Script

Mycotoxins in poultry

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1. Introduction

The poultry industry in India represents a major success story. What was largely a backyard venture before the 1960s has been transformed into a vibrant agribusiness with an annual turnover of Rs 30 000 crores. Today, India is the third largest egg producer in the world (after China and the United States of America), and the nineteenth largest broiler producer. Feed is the most important component in poultry production and represents up to 50% of total costs.

World compound feed production is approaching 1 billion tonnes per year according to the International Feed Industry Federation (IFIF). The poultry feed industry comprises 45% of the total market. Asia is the largest feed producing region and covers 38% of the world feed market. Feed is the single largest cost item in poultry production, accounting for 55-64% of variable costs in India, depending on the region. According to industry sources, domestically produced corn (energy) and soybean meal (protein) are the dominant feed ingredients in broiler rations. Nearly all of India's feed demand is met from indigenously produced feeds. The continued growth of poultry production, however, could eventually outstrip gains in feed production, particularly if poultry output continues to expand at its current rapid rate.

The latest Mycotoxin Survey 2013 focused on the main feed ingredients for poultry (corn, wheat, soybean, rice etc) analysed between January and September 2013 and the data showed that mycotoxin levels were above the detection limits in 73% of all samples tested. In view of this, procuring high quality feed is an indispensable but challenging requisite due to the unavoidable contamination of grains with mycotoxins.

The word mycotoxin stems from the Greek word "mykes", means mould, and "toxicum" means poison. Mycotoxins are toxic secondary metabolites produced by fungi growing on crops in the field, during handling and in storage. They enter the animal production system via feed (concentrate, silage or forage) or via bedding.

A mycotoxicosis is a problem caused by a natural mycotoxin produced by a fungus. In poultry, this usually results when toxin-producing fungi grow in grain and feed. Hundreds of mycotoxins have been identified, and many are pathogenic. Mycotoxins may have additive or synergistic effects with other natural toxins, infectious agents, and nutritional deficiencies. Many are chemically stable and maintain toxicity over time. Economic losses associated with mycotoxicosis include:

- Poor growth
- Reduced egg production

- Reduced feed conversion
- Increased mortality
- Poor egg shell quality
- Reduced fertility
- Leg problems
- Carcass condemnation
- Increased susceptibility to disease

All poultry species are affected by mycotoxins. However, species differences have been reported. Ducks for example are particularly sensitive towards aflatoxin.

In poultry production, feed is the key vector bringing mycotoxins into the production system and control strategies should mainly focus on optimising feed quality. However, cases have been reported where significant concentrations of mycotoxins have been introduced into the production systems through litter. Straw may already be contaminated with significant concentrations of mycotoxins at the time of harvest, although any type of litter can be contaminated during unfavourable storage conditions.

In order to effectively recognize mycotoxicosis, flocks have to be carefully inspected for symptoms. Careful recognition of symptoms and post-mortem analyses combined with adequate feed analyses, provide the most accurate mean of a mycotoxicosis diagnosis.

2. Types of mycotoxins in poultry

Aflatoxin

Aflatoxin is a mycotoxin produced by the fungus *Aspergillus* mainly *Aspergillus flavus*, *A. parasiticus* etc. Aflatoxins are of concern in warm and humid climatic conditions. Although aflatoxins are not considered to be a major problem in cold or more temperate regions, caution must be exercised in colder climates when using feedstuffs imported from warm and humid countries.

Among poultry, ducks are the most susceptible to aflatoxin, followed by turkeys, broilers, laying hens and quail. In all species, aflatoxins are hepathotoxic with fatty changes, causing hepathocyte degeneration, necrosis, and altered liver function. Suppression of hepatic protein synthesis is the main factor resulting in growth suppression and reduced egg production. Aflatoxin is also known to interfere with vitamin D metabolism, contributing to reduced bone strength and leg weakness. By reducing bile salt production, aflatoxin negatively affects lipid and pigment absorption. Additionally the metabolism of other minerals including iron, phosphorus and copper are also affected by aflatoxin. Aflatoxin increases the fragility of capillaries, reducing prothrombin levels thereby drastically increasing the incidence of bruising in carcasses and carcass downgrading. Due to the transfer of aflatoxin into edible products and its carcinogenic effects, most countries have set upper legal limits for aflatoxin in feed. See the regulations page on this web site for more information

Clinical signs of aflatoxin toxicity include:

- Decreased weight gain / anorexia
- Decreased egg production

- Reduce feed conversion efficiency
- Increased mortality
- Immune suppression and increased disease susceptibility
- Reduced fertility and hatchability
- Embryo toxicity
- Specific visceral haemorrhage
- Increased susceptibility to environmental and microbial stressors
- Leg weakness and reduced bone strength
- 'Pale bird syndrome'
- Fatty liver
- Liver necrosis
- Bile duct hyperplasia
- Increased incidence of bruising and downgrading

Ochratoxin

Ochratoxins are important storage toxins. They are produced by different fungi mainly *Aspergillus ochraceus* etc. and are prevalent in temperate as well as in tropical regions.

