Review Article

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Aspects on the Influences of Mycotoxicosis in Domestic Birds: Review

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Abstract

Mycotoxicosis is a significant concern in the poultry industry worldwide, as it is caused by the ingestion of mycotoxins, toxic secondary metabolites produced by fungi that commonly contaminate animal feed. This review aims to explore various aspects of mycotoxicosis and its influences on domestic birds. It discusses the major mycotoxins affecting poultry, including aflatoxins, ochratoxins, trichothecenes, zearalenone, and fumonisins, and their sources of contamination, such as pre-harvest, post-harvest, and storage factors. In addition, the review highlights the impact of mycotoxicosis on avian health, including immune system suppression, gastrointestinal disturbances, reduced growth and development, and reproductive disorders, as well as its effects on egg quality and hatchability. Furthermore, it presents potential preventive and management strategies, such as implementing good agricultural practices, feed management and quality control, the use of mycotoxin binders and adsorbents, feed additives and supplementation, and veterinary interventions. The review emphasizes the importance of regulatory guidelines and monitoring programs to ensure food safety and mitigate mycotoxin-related issues. Overall, understanding the influences of mycotoxicosis in domestic birds is vital for implementing effective measures to prevent mycotoxin contamination and promote the health and productivity of poultry.

Keywords: Domestic birds, mycotoxins, contamination, health.

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Introduction

Mycotoxicosis, the toxic condition resulting from the ingestion of mycotoxins, poses a significant threat to the health and productivity of domestic birds in the poultry industry. Mycotoxins are secondary metabolites produced by various molds that contaminate feed ingredients. These toxic compounds have the potential to cause a range of adverse effects, including immunosuppression, impaired growth and development, reproductive issues, and altered nutrient metabolism in birds (1).

The prevalence of mycotoxin contamination in poultry feed is a global concern, as it can lead to substantial economic losses and compromises the welfare of domestic birds. The adverse effects of mycotoxicosis not only impact bird health but also diminish production efficiency, quality of meat and eggs, and overall profitability of the poultry industry. Therefore, understanding the influences of mycotoxicosis on domestic birds and implementing effective prevention and control strategies are paramount (2).

This review aims to provide an overview of the influences of mycotoxicosis in domestic birds, encompassing the sources of mycotoxin contamination, common types of mycotoxins, routes of exposure, and the adverse effects on avian health. Furthermore, it explores the preventive, control, and mitigation strategies for managing poultry feed mycotoxin contamination. Poultry producers and researchers can make informed decisions to safeguard bird welfare and ensure optimal production outcomes by addressing these aspects.

Sources of Mycotoxin Contamination:

Mycotoxin contamination in poultry feed can originate from various sources throughout the production and storage chain. Some common sources of mycotoxin contamination include:

Field Contamination: Fungi, such as Aspergillus, Fusarium, and Penicillium, can infect crops in the field, particularly during periods of high humidity and temperature. Infected crops such as grains, oilseeds, and forages serve as a primary source of mycotoxin contamination (3).

Storage Conditions: Improper storage conditions, including high moisture content, inadequate ventilation, and temperature fluctuations, can promote fungal growth and mycotoxin production in stored grains and feedstuffs (4).

Processing and Handling: Contamination can occur during the processing and handling of feed ingredients including transportation, milling, and mixing. Equipment, storage bins, and conveyance systems that are not properly cleaned and maintained can contribute to mycotoxin contamination (4).

Feed Additives: Some feed additives such as certain mold inhibitors or contaminated ingredients used in their production, may contain mycotoxins, further increasing the risk of contamination (5).

Environmental Factors: Environmental conditions, such as climatic variations and natural disasters, can impact fungal growth and mycotoxin production in crops, potentially leading to higher levels of contamination (6).

