from mycotoxin contamination. The characteristic symptoms of mycotoxicosis in animals are a manifestation of the effects of mycotoxins on critical metabolic processes in the animals. Some of the most common effects include:

1. Pale and enlarged livers
2. Swollen kidneys
3. Oral lesions
4. Impaired immune function
5. Increased susceptibility to bruising
6. Decreased egg production and egg weight
7. Decreased bone strength
8. Increased intestinal fragility
9. Reduction in pigmentation
10. Inhibition of nutrient absorption
11. Reduced growth rate

The severity of the response to mycotoxins depends largely upon the specific mycotoxin present and the level of contamination. High levels can result in extreme mortality in a short period of time (Table 4), but more often, the adverse effects of low level mycotoxin contamination represents the greatest problem to the poultry industry. USDA workers (Bacon and Burdick, 1977) made a study of fungal population and growth in broiler houses that were termed "problem houses" compared to control houses; the "problem houses" consistently produced broilers with suboptimal performance.

An analysis of the mold flora taken at various locations throughout both the "problem" and control houses revealed a definite difference in the types and degree of growth of toxic-producing molds in the "problem" houses compared to the control houses.

In a classic case study (Jones *et al.*, 1982), broiler growers from different companies were divided into three categories (good, mediocre, and poor) based on previous performance. Feed samples within the house were examined for mycotoxins and performance correlated to incidence and levels. Growers classified as "good" had a lower incidence of aflatoxin in their feed with a lower level of contamination than those classified as "mediocre" or "poor", even though receiving feed from the same mill (Table 5). As a result, their birds grew better and more efficiently with less condemnation at the processing plant and consequently, these growers received a higher payment for their chicks.

Establishing a "safe" level of mycotoxins is literally impossible. The response to mycotoxins is influenced by a number of factors such as nutritional adequacy of the diet, the age and specie of the animal, environmental and disease stress, and the type of mycotoxins present. Numerous studies have demonstrated that various mycotoxins found in combination have more severe effects upon poultry than the individual mycotoxins alone. Thus, the poultry industry should make all effort to reduce or eliminate the incidence or severity of mycotoxin contamination at every point in the production cycle.

Controlling the adverse effects of mycotoxins requires a multi-faceted approach, involving purchase of quality grains, management practices in the feed mill and in growing facilities, adjustment in nutrient content of the diet, and use of selected feed additives. Each of these plays an important role in prevention or control of mycotoxicosis.

FEED MILL STRATEGIES

Even though incoming feed ingredients may test negative to aflatoxins, there are a number of areas in the mill that may serve as sources of contamination. Proper attention to these areas aid in maintaining low levels of mycotoxins in finished feeds. Areas that warrant special attention include:

Storage areas Of prime concern are those areas where ingredients can build up over time, such as in crevices or corners of storage bins. Where possible, a routine should be followed where storage bins can be periodically emptied and throughly cleaned.

Ingredient bins Determine if feed build up is occuring on the side walls or in the bottom of screw conveyors or elevator legs. Check for possible leaks in the roof. Clean out periodically.

Elevators Pay particular attention to build up of feeds in buckets and the bottom of the elevator leg where moisture can be a problem.

Pellet Cooler Due to the high temperature and moisture of pelleted feeds, the pellet cooler can be an excellent area for continuous mycotoxin contamination. Check

and thoroughly clean, at least weekly.

Feed storage Be sure that feed is not building up on the side walls and in conveying equipment. All bins and associated equipment should receive periodic cleanout.

Feed trucks Careful attention should be given to augers or blower hoppers. All feed residues should be removed at every other day and throughly cleaned on a periodic basis.

MANAGEMENT STRATEGIES

A number of different management strategies can be employed at the farm level to alleviate the development of mycotoxins in feeds and consequently reduce the adverse effects. It has been determined that the residence time of feed at the farm is highly correlated with both the frequency and concentration of aflatoxin (Table 6); thus, more frequent delivery of feed with shorter residence time is one means of reducing problems with aflatoxicosis.

In a field trial conducted by Good and Hamilton (1981), it was shown that reducing the normal quantity of feed delivered per house by half resulted in improved body weight and feed conversion and a reduction in production cost (Table 7). Subtracting the extra cost associated with increased number of deliveries from the difference in production cost indicated that there was a net profit of $.0064/kg in favor of decreased feed quantities.