Ochratoxin A is the most important of the ochratoxins. The primary effect of ochratoxin A in all poultry species is nephrotoxicity. In poultry the proximal tubules are mainly affected and the kidney is pale and grossly enlarged. As with aflatoxin, fatty liver can also occur due to ochratoxin exposure. In acute cases mortalities can occur due to acute renal failure. In young chicks, ochratoxin A is approximately three times more toxic than aflatoxin.

Ochratoxin has been implicated in significant field outbreaks of mycotoxicosis in poultry.

Clinical signs of ochratoxin toxicity include:

- Reduced feed intake
- Reduced growth rate and egg production
- Reduced feed conversion efficiency
- Mortality due to acute renal failure
- Poor egg shell quality and higher incidence of eggs with blood spots
- Reduced embryo viability and decreased hatchability
- Reduced feathering
- Polyurea with large volumes of wet faeces
- Pale and grossly enlarged kidney
- Fatty liver
- Urate deposition in joints and abdominal cavity (at high exposure levels)
- Depletion of lymphocytes and with it strong suppression of cellular immunity, thus enhanced susceptibility to viral infections.

Trichothecenes (T-2 toxin, deoxynivalenol (DON), HT-2 toxin, etc)

Trichothecenes are a group of mycotoxins produced by the different species of *Fusarium*. Trichothecenes are typical field mycotoxins and are produced on crops entering the feed via contaminated ingredients. Trichothecenes are proven tissue irritants with the major

observation associated with their ingestion being oral lesions, dermatitis and intestinal irritation.

The major physiological response to trichothecenes mycotoxins is loss of appetite, thus earning them the name, feed refusal toxin.

Of the different trichothecenes, poultry are most sensitive to T-2 toxin and DAS. Trichothecenes are strong immune suppressive mycotoxins affecting cellular immune response by direct effects on bone marrow, spleen, lymphoid tissues, thymus and intestinal mucosa, where actively dividing cells are damaged.

Clinical signs of trichothecenes toxicity include:

- Oral lesions: circumscribed proliferate yellow caseous plaques occurring at the margin of the beak, mucosa of the hard palate and the angle between the mouth and the tongue.
- Reduced feed intake
- Reduced weight gain and egg production
- Poor shell quality
- Reduced female fertility and hatchability of fertile eggs
- Immune suppression, reduced vaccination response
- Tibia dyschondroplasia
- Gizzard erosion
- Necrosis of proventricular mucosa
- Regression of ovaries
- Increased liver weight

Zearalenone (ZEA)

Zearalenone mainly produced by the fungus *Fusarium*, often occurs with DON in naturally-contaminated cereals. Zearalenone is responsible for reproductive disorders due to its oestrogenic effect at high concentrations. However, in general ZEA has limited toxicity to birds. At high concentrations the following symptoms have been observed:

- Vent enlargement
- Enhanced secondary sex characteristics

Fumonisin

Fumonisin is a mycotoxin produced by the fungus *Fusarium* mainly *Fusarium verticillioides*, *F. proliferatum* etc. Broilers and turkeys seem relatively resistant to acute toxic effects of fumonisins. Fumonisin mycotoxicosis leads to a very specific increase in sphiganine:sphingosine ratio. However, as sphinganine and sphingosine analyses are quite complex this ratio is seldom used as a biomarker in field situations.

Clinical signs of fumonisin toxicity include:

- Spiking mortality (paralysis, extended legs and neck, wobbly gait, gasping)
- Reduced growth rate
- Increased organ weights

- Hepatocellular hyperplasia
- Poor vaccination response
- Increased liver sphinganine : sphingosine ration (biomarker)

Co-contamination and further mycotoxins

Contaminated feeds or ingredients typically contain more than one known and probably several unknown mycotoxins. The toxic responses and clinical signs observed in poultry when more than one mycotoxin is present in feed are complex and diverse.