2-**Common Types of Mycotoxins:**

There are numerous types of mycotoxins that can affect domestic birds. Some of the most common mycotoxins include:

Aflatoxins: Aflatoxins are toxic secondary metabolites primarily produced by Aspergillus fungi, such as A. flavus and A. parasiticus. These compounds, characterized by furanocoumarins derived from polyketides, can naturally contaminate animal feeds. Feeds may contain Aflatoxin B1 (AFB1), Aflatoxin G1 (AFG1), Aflatoxin B2 (AFB2), and Aflatoxin G2 (AFG2). When animals consume contaminated feed, their biological fluids, including milk and tissues, may contain Aflatoxin M1 (AFM1) and Aflatoxin M2 (AFM2), which are 4-hydroxy metabolites of AFB1 and AFB2, respectively. The presence of AFM1 and AFM2 indicates exposure to aflatoxin-contaminated feed (7). In the early 1960s, AFB1 was identified as the primary causative agent of "Turkey X Disease," which resulted in the death of young turkeys in England due to contaminated peanut-based feed. AFB1 is a widespread dietary hepatotoxin and hepatocarcinogen, raising significant public health concerns worldwide. The toxic effects of AFB1 vary among species and domestic turkeys (Meleagris gallopavo) are known to be highly susceptible. Among the different forms of aflatoxins, AFB1 is the most common and biologically active (8). Aflatoxins are a significant public health concern worldwide, as they are widespread dietary hepatotoxins and hepatocarcinogens. The toxic effects of Aflatoxin B1 (AFB1) vary among different species and domestic turkeys (Meleagris gallopavo) are known to be particularly susceptible. These toxins are commonly found in feed ingredients used for poultry rations. The most prevalent and biologically active forms of aflatoxins are AFB1, AFB2, AFG1, and AFG2, with AFB1 being the most widespread (9). AFB1 is considered a "pro-carcinogen" that undergoes activation through the action of hepatic microsomal cytochrome P450 (CYP450) enzyme. This activation process leads to the formation of the reactive compound AFB1-8,9-epoxide (AFBO), which is essential for the carcinogenic and toxic effects of AFB1. AFBO can form adducts with DNA, specifically AFB-N7-guanine adducts. However, these adducts are unstable and undergo transformation into a compound called for-aminopyrimidine (10).

DNA adducts and the modulation of repair activities are significant indicators of susceptibility to carcinogenesis. AFB-N7-guanine adduct, formed by the binding of AFB1 to guanine in DNA, is a crucial biomarker of AFB1 exposure in both animals and humans. Detection of AFB-N7-guanine adducts in urine plays a vital role in estimating exposure levels and potential risks for individuals who consume AFB1-contaminated substances. By measuring the presence of these adducts, it becomes possible to assess the extent of AFB1 exposure and evaluate the associated health risks (11). This detoxification process is

catalyzed by glutathione S-transferases (GSTs), which are classical detoxification enzymes operating in phase-II metabolism. GSTs play a crucial role in the metabolism of xenobiotics, including chemical carcinogens and environmental contaminants. Mice, for instance, possess the A3 subunit (mGSTA3), which allows them to bioactivate AFB1 and exhibit resistance to its toxic effects due to their efficient catalytic activity for AFBO. It is assumed that mice are AFB1-resistant because of the expression of mGSTA3 and their ability to efficiently metabolize AFBO. The current understanding suggests that the efficiency of GST conjugation is a major determinant that limits the action of AFB1 in individuals and species (12). This efficiency is considered crucial, regardless of the efficiency of AFB1 bioactivation. In other words, the capability of GSTs to effectively detoxify AFB1 by conjugating it with GSH plays a significant role in mitigating the toxic effects of AFB1 (12).

Ochratoxins: Ochratoxins belong to a family of metabolites produced by various fungal species, including Aspergillus and Penicillium. Common producers of ochratoxins include A. ochraceus, A. niger, and P. verrucosum (13). The most prevalent member of this family is ochratoxin A (OTA), followed by its non-chlorinated metabolite ochratoxin B (OTB) and the ethyl ester form ochratoxin C (OTC). OTA is considered the most significant and frequently encountered form, while OTB and OTC are generally regarded as being of lesser importance (14). The International Agency for Research on Cancer (IARC) has classified ochratoxin A as a potentially carcinogenic compound to humans, placing it in Group 2B. Aspergillus species have the ability to produce both OTA and OTB simultaneously. Experiments conducted with A. ochraceus have shown that the production of OTA and OTB is associated with fungal growth, and the yield and ratio of these toxins depend on specific culture conditions. In most cases, the amount of OTB produced is considerably lower than that of OTA, but under certain conditions, the level of OTB production can be comparable to OTA. The observed ratios of OTA to OTB production range from 2:1 to 34:1 (15).

