# Comprehensive strategy to fight mycotoxins in poultry

ycotoxins are well known for exerting adverse effects in animals and humans. This field of research is inherently complicated, and very technical. Accordingly, this article aims to provide key points to better understand the effects of mycotoxins, especially in poultry.

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Before talking about mycotoxins, it is important to remember that they are present at extremely low concentrations. Whereas the concentration of proteins is expressed as a percentage (0.01), and that of vitamins in parts per million (ppm, 0.000001), mycotoxin concentrations are expressed in parts per billion (ppb, 0.00000001).

A creative way to better understand the extremely low levels at which mycotoxins are present, would be to think of it as one second out of the approximately 1 billion seconds that make up 32 years! Despite being present at such low concentrations, they can nevertheless trigger toxic effects in animals.

The modes of action through which toxicity is exerted by such molecules in animals, can differ depending on the type of mycotoxin. It has been scientifically proven that mycotoxins can act on several

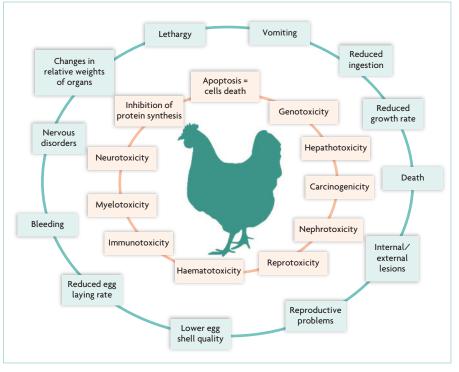


Fig. 1. Modes of action through which mycotoxins exert adverse effects, and the resulting impact on animals.

organs, including the liver, kidney, brain, and reproductive organs, and furthermore they may have an effect on various cellular functions by triggering apoptosis, inhibiting protein synthesis, or stimulating myolosis. In addition they can target various cellular structures, such as the DNA, and blood cells.

Currently, these adverse effects are still not well diagnosed. The resulting symptoms in farm animals are usually non-specific, such as vomiting, a decrease in feed intake and growth, reproductive problems, lethargy, and in extreme cases, death (Fig. 1).

Of course, some mycotoxins have been studied quite well, and the more well known mycotoxins include aflatoxin B1, deoxynivalenol (DON), zearalenone, ochratoxin A and fumonisin B1.

Moreover, it is possible to find information in the scientific literature on more than 40 types of mycotoxins and metabolites (see Table 1).

Although not all areas around the world are affected by mycotoxin contamination, it is now acknowledged that a contamination profile can be estimated by evaluating areas where the raw vegetal materials susceptible *Continued on page 33* 

## Table 1. Data available on the types of mycotoxins and metabolites found in poultry production.

Mycotoxin families	Molecules
Aflatoxin	B1, B2, G1, G2, M1
Zearalenone	Zearalenone, $\alpha$ -Zearalenone, $\beta$ -Zearalenone
Trichothecene type A	T-2, HT-2, 30H-HT2, T-2 tetraol, Neosolaniol (NEO), 8-acetyl-NEO, TAS, MAS, 3-MAS, 4-MAS, 15-MAS, DAS, 3,4-DAS, 3,15-DAS, Scirpentriol
Trichothecene type B	DON, DOM-1, Nivalenol, Fusarenon X, 15-acetyl-DON, 3-acetyl-DON
Ochratoxin	Α, Β, C, α
Fumonisin	B1, B2, B3
Others	Cyclopiazonic acid, citrinin, ergot alkaloids, monoliformin



Fig. 2. Global mycotoxin contamination profile.

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to contamination are grown (Fig. 2). In this manner, regions with potential contamination can be identified and appropriately managed. Furthermore, in cases requiring imported raw materials, the mycotoxin profile of the exporting country can be taken into account.

Once the mycotoxin profile has been identified, the precise toxic effects still remains difficult to predict. In fact, even if the modes of action of different molecules belonging to the same mycotoxin family are quite similar, the resulting toxicity they cause may be very different. This can be illustrated with the lethal dose 50 values (LD50, the dose lethal to 50% of the animals) for some trichothecenes, as presented in Table 2.

With a simple ratio calculation, it is easy to demonstrate that the T-2 toxin is 36 times more toxic than DON in broilers. It is even worse for the monoacetoxyscirpenol (MAS) toxin, which seems to be 56 times more toxic compared to DON. Therefore, when evaluating mycotoxin contamination, predicting their harmful effects on animals is made easier upon a full analysis.

However, once a thorough analysis has been performed, and the mycotoxin concentrations have been measured, it is still difficult to predict the effects on animals, because this depends on the animal species and age.

The difference in toxicity between various species can be highlighted with the example of aflatoxin B1. This mycotoxin has

an estimated LD50 of 6.65 parts per million (ppm) in broilers, while in ducks it is only 0.46ppm, showing that ducks are about 15 times more sensitive to aflatoxin B1 than broilers. Furthermore, the influence of age on the expression of mycotoxin toxicity is best illustrated with the example of ochratoxin A.

Chang et al. (1981) have shown that 21-day old turkeys are almost two times less sensitive to ochratoxin A, compared to one-day old turkeys. Huff et al. (1974) demonstrated a similar difference in sensitivity to ochratoxin A toxicity between 21 day-old and one-day old broilers.

In addition, if we include factors relating to the animal's immune status before being exposed to contaminated feed, and the synergistic or additional effects of various mycotoxins, it becomes much more complicated to understand and analyse mycotoxin contamination, and the consequent effects in animals.

Using an ordinary toxin binder is not enough to manage this major risk, and studies have shown the importance of employing a comprehensive mycotoxin management program, including a combination of diagnostic services to create a thorough contamination profile, and customising an action plan that is tailor-made for customers, to support the health and profitability of their farms.

References are available from the author on request

## Table 2. LD 50 for some trichothecenes for broilers.

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