Interaction and effects of mycotoxins in poultry

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Mycotoxins are a group of structurally diverse secondary metabolites of fungi that occur as contaminants of grains worldwide. Aspergillus, alternaria, claviceps, fusarium and penicillium species of fungi are ubiquitous in nature and under ideal conditions often infect economically important crops and forages in the field, during storage, shipment and processing.

Many of the secondary metabolites produced by these fungi can cause serious health problems in poultry and their presence in agricultural commodities may result in serious economic losses.

Mycotoxins	Toxic effect reported	Reference
Aflatoxin and ochratoxin A	Synergistic	Huff and Doerr, 1981
Aflatoxin and T- 2 toxin	Synergistic	Huff et al.1988
Aflatoxin and DAS	Synergistic	Kubena et al. 1993
Aflatoxin and kojic acid	Antagonistic	Giroir et al. 1991
Aflatoxin and fumonisin	Additive	Kubena et al. 1995
Aflatoxin and moniliformin	Additive or less	Kubena et al. 1997
Fumonisin and monilformin	No synergistic	Li et al. 2000
Fumonisin and DAS or ochratoxin	Additive or less	Kubena et al. 1997
Fumonisin and T-2 toxin	Additive	Kubena et al. 1995
Fumonisin and T-2 toxin or DON	Additive	Kubena et al. 1997
DON and T-2 toxin	Additive	Kubena et al., 1989
Citrinin and ochratoxin A	Antagonistic	Manning et al. 1985
Ochratoxin and T-2 toxin	Additive	Wang et al, 2009
Moniformin and DON	No effect by DON alone	Morris et al. 1999
Cyclopiazonic acid and ochratoxin A	Additive	Gentles et al. 1999

Table 1. Some mycotoxin combinations published in poultry.

Interaction of mycotoxins

Over 300 mycotoxins have been reported in the literature and the target sites and mechanism of toxicity are varied for each one, as are their chemical structures.

More than a single mycotoxin may simultaneously contaminate feed ingredients and finished feed. The toxicity and clinical signs observed in poultry when more than one mycotoxin is present in feed are complex and diverse.

The interaction of mycotoxins in poultry feeds is probably intended to answer a complex question of the safe levels when comparing the toxicity of individual versus multiple mycotoxin contamination as well as the interaction of mycotoxins with other factors including feed ingredients, infectious diseases, environmental conditions, heat or cold stress, ammonia, heavy metals, etc., just to mention few of the many possibilities that could interact under field conditions.

There are many ways that feed ingredients and finished feeds can become contaminated with multiple mycotoxins: by a single fungus that produces more than one mycotoxin in a given feed ingredient; by two separate ingredients containing different mycotoxins being used in the manufacturing of feed; or by contamination of a single ingredient with two separate fungi that produce different mycotoxin.

When mycotoxins are present simultaneously, interactive effects can be classified as additive, antagonistic or synergistic (Table 1).

The field diagnosis of these mycotoxin interactions are difficult and this emphasises the importance of fully characterising them so that they can be recognised as they occur in the animal industry.

It appears that the level of a particular mycotoxin in the field that produces a certain effect can be much lower than the level that produces the same effect in the laboratory. Obviously, this difference can be explained by the complexity of environmental, social and other stressors that exist in the field compared to the carefully controlled laboratory experiment.

This difference in minimal effective dosage may be due, to some extent, to the complexity of the total and multiple mycotoxin exposure of the animal versus any single mycotoxin.

Table 2. Individual and combined effects of dietary aflatoxin and ochratoxin A on body weight and liver lipid of three week old broiler chickens (Adapted from Huff and Doerr, 1981).

Aflatoxin (µg/g)	Ochratoxin A (µg/g)	Body weight (g)*	Liver lipid (% dry weight)*
0	0	625.8[a]	16.49[a]
0	2.0	549.6[b]	14.59[a]
2.5	0	551.1[b]	24.48[b]
2.5	2.0	380.4[c]	17.25[a]

*Values represent the mean of six groups of 10 birds each. Values without a common superscript differ (P<0.05)

Aflatoxin/ochratoxin A

Aflatoxin is a potent hepatotoxin in young broilers characterised by enlarged, pale, friable, and fatty livers. Ochratoxin A is primarily a nephrotoxin in broiler chickens.