Co-contamination of mycotoxins appears to exert greater negative effects on health and productivity than do single mycotoxins. For this reason, symptoms typical of mycotoxicosis are often seen in poultry despite analyses of the feed indicating only very low or zero concentrations of individual mycotoxins. Toxicity may be due to interactions between different mycotoxins that exaggerate the toxicity symptoms.

With mycotoxins the risk directly depends on the level of the major mycotoxins in the feed, the co-occurrence and level of other mycotoxins as well as the avian species, their age and health status. Therefore strictly speaking it is not possible to define safe levels of mycotoxins. This complex situation makes it critical to take the necessary precautions.

3. Sampling and testing for mycotoxins

Mycotoxicosis should be suspected when the history, signs, and lesions are suggestive of feed intoxication, and especially when moldy ingredients or feed are evident. Toxin exposure associated with consumption of a new batch of feed may result in subclinical or transient disease. Chronic or intermittent exposure can occur in regions where grain and feed ingredients are of poor quality or when feed storage is substandard or prolonged. Impaired production can be a clue to a mycotoxin problem, as can improvement because of correction of feed management deficiencies.

Definitive diagnosis involves detection and quantitation of the specific toxin(s). This can be difficult because of the rapid and high-volume use of feed and ingredients in poultry operations. Diagnostic laboratories differ in their respective capabilities to test for mycotoxins and should be contacted before sending samples. Feed and also birds that are sick or recently dead should be submitted for testing.

If clinical signs of mycotoxicosis are observed, it is important to properly collect a grain or feed sample and send it to a laboratory to determine the presence and level of the suspected mycotoxin(s). Sampling accounts for 80 - 90% of the error associated with measuring mycotoxins in grain or feed. Analytical tests for mould spore counts are of little or no value.

Random samples (10 to 30) should be collected from several locations within a batch of grain or feed and combined thoroughly to provide a composite sample for submission. Using a grain probe at several evenly distributed locations will provide the most representative sample.

Samples can also be collected periodically from grain being augured which can also be an effective form of sampling. Paper bags should be used to transport sample(s), since plastic

bags retain moisture, and therefore can promote additional fungal growth. Contact the laboratory for specific sampling requirements prior to submission.

Samples should be collected at sites of ingredient storage, feed manufacture and transport, feed bins, and feeders. Fungal activity increases as feed is moved from the feed mill to the feeder pans. Samples of 500 g (1 lb) should be collected and submitted in separate containers. Clean paper bags, properly labeled, are adequate. Sealed plastic or glass containers are appropriate only for short-term storage and transport, because feed and grain rapidly deteriorate in airtight containers.

It is important to remember that based on the uncertainties associates with any mycotoxin test procedure, it is difficult to determine the true concentration of a bulk lot.

Mycotoxins are difficult to measure for a number of reasons:

- Many different mycotoxins can be present simultaneously, making analysis difficult and expensive. Under commercial conditions analyses is normally limited to a couple of indicator mycotoxins.
- Sampling of bulk feeds is difficult. Mycotoxins are present in 'hot' spots and are not evenly distributed throughout the feed. Therefore strict sampling procedures should be followed with many samples taken from a particular batch to get a realistic reading.
- Latest research has identified complexes of mycotoxins and their metabolites for which there is no accurate analysis method.

4. Feeding Strategies

As the risk if mycotoxicosis is very difficult to predict or evaluate, prevention strategies should be initiated when assessing even a low risk situation. Prevention strategies must primarily aim at minimising mycotoxin formation in the field and during storage.

A significant reduction in mycotoxin formation can be achieved by good agronomic practices, for example:

- Selection of crop varieties that are more resistant to fungal foliar diseases
- Ploughing up harvest residues
- Avoiding no-till soil management practices
- Proper crop rotation
- Avoiding monoculture

During storage of dry feed ingredients, mycotoxin formation can be successfully controlled by monitoring the moisture content of the feed. If the moisture content is below 12%, moulds become metabolically inactive, and the risk of mycotoxin formation is strongly reduced. To avoid mycotoxin formation, be aware of the following:

- Moisture content below 12%
- Relative humidity below 60%
- Storage temperature below 20 °C
- Clear grain, avoid broken kernels
- Control insects and rodents

- Avoid stress (frost, heat, pH changes)

The incorporation of technical mould inhibitors such as Moldzap further enhances stability of feed and ingredients during storage.