Studies have revealed that the production of ochratoxins, particularly OTA and OTB, is influenced by a complex interplay of various carbon sources, basal media, and nitrogen sources. High levels of OTA production have been associated with the induction of OTA polyketide synthase expression, indicating a connection between gene transcription and OTA synthesis. On the other hand, OTB production does not appear to be linked to the transcription of the polyketide synthase gene.Laboratory fermentation experiments conducted with A. ochraceus have shown that high yields of OTA (up to 10 mg/g) can be achieved, along with the production of OTB and, temporarily, ochracin. The intermediate metabolite OTβ has been found to undergo efficient biotransformation into both OTA (approximately 14%) and OTB (approximately 19%). Conversely, OTα is primarily transformed into OTA (approximately 4.9%). Interestingly, OTB is inadequately converted into OTA, with only a small proportion (approximately 1.5%)

undergoing conversion. Additionally, it is worth noting that some OTB may be produced through the dechlorination of OTA (16).

Fumonisins: Fumonisins (FUM) are a group of mycotoxins that were initially discovered and chemically characterized in 1988 by Gelderblom and colleagues (17). They were first isolated from cultures of Fusarium moniliforme, a fungal species. Six different fumonisins have been identified and their structures elucidated, namely A1, A2, B1, B2, B3, and B4. Among them, fumonisin B1 (FB1) has been reported as the predominant form produced by Fusarium moniliforme (18). The metabolism of fumonisins is still not fully understood. In both the gastrointestinal tract and liver, FB1 undergoes partial hydrolysis to form partially hydrolyzed FB1, which is further metabolized into the hydrolyzed form known as HFB1. In pigs, HFB1 persists at low concentrations for a few more days after FB1 metabolism. It has been observed that FB1 is more toxic than HFB1 in piglets. Although N-acylation of FB1 and the formation of HFB1 have been demonstrated in human cell lines and rats, the metabolic pathways in avian species remain uncertain. Therefore, it is not possible to generalize the metabolic processes of fumonisins in all animals at present (19). The toxicity of fumonisins in animals is believed to stem from their interference with sphingolipid metabolism. Current evidence indicates that fumonisins act as specific inhibitors of ceramide synthase, an essential enzyme involved in the synthesis of ceramide and other complex sphingolipids. By inhibiting this enzyme system, fumonisins disrupt the normal metabolism of sphingolipids. This disruption leads to an accumulation of sphingosine (SO) and sphinganine (SA), two important sphingolipid intermediates, in the tissues. Consequently, there is an alteration in the ratio of SA to SO. Studies have shown that feeding fumonisins, particularly FB1, to broilers, turkeys, and ducklings leads to an increase in the SA:SO ratio in their tissues. This change in sphingolipid composition is believed to contribute to the toxic effects associated with fumonisin exposure in these animals (20).

Trichothecenes:

Trichothecene mycotoxins are a class of fungal metabolites characterized by a shared core structure. This group includes various toxins such as T-2 toxin, HT-2 toxin, diacetoxyscirpenol (DAS), monoacetoxyscirpenol (MAS), neosolaniol, 8-acetoxyneosolaniol, 4-deacetylneosolaniol, nivalenol, 4-acetoxynivalenol (Fusarenone-X), DON (vomitoxin), and 3-acetyl deoxynivalenol. These mycotoxins are recognized as potent inhibitors of protein synthesis in cells. Their primary toxic effect at the cellular level is the inhibition of protein synthesis, which can have significant impacts on cellular functions. Additionally, the disruption of DNA and RNA synthesis is observed as a secondary effect of trichothecene mycotoxin exposure. Overall, trichothecene mycotoxins exert their toxicity by impairing protein synthesis, which subsequently leads to disturbances in cellular processes and can have detrimental effects on various biological systems (21). Among livestock, deoxynivalenol (DON) is considered the

most significant trichothecene mycotoxin, commonly contaminating corn, wheat, and other grain commodities. Lower amounts of T-2 toxin and diacetoxyscirpenol (DAS) are sporadically found in the same sources. Poultry and cattle exhibit greater tolerance to trichothecenes compared to pigs.While, DON is prevalent in crops, T-2 toxin occurs less frequently. European grain samples have reported higher occurrences of trichothecenes such as DON (57%), nivalenol (16%), and fusarenon X (10%) compared to other trichothecenes like T-2 toxin (20%), HT-2 toxin (14%), T-2 tetraol (6%), neosolaniol (1%), DAS (4%), and MAS (1%) (22).