When these two mycotoxins were fed simultaneously to broiler chicks, synergistic toxicity resulted (Table 2). The effects observed in the combination treatment were greater than could have been produced by individual mycotoxins. However, the kidneys appeared to be the target organ of this mycotoxin combination (Table 3).

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Aflatoxin (µg/g)	Ochratoxin A (µg/g)	Liver (g/100g)*	Kidney (g/100g)*
0	0	2.82[a]	0.56[a]
0	2.0	3.20[b]	0.76[b]
2.5	0	3.50[c]	0.75[b]
2.5	2.0	4.11[d]	1.12[c]

*Values represent the mean of six groups of 10 birds each
Values within a column without a common superscript differ (P<0.05)

Table 3. Individual and combined effects of dietary aflatoxin and ochratoxin A on relative weight of the liver and kidney of three week old male broiler chickens (Adapted from Huff and Doerr, 1981).

Aflatoxin (µg/g)	Ochratoxin A (µg/g)	Liver lipid	
		Full-term (% dry weight)*	0 to 3 weeks (% dry weight)**
0	0	16.16[a]	16.00[a]
0	2.0	15.22[a]	15.10[a]
2.5	0	24.32[c]	15.37[a]
2.5	2.0	21.23[b]	15.12[a]

*Values represent the mean of three replicates of 25 broilers per replicate
Values within a column without a common superscript differ (P<0.5)

Table 4. Individual and combined effects of aflatoxin and ochratoxin A on liver lipid of six week old broiler chickens fed only during the first three weeks of their lives or fed continuously full term of six weeks (Adapted from Huff et al, 1983).

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Furthermore, the increase in liver lipid, normally a presumptive diagnostic lesion of aflatoxicosis, was spared when ochratoxin A was present (Table 2). This interaction between aflatoxin and ochratoxin A demonstrated the complexity of the effects that can occur during mycotoxin interactions through synergistic toxicity and altered clinical signs. Ochratoxin A's lesions persisted longer than the aflatoxin effect.

The seemingly longer effects of ochratoxin A compared to the aflatoxin may reflect the difference in regenerative capacity of the liver opposed to the kidney, since ochratoxin is primarily a nephrotoxin and aflatoxin is primarily a hepatotoxin.Aflatoxin, ochratoxin A, and the combination appear to increase the incidence and severity of bloody thighs. Furthermore, an increased susceptibility to bruising persisted at least three weeks after broilers were taken off mycotoxin contaminated feed.

Citrinin/ochratoxin A

Citrinin, like ochratoxin A, is a nephrotoxin in poultry. When citrinin and ochratoxin A were fed simultaneously to young broiler chicks, the resulting interaction can be best described as antagonistic (Table 5).

Table 5. The individual and combined effects of dietary ochratoxin A and citrinin on body weight of three week old broiler chickens (Adapted from Manning et al, 1985).

Ochratoxin A (µg/g)	Citrinin (µg/g)	Body weight (g)	Water consumption *(ml/bird)*
0	0	635[a]	831[a]
2.0	0	548[b]	770[a]
0	400	561[b]	2289[b]
2.0	400	516[c]	1181[c]

*Values represent the mean of six replicates of eight broilers per replicate. Values without a common superscript differ (P<0.05)

Citrinin causes a dramatic increase in water consumption and excretion, a useful diagnostic index. However, when ochratoxin A is also present in the feed, this effect of citrinin is reduced (Table 5).

Therefore, a specific and important diagnostic index of citrinin mycotoxicosis is considerably altered when ochratoxin A is a co-contaminant of feed.

Diseases in poultry

Aflatoxin has demonstrated immunosuppressive properties for poultry. Affected animals have increased susceptibility to some infectious diseases. The combination of aflatoxin and Eimeria tenella resulted in a synergistic effect on mortality. In addition, with E. acervulina and E. tenella, weight gain and body weight were reduced significantly more when coccidia and dietary aflatoxin were present together. Even mortality, which is rare with E. acervulina, was markedly increased by dietary aflatoxin. Either aflatoxin or E. acervulina alone reduced the plasma pigment.

When both were present, despigmentation was greater than with each alone. ■