5. Mycotoxin adsorbents and binders

As we know mycotoxins are usually found in combinations in complete animal feeds. A broad substrate binding capacity will ensure at least some fraction of all the mycotoxins will be rendered non-bioavailable and the bioavailable mycotoxins will be below the threshold of biological activity. Broad substrate binding capacity of a binding agent will also minimise the potential for toxicological synergy between mycotoxins.

Speciality feed additives, known as mycotoxin adsorbents or binding agents are the most common approach to prevent and treat mycotoxicosis in animals. It is believed that the agents bind to the mycotoxin preventing them from being absorbed. The mycotoxins and the binding agent are excreted in the manure.

The effective level of dietary inclusion for mycotoxin adsorbents will depend on the mycotoxin binding capacity of the adsorbent and the degree of contamination of the feed in question. A high binding capacity will minimise the level of inclusion and minimise the reduction in nutrient density caused by the feeding of the adsorbent. High levels of inclusion of adsorbents can also alter the physical properties of the feed which might impair feed processing such as pellet formation, in addition to altering the actual diet specification.

Mycotoxin binding is achieved through both:

- Physical adsorption: Relatively weak bonding involving van der Waals interactions and hydrogen bonding
- Chemical Adsorption: (Chemisorption) is a stronger interaction which involves ionic or covalent bonding.

An effective binder or sequestering agent is one that prevents or limits mycotoxin absorption from the gastro-intestinal tract of the animal. In addition, they should be free from impurities and odours. Be aware that not all are equally effective. Many can impair nutrient utilisation and are mainly marketed, based on *in-vitro* data only.

There are two types of mycotoxin adsorbent/binder:

- Inorganic binders
- Organic adsorbents

Inorganic binders: Inorganic mycotoxin binders are silica based polymers. Examples could include:

- Zeolites
- Bentonites
- Bleaching clays from the refining of canola oil
- Hydrated sodium calcium aluminosilicates (HSCAS)

- Diatomaceous earth
- Numerous clays

They can be grouped into two categories: Phyllosilicates and Tectosilicates:

Phyllosilicates: *bentonites/montmorillonites*

- Phyllosilicates are characterised by alternating layers of tetrahedral silicon and octahedral aluminium coordinated with montmorillonite oxygen atoms
- Isomorphous substitution leads to a net negative charge which must be satisfied by the presence of inorganic cations (Na, Ca, Mg, K)
- Applications: Adsorbents for heavy metals, suspension-stabilising agents in coatings, bonding agents for foundry sands and washes, binder in pelletisation processes, desiccants in feed products.

Tectosilicates: *zeolites*

- Tectoalumosilicates of alkali and alkaline earth cations that have an infinite three-dimensional cage-like structure
- Isomorphous substitution leads to a net negative charge which is satisfied by the presence of inorganic cations (Na, Ca, Mg, K)
- Applications: Adsorbents for ammonia, heavy metals, radioactive cesium and mycotoxins.

Such materials are often inexpensive and easy to handle. These products are traditionally mixed with compound feed at a mill or mixed on farm for home mixers. Costs are cheap but require a high inclusion rate in animals.

Organic Adsorbents: Organic mycotoxin adsorbents are carbon based polymers. Examples could include:

Fibrous plant sources such as: Oat hulls, wheat bran, alfalfa fibre, extracts of yeast cell wall, cellulose, hemi-cellulose, pectin.

Such materials are biodegradable but can, in some cases, also be vectors of mycotoxin contamination. Benefits of yeast cell wall are low inclusion, high surface area and certainly no toxic contaminants.

Mycotoxin adsorbents offer an attractive short-term solution to the challenge of mycotoxin-contaminated animal feeds. The only complete solution to the mycotoxin challenge will be the long-term goal of eliminating mycotoxins from the food and feed chains through improved quality control based on better analytical techniques coupled with genetic advances in plant resistance to fungal infestation.

Conclusion: It is clear that the presence of mycotoxins in poultry feed can result in significant economic losses for the poultry producer. The world demand for poultry keeps rising despite high feed consumption and production prices. Demands for quality poultry are directly linked to demands for quality feed. As the effects of mycotoxins in poultry are often subclinical, they are frequently overlooked by farm technicians. Mycotoxin risk management is crucial to eliminate the effect of fungal toxins and their toxicity and should be carried out

on a continuous basis through a proper mycotoxin risk management tool which uses different strategies.