Producers have little or no control over the frequency of feed delivery, but can aid in the control of mycotoxicosis through management practices. A field trial was conducted (Hamilton, 1975) in which moldy caked feed was removed from feed delivery trucks, storage bins, feed conveyors and feed troughs by scrubbing and disinfecting with a 5% sodium hypochlorite (liquid bleach) solution (Table 8). Two farms with side-by-side houses were compared. These farms were consistently in the bottom quarter of growers. It was not possible to obtain feed conversion data, but the "cleaned" facilities resulted in an improvement in body weight, increased serum carotenoid content (indicating better nutrient digestibility), and improved carcass grades.

Some of the areas on the farm that warrant special attention include the following:

Feed bins

Feed bins are probably one of the most troublesome areas in regard to buildup of mycotoxins. Bins should be checked periodically to make certain feed is not building up in auger troughs or on the bin walls. Bins should have good ventilation to minimize heat buildup and "sweating" of sidewalls.

Bagged feed

Storage of finished feed or raw material in bags presents problems especially in humid climates. Condensation and mold growth can occur easily in solid walled and lined bags as temperatures change. Jute and plastic mesh bags are particularily troublesome if recycled and used more than once for feed. Furthermore, the open nature of these bags allows humidity and mold spores to penetrate to the feed within.

Feeders

Generally conditions are ideal for mold growth and contamination in and around feeders. This area should be routinely checked for excess buildup and moisture.

"Old feed" procedures

A standardized procedure should be followed regarding the use of old feed after the flock has been removed. Bringing back leftover feed to the mill can be a significant source of contamination.

Between flock procedures

All facilities should be throughly cleaned before new birds are placed back into the houses. Special attention should be given to

all feed storage and handling equipment.

USE OF MOLD INHIBITORS

Feed manufacturers have increasingly incorporated various mold inhibitors in their diets. Many feed mills have installed systems to routinely spray mold inhibitor on grains during the receiving process, while others add it to the feed during mixing. A number of products are available, generally in the form of low molecular weight organic acids and their salts; propionic acid or products containing this acid are widely used. These may vary widely in cost and relative effectiveness (Dixon and Hamilton, 1981; Paster, 1979; Paster *et al.*, 1985; Paster *et al.*, 1987). It is important to remember that these are fungistats and not fungicides; that is, they only inhibit growth of molds and do not inactivate any toxins already present. Mold inhibitors will not keep mold growth in check indefinitely. In general, the salts of the acids last longer, but are not as effective as the free acids; the volatile free acids have greater efficacy by are not as long lasting. Both the difference in efficacy and the length of time that the mold inhibitors maintain efficacy is thought to be related to the volatilization of these acids as the more volatile the acid the better the penetration. However, because of their greater volatility, the more rapidly they are dispersed.

PELLETING

A commonly overlooked aspect of pelleting is that it influences the microbiological burden of the feed. The high temperatures encountered during pelleting can reduce the incidence of salmonellosis (MacKenzie and Bains, 1976), *enterobacteriaceae* (Mossel, 1971), and the counts of molds in feeds (Tabib *et al.*, 1981). Studies with poultry feed pelleted with or without a propionic acid-based mold inhibitor indicated that pelleting and propionic acid interacted to reduce the mold count in a model system (Tabib *et al.*, 1984). It appeared that the effectiveness of propionic acid as a mold inhibitor can be greatly increased by the pelleting process and that a decrease in the fungal burden of feed is an attribute of pelleting.

DIETARY MANIPULATIONS

Various nutritional strategies have been employed to alleviate the adverse effects of mycotoxins. Some of these have been effective, while others appear to have little effect.

Crude protein

Because mycotoxins typically reduce nutrient absorption, one approach to alleviate these effects has been to increase levels of critical dietary nutrients. Because aflatoxin metabolites appear to affect protein and amino acid metabolism, researchers have examined the possibility of increasing dietary protein levels as a means of alleviating aflatoxicosis. Smith *et al.* (1971) demonstrated that when broilers were fed diets containing 5 ppm aflatoxin, increasing the crude protein level from 20 to 30% alleviated the growth, depressing effects of aflatoxin (Table 9). More recently, Gibson *et al.* (1989) observed that increasing dietary crude protein helped alleviate but did not eliminate the adverse effects of ochratoxin A on body weight and feed conversion; mortality rates did not appear to be affected (Table 10). However, increasing protein levels is a costly approach to mycotoxin control.

