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Fight mycotoxins in poultry with comprehensive strategy

The toxic effects of mycotoxin contamination are difficult to predict in animals. Employing a comprehensive mycotoxin management program will support farm health and profitability.

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MYCOTOXINS 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.

Before talking about mycotoxins, it is important to remember that they are present in extremely low concentrations. Whereas the concentration of proteins is expressed as a percentage (0.01) and vitamins in parts per million (0.000001), mycotoxin concentrations are expressed in parts per billion (0.000000001).

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 exist in a time span of 32 years.

Despite being present at such low concentrations, mycotoxins 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 proved that mycotoxins can act on several organs, including the liver, kidneys, 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 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, problems with reproduction, lethargy and, in extreme cases, death (Figure).

Of course, some mycotoxins have been studied quite extensively, 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 (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 plant materials susceptible to contamination are grown (Map).

In this manner, regions with potential contamination can be identified and appropriately managed. Furthermore, in cas-

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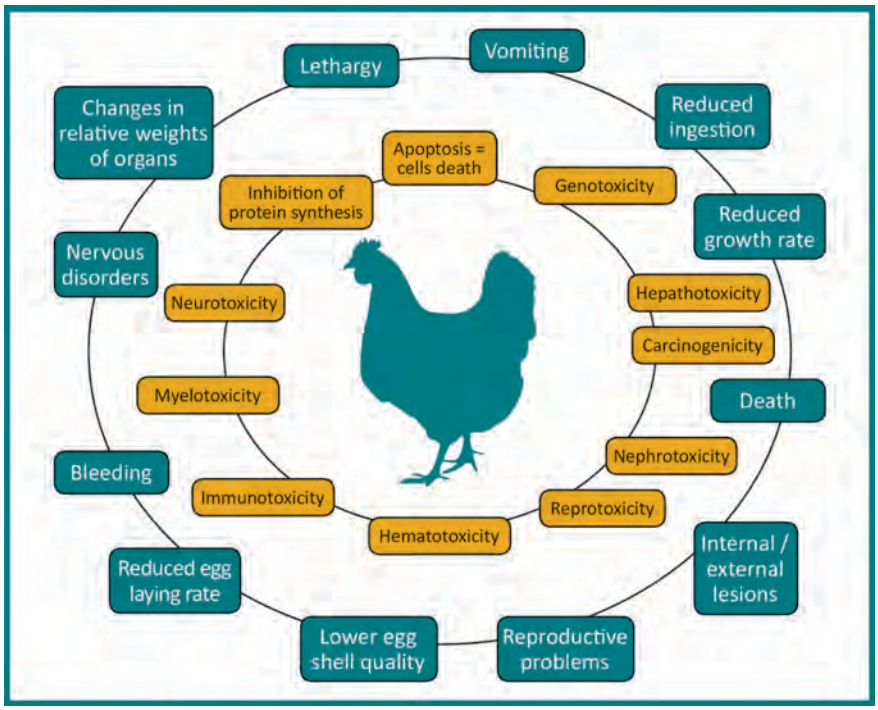

1. Data available on the types of mycotoxins and metabolites found in poultry production

Mycotoxin family	Molecules
Aflatoxin	B1, B2, G1, G2, M1
Zearalenone	Zearalenone, alpha-zearlenone, beta-zearalenone
Trichothecene type A	T-2, HT-2, 3OH-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
Ochratoxin	A, B, C, alpha
Fumonisin	B1, B2, B3
Others	Cyclopiazonic acid, Citrinin, Ergot alkaloids, monliformin

2. LD50 of some trichothecenes for broilers

Mycotoxins	LD50 (ppm)	Authors
DON	140.0	Huff et al., 1981
T-2 toxin	3.9	Chi et al., 1978a; Who, 1990; Sato and Ueno, 1977
MAS	2.5	Richardson, 1990
NEO	24.9	Chi et al., 1978a

es requiring imported raw materials, the mycotoxin profile of the exporting country can be taken into account.

Once the mycotoxin profile has been identified, however, the precise toxic effects still remain 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 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 T-2 is 36 times more toxic than DON in broilers (*Gallus gallus*). Even worse is monoacetoxyscirpenol (MAS) toxin, which seems to be 56 times more toxic than DON.

Therefore, when evaluating mycotoxin contamination, predicting the harmful effects on animals can be made easier with 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 species and age of the animals.

The difference in toxicity between various species can be highlighted by using the example of aflatoxin B1. This mycotoxin has an estimated LD50 of 6.65 ppm in broilers (Smith and Hamilton, 1970; Patterson, 1973), while it is only 0.46 ppm in ducks (Patterson, 1973; Galtier et al., 2005), 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) showed that 21-day-old turkeys are almost two times less sensitive to ochratoxin A than 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 factors are included relating to the animal's immune status before being exposed to contaminated feed as well as the synergistic or additional effects among mycotoxins, it becomes much more complicated to understand and analyze 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 customizing an action plan that is tailor-made for customers — to support the health and profitability of their farms. ■