Mechanisms of Toxicity :

The mechanisms of mycotoxin toxicity can vary depending on the specific mycotoxin and the target organs or systems affected. Here are some general mechanisms by which mycotoxins exert their toxic effects:

Inhibition of Protein Synthesis: Some mycotoxins, such as trichothecenes (e.g., deoxynivalenol) and ribotoxins (e.g., ricin), inhibit protein synthesis by targeting components of the ribosome or the protein synthesis machinery. This disruption of protein synthesis can impair cell function and lead to various toxic effect (23).

DNA Damage: Certain mycotoxins, including aflatoxins and fumonisins, can induce DNA damage in cells. They can bind to DNA, form DNA adducts, or interfere with DNA replication or repair mechanisms, leading to genetic mutations and cellular dysfunction (24).

Oxidative Stress: Many mycotoxins, such as aflatoxins, ochratoxins, and trichothecenes, generate reactive oxygen species (ROS) or disrupt the cellular antioxidant defense system. This imbalance between ROS production and antioxidant capacity can result in oxidative stress, causing damage to lipids, proteins, and DNA within cells (25).

Disruption of Cell Membranes: Some mycotoxins, like fusaric acid and certain trichothecenes, can disrupt the integrity and functionality of cell membranes. They can alter membrane fluidity, ion transport, and membrane-bound enzyme activities, leading to cellular dysfunction and death (26).

Hormonal Disruption: Certain mycotoxins, such as zearalenone and aflatoxins, exhibit estrogenic or anti-estrogenic activities, interfering with hormonal signaling pathways. This can disrupt endocrine function, leading to reproductive abnormalities, altered metabolism, and other hormonal disturbances (27).

Immune Modulation: Mycotoxins can modulate the immune system by suppressing immune cell function, altering cytokine production, or impairing the response to pathogens. This immunomodulatory effect can weaken the immune response and increase susceptibility to infections. It is important to note that the specific mechanisms of mycotoxin toxicity can vary among different mycotoxins and target organs, and there may be overlap between different mechanisms for some mycotoxins. The severity and specific effects of

mycotoxin toxicity also depend on factors such as the dose, duration of exposure, and individual susceptibility (28).

Routes of Exposure:

Domestic birds can be exposed to mycotoxins through various routes, including:

1. Ingestion: The primary route of exposure is through the ingestion of mycotoxin-contaminated feed or water. Birds consuming contaminated feed are at risk of mycotoxicosis (29).
2. Inhalation: Airborne mycotoxin-contaminated dust particles generated during feed handling, milling, or storage can be inhaled by birds, leading to respiratory exposure (30).
3. Transovarian Transmission: Some mycotoxins, such as aflatoxins, can cross the placenta or transovarianly transfer from the hen to the developing embryos inside the eggs. This can result in exposure of the birds to mycotoxins even before hatching (31).

Clinical Signs and Symptoms:

Mycotoxicosis in domestic birds can manifest through a variety of clinical signs and symptoms, which may vary depending on the specific mycotoxin involved and the duration and level of exposure. Common clinical signs include: Reduced feed intake, Weight loss, Poor feather quality, Diarrhea, decreased egg production, decreased fertility, Increased mortality and Neurological abnormalities (tremors, ataxia, convulsions) (32).

Gross Lesions:

Mycotoxicosis in domestic birds can result in various gross lesions or visible abnormalities in affected organs or tissues. The specific gross lesions observed may vary depending on the type of mycotoxin, the level and duration of exposure, and the target organ affected. Here are some examples of gross lesions commonly associated with mycotoxicosis.

Liver Lesions: Hepatomegaly (enlarged liver) Aflatoxins, for example, can cause hepatomegaly characterized by an enlarged, pale, and friable liver.

Hepatic necrosis: Some mycotoxins, such as aflatoxins and ochratoxins, can induce necrotic lesions in the liver, leading to areas of tissue death (33).