Methionine supplementation

Addition of specific amino acids that tend to be limiting, such as methionine, may be a more cost-effective approach. Veltmann *et al.* (1981; 1983) observed that increasing the dietary total sulfur amino acids (TSAA) to levels in excess of NRC protected chicks from

the growth depressing effects of aflatoxin (Table 11), possibly through an increased rate of detoxification by glutathione, a sulfur amino acid metabolite.

Phenylalanine supplementation

In vitro studies have indicated that the toxicity of Ochratoxin A may be alleviated by phenylalanine supplementation (Creppy *et al*., 1980; Klinkert *et al.*, 1981). However, in studies by Gibson *et al*. (1990), addition of phenylalanine to low-protein, OA-contaminated diets did not improve body weight or feed conversion, but appeared to reduce mortalty (Table 12). Further studies by Bailey *et al*. (1990) suggested that supplemental phenylalanine improved the health status of birds fed diets containing OA.

Dietary lipids

Smith *et al*. (1971) incorporated 2, 6, or 16% cottonseed oil into semipurified diets containing 10 ppm aflatoxin. Body weight was not improved by the oil supplementation, but mortality was significantly reduced. In a further study to evaluate the effects of different types of lipids, 2 or 16% olive oil, coconut oil, safflower oil, or animal fat were included in low-fat diets with 10 ppm aflatoxin. The higher levels of each fat reduced mortality and in some instances, improved body weight (Table 13). The authors concluded that the lipids exerted their effects in part by interfering with absorption of the aflatoxin. Supplementation with olive oil and safflower oil, both sources of unsaturated fatty acids, also improved body weight. Lanza *et al*. (1981) suggested that diets containing higher levels of linoleic acid supported better feed conversion and lower mortality in chicks fed diets with aflatoxin. This may explain some of the benefits observed with safflower oil in the studies by Smith *et al*. (1971).

Vitamin supplementation

The reduced fat absorption generally associated with aflatoxicosis leads one to assume that increasing the levels of fat-soluble vitamins in the diet may aid in alleviating adverse effects. However, extensive research has indicated that because most commercial broiler diets already contain fat-soluble vitamins in quantities several fold greater than their minimum requirements, addition of increased levels of A, D, E, or K were of little benefit in alleviating the clinical symptoms of aflatoxicosis. In one study (Hamilton *et al*., 1974), broilers fed diets with NRC recommended levels of supplemental vitamins did not differ in their response to dietary aflatoxin as compared to birds fed diets with four times the NRC recommended levels (Table 14).

A possible exception to this is vitamin D. The typical vitamin D_3 supplement fed to poultry must undergo two different metabolic conversions to become the active metabolite. These changes take place in the liver and the kidney, two organs that are severely affected by mycotoxicosis. Bird (1978) reported that 1 ppm of aflatoxin B_1 in a poultry diet increased the vitamin D requirement by 8.84 ICU/kg of diet. In a field outbreak of rickets in a broiler flock in Germany, addition of vitamin D to the diet alleviated the rachitic lesions, even though the diet was apparently adequate in calcium, phosphorus, and vitamin D. Although no toxins could be found in the feed using the techniques available at that time, they suspected that some toxin was present that interfered with vitamin D metabolism (Gedek *et al*., 1978; Kohler *et al.*, 1978). However, Britton and Wyatt (1978) reported that feeding 2.5 ppm aflatoxin to broilers for four weeks does not greatly alter vitamin D metabolism. Nevertheless, dosage of drinking water with a water-miscible D_3 product remains one of the first responses to an outbreak of "field rickets".

Recent work with fusarium toxicity (Nagaraj *et al.*, 1994) suggests that an anti-thiamin factor is a major toxic factor of *Fusarium proliferatum* M-7176; supplementation of diets with thiamin aided in ameliorating the toxicity of the mycotoxin in chick diets.

Antibiotic supplementation

Addition of 55 ppm of the broad spectrum antibiotic chlortetracycline to broiler diets (Smith *et al.*, 1971) helped alleviate, but did not overcome the adverse effects of 10 ppm aflatoxin on body weight, feed conversion, and mortality (Table 15).

Antioxidants

Shen *et al*. (1994) have recently shown that aflatoxin B_1 causes lipid peroxidation in rat liver. The trichothecenes deoxynivalenol (DON) and T-2 toxin have also been shown to stimulate lipid peroxidation in rat liver along with a depletion of hepatic glutathione (Rizzo *et al.*, 1994). Antioxidants aid in the overall detoxification process in the liver and in cells and thus, may aid in alleviation mycotoxicosis. Larsen *et al*. (1985) reported that the growth depression caused by aflatoxin B_1 was significantly less in birds given an antioxidant (BHT) than in those fed diets without the antioxidant. Ehrich *et al*. (1986) noted that antioxidants differed in their ability to alleviate aflatoxicosis; BHT was able to relieve the adverse effects of aflatoxcosis while ethoxyquin did not. It should be noted that in both studies BHT was added at levels of eight to 30 times the normal usage level and in no case were the adverse effects of aflatoxicosis completely overcome.

Both vitamin E and selenium are involved in the formation of glutathione peroxidase, a compound vital in the cellular detoxification mechanism. Hoehler and Marquardt (1996) reported that lipid peroxides are formed in vivo by T-2 toxins and especially by OA and that these effects can be partially counteracted by an antioxidant vitamin such as vitamin E, but not by vitamin C. Gregory and Edds (1984) reported that selenium enhances the formation of water-soluble conjugated forms of aflatoxin which promotes the clearance of the toxin and enhanced chick growth. Dalvi and McGowan (1984) reported that inclusion of reduced glutathione in drinking water of birds fed diets with 10 ppm aflatoxin B_1 alleviated some of the adverse effects of the aflatoxicosis. Cystine, a sulfur-containing amino acid, is involved in the formation of glutathione; the role of increased dietary sulfur amino acids on alleviation of aflatoxicosis has been previously mentioned.

NON-NUTRITIVE ADSORBENTS

A variety of physical, chemical, and biological approaches to counteract the mycotoxin problem have been reported (Doyle *et al.*, 1982; Samarajeewa *et al.*, 1990), but large-scale, practical, and cost-effective means for complete detoxification of mycotoxin-containing feedstuffs are limited. One of the most promising aspects of control of mycotoxicosis has been the discovery that various adsorbents may sequester aflatoxins and reduce their adverse effects (Phillips *et al*., 1988; Kubena *et al*., 1990a; 1990b; 1993a; 1993b; Harvey *et al*., 1993; Scheideler, 1993; Abo-Norag *et al*., 1995). Some types of hydrated sodium calcium aluminosilicates (HSCAS) are known to sequester aflatoxins and are often added to poultry feed[1]; however, these have not proven effective against other mycotoxins such as ochratoxin A (Huff *et al*., 1992). As shown in Figure 1, the adverse effects of aflatoxin are mediated by an HSCAS product (NovaSil); however, this product was not effective against T-2 toxins (Figure 2) or DAS (Figure 3). The specificity of various HSCAS products against aflatoxins and other mycotoxins varies widely and should be demonstrated for the particular

product considered for dietary supplementation. An excellent review of the prevention of toxic effects of mycotoxins by means of nonnutritive adsorbent compounds was recently published (Ramos *et al*., 1996).

SUMMARY AND CONCLUSIONS

Minimizing the adverse effects of mycotoxins in poultry diets is not a simple task that can be accomplished with one intervention strategy. It begins with the purchase of quality grains (characterized by low moisture content and minimal broken kernels), followed by proper handling of grains during mixing and delivery.

Routine mycotoxin analysis of feed ingredients is an important step in a control program, as it will enable one to determine the amount of mold inhibitor that must be used and the extent to which dietary modifications must be carried out. There are many ELISA assay kits now available that are relatively inexpensive and do not require elaborate laboratory facilities. Mycotoxins to be assayed should include aflatoxins, zearalonone, and deoxynivalenol.

A rigid and routine sanitation program of the feed mill along with attention to feed handling and distribution systems on the farm will aid in reducing the buildup of mycotoxins in the feed. Minimizing feed residence time at the farm level by means of more frequent delivery should be given high priority in a mycotoxin reduction program.

Where mycotoxins are known or suspected to be a problem the use of effective mold inhibitors early in the feed manufacturing process is highly recommended. The addition of proven adsorbents to the diet may aid in alleviating some of the adverse effects. Dietary measures to aid in combating mycotoxicosis may include elevation of crude protein or methionine levels, use of fat sources high in unsaturated fatty acids, and supplementation with a broad spectrum antibiotic.

Increased vitamin therapy may be of some benefit, particularly thiamin, vitamin E, and vitamin D. Although increased levels of the antioxidant BHT has proven beneficial in experimental trials, the quantity needed to elicit an improvement does not appear to warrant this practice. However, diets should be adequately fortified with an antioxidant, selenium, and vitamin E as they interrelate in the overall cellular detoxification scenario.

Because of the ubiquitous nature of mycotoxins in nature, the problem of minimizing mycotoxicosis will not be easy to solve and will require constant attention to detail throughout the entire process of grain harvest, shipping, storage, feed manufacturing, and animal production. No one segment of the animal production chain can totally protect or prevent the problems associated with mycotoxicosis. Complete cooperation by all involved persons is needed to minimize the adverse effects and enhance overall animal production.

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Table 1. Survey of feed samples for aflatoxin

Location	% Positive samples
Corn	30
Soybean meal	5
Other ingredients	0
Feed from mill	52
Feed from troughs in house	91

Hamilton, 1974. Poultry Digest (Dec.):509-511.

Table 2. Mold growth on stored intact and broken corn kernels

	Months of storage			
Kernel condition	0	1	2	7
	---------- (% moldy) ----------			
Intact	8	5	30	35
Broken	55	62	65	83

Cook, 1991. Proc. BASF Technical Symposium. pp. 106-114.

Table 3. LD_{50} values of ochratoxin and aflatoxin in chickens

Age	Ochratoxin (mg/kg)	Aflatoxin (mg/kg)
1 day old	2.1 $\pm$ 0.5	6.8 $\pm$ 0.5
3 wk old	3.6 $\pm$ 0.6	9.7 $\pm$ 0.8

Huff *et al.*, 1974. Poultry Sci. 53:1585-1591.

Table 4. Mortality from ochratoxicosis in a flock of 8,000 poults

Age (days)	No. Dead poults	% Mortality Cumulative
6	19	.23
7	12	.38
8	57	1.10
9	583	8.38
10	2466	39.21
11	983	51.50
12	863	62.28
13	370	66.91
14	547	73.75
15	1033	86.66
16	826	96.98

Hamilton *et al.*, 1982. Poultry Sci. 61:1832-1841.

Table 5. Relationship of aflatoxin levels and relative performance of broilers in commercial production facilities

Parameter	Grower classification		
	Good	Mediocre	Poor
Number of growers	10	10	10
Number birds marketed	122,052	110,290	105,267
Average weight (lbs)	3.88	3.83	3.79
Average feed conversion (lb/lb)	2.13	2.15	2.16
Livability (%)	95.98	95.61	92.78
Condemnations (%)	1.39	1.19	1.73
Grower payment (cents/chick)	12.02	11.49	10.86
% Aflatoxin positive feed	18.04	22.08	31.25
Aflatoxin concentration (ppb)	6.13	6.49	13.99

Jones *et al.*, 1982. Poultry Sci. 61:861-868.

Table 6. Effects of residence time of feed on the farm and the incidence and severity of aflatoxin contamination

Age of feed (days)	Samples	Aflatoxin (ppb)	Aflatoxin (% positive)
1-5	132	7.9	20.5
6-10	64	8.0	23.4
11-15	20	10.7	30.0
16-20	6	27.9	66.7

Jones *et al.*, 1982. Poultry Sci. 61:861-868.

Table 7. Effects of varying the feed residence time by reducing the quantity delivered on productivity of broiler flocks

Feed per delivery	Birds placed	Body weight	Feed Conversion	Production Costs
(kg)		(kg)	(feed/gain)	($/kg)
2,740	42,357	1.64	1.967	0.3227
5,440	42,192	1.59	2.019	0.3302

Good and Hamilton, 1981. Poultry Sci. 60:1403-1405.

Table 8. Effect of clean feed handling equipment on performance and carcass quality of broilers

Parameter	Control Houses	Cleaned Houses
Body weight (kg)	1.53	1.62
Serum carotenoids (ppm)	21.7	26.1
Carcass grades (%)		
#1	28.8	59.6
#2	59.5	37.9
#3	11.2	2.5
#4	.25	0
#5	.25	0
Weighted grade avg.	1.84	1.43

Hamilton, 1975. Poultry Sci. 54:1706-1708.

Table 9. Effect of dietary level of crude protein and aflatoxin on body weight of broilers

	Dietary aflatoxin level	
% Protein	0 ppm	5 ppm
	--- g ---	
10	180	137*
20	318	254*
30	389	368

*Body weight significantly less than same protein level w/o aflatoxin.
Smith *et al.*, 1971. Poultry Sci. 50:768-774.

Table 10. Effect of dietary protein on body weight, feed conversion, and mortality of broilers fed diets containing ochratoxin A (OA)

OA (mg/kg)	% Dietary protein 14	18	22	26	Mean
21-d Body weight (g)					
0	464	613	645	638	590[a]
2	286	422	483	509	425[b]
4	141	190	241	275	212[c]
Mean	297[c]	408[b]	456[ab]	474[a]	
21-d Feed conversion (g:g)					
0	1.90	1.73	1.52	1.51	1.67[b]
2	1.94	1.74	1.63	1.78	1.77[b]
4	2.09	2.51	1.88	2.08	2.14[a]
Mean	1.98[a]	1.99[a]	1.68[b]	1.79[b]	
21-d Mortality (%)					
0	0.0	2.5	2.5	5.0	2.5[b]
2	5.0	2.5	2.5	0.0	2.5[b]
4	35.0	27.5	7.5	17.5	21.9[a]
Mean	13.3[a]	10.8[a]	4.2[a]	7.5[a]	

Gibson *et al*., 1989. Poultry Sci. 68:1658-1663.

Table 11. Interaction of aflatoxin with total sulfur amino acid content of broiler diets

Percent of NRC TSAA	Dietary aflatoxin (ppm) 0	1.25
	--- Body weight (g) ---	
66	500[c]	411[d]
100	660[a]	573[b]
134	653[a]	627[a]

Veltmann *et al.*, 1981. Poultry Sci. 60:1748 (Abstr.)

Table 12. Effects of phenylalanine supplementation on body weight, feed conversion, and mortality of broilers fed diets containing ochratoxin A (OA)

OA (mg/kg)	% Supplemental phenylalanine[1] 0.0	0.8	2.4	Mean
21-d Body weight (g)				
0	423	375	222	339[a]
4	148	144	121	138[b]
Mean	285[a]	259[a]	171[a]	
21-d Feed conversion (g:g)				
0	2.10	2.05	3.04	2.39[b]
4	3.48	2.97	3.45	3.30[a]
Mean	2.79[a]	2.51[a]	3.24[a]	
21-d Mortality (%)				
0	2.5	2.5	5.0	3.3[a]
4	42.5	12.5	15.0	23.3[a]
Mean	22.5[a]	7.5[a]	10.0[a]	

[1]Basal diet contained 14% crude protein and 0.79% phenylalanine.
Gibson *et al.*, 1990. Poultry Sci. 69:414-419.

Table 13. Effect of different levels of supplemental fats on aflatoxicosis in broilers fed diets with 10 ppm aflatoxin

Treatment	21-day weight (g)	Mortality
2% olive oil	211	24/50
16% olive oil	263*	10/50
2% coconut oil	212	27/50
16% coconut oil	237	3/50
2% safflower oil	325	16/50
16% safflower oil	421*	0/50
2% animal fat	347	22/50
16% animal fat	347	0/50

*Significant increase in body weight compared to 2% level.
Smith *et al.*, 1971. Poultry Sci. 50:768-774.

Table 14. The effect of increasing levels of supplemental vitamins on body weight of broilers during aflatoxicosis

	Dietary aflatoxin level	
Vitamin level	0 ppm	5 ppm
	- g -	
NRC	429a	252b
4 X NRC	407a	252b

Hamilton *et al.*, 1974. Poultry Sci. 53:871-877.

Table 15. Effects of addition of 55 ppm chlortetracycline to diets containing 10 ppm aflatoxin on performance of broiler chickens at 21 d

Treatment	Aflatoxin (ppm)	Body weight (g)	Feed conversion (g:g)	Mortality
Control	0	363	1.73	0/50
+ chlortetracycline	0	360	1.75	0/50
Control	10	195	2.23	12/50
+ chlortetracycline	10	211*	1.98*	6/50

*Significant improvement over respective control group.
Smith *et al.*, 1971. Poultry Sci. 50:768-774.