Kidney Lesions: Renomegaly (enlarged kidneys): Certain mycotoxins, like ochratoxins, can cause renomegaly, where the kidneys appear swollen and enlarged. Renal tubular degeneration: Mycotoxins, including ochratoxins and citrinin, can induce degenerative changes in the renal tubules.

Gastrointestinal Lesions: Gastroenteritis: Some mycotoxins, such as deoxynivalenol (DON) and T-2 toxin, can cause inflammation and damage to the gastrointestinal tract, resulting in lesions such as hemorrhage, ulceration, and congestion. Respiratory Lesions: Pulmonary edema: Mycotoxins like T-2 toxin can

induce pulmonary edema, where fluid accumulates in the lungs. Tracheitis and bronchitis: Inhalation of mycotoxin-contaminated dust particles can cause inflammation and damage to the respiratory tract, leading to lesions such as tracheitis and bronchitis (34).

Reproductive Lesions:

Ovarian atrophy: Some mycotoxins, including zearalenone, can cause ovarian atrophy, where the ovaries appear smaller and have reduced follicular development. Testicular degeneration: Mycotoxins can induce degenerative changes in the testes, resulting in reduced sperm production and fertility in male birds (35).

Histopathological changes of Mycotoxin:

Histologically, in broilers fed aflatoxin, livers, kidneys, and bursa exhibit specific changes.

Liver: Histopathological examination of the liver may reveal hepatocellular degeneration, necrosis, and fatty changes. Inflammatory infiltrates and bile duct proliferation may also be observed in response to mycotoxin exposure.

Kidneys: Proximal tubules show dilatation and necrosis, with affected epithelium displaying enlarged and abnormal-shaped nuclei. Focal aggregation of lymphoid cells is observed among degenerated renal tubules. The glomerular basement membrane thickens, and the glomerular tuft collapses (36).

Bursa of Fabricius: The bursa of Fabricius, an organ involved in immune function in birds, shows significant alterations in the group fed aflatoxin. The normal bursal architecture is lost, with thinning of the cortical layer. Medullary lymphoid depletion and lymphocytolysis occur. Edema and interfollicular fibrosis are present. The bursal epithelial layer displays hyperplasia, disruption, and corrugation. These histological findings indicate the detrimental effects of aflatoxin on the liver, kidneys, and bursa of Fabricius in broiler chickens (36).

Muscles: Mycotoxin exposure can lead to muscle degeneration characterized by the loss of normal muscle structure and integrity. This can include muscle fiber atrophy, disorganization, and fragmentation, Inflammation: Mycotoxin-induced inflammation may be observed in the muscles. Inflammatory infiltrates, consisting of immune cells such as lymphocytes and macrophages, can be seen in the affected muscle tissue. (37).

Treatment of Mycotoxicosis:

Treatment of mycotoxicosis in poultry involves various strategies aimed at mitigating the adverse effects of mycotoxins and restoring the health and productivity of affected birds. It is important to note that prevention and management practices are crucial in controlling mycotoxin contamination in poultry feed. However, if mycotoxicosis occurs, the following treatment approaches can be considered.

Adsorbents and Binders: The use of adsorbents and binders is a common strategy to reduce the absorption and bioavailability of mycotoxins in the gastrointestinal tract of poultry. These substances bind to

mycotoxins and prevent their interaction with the intestinal lining, reducing their harmful effects. Examples of commonly used adsorbents and binders include activated charcoal, bentonite clay, zeolites, and certain yeast cell wall components (38).

Nutritional Interventions: Nutritional strategies can help alleviate the negative effects of mycotoxins on poultry. This may involve the supplementation of specific nutrients, such as antioxidants (e.g., vitamin E, selenium) and certain amino acids (e.g., methionine), which can support the detoxification and repair mechanisms of the bird's body. Additionally, providing a well-balanced diet with adequate levels of essential vitamins and minerals can enhance the overall health and immune function of poultry (39).

Veterinary Support: In severe cases of mycotoxicosis, it is important to seek veterinary assistance for appropriate diagnosis and treatment. Veterinary professionals can administer specific medications or supportive therapies tailored to the specific condition and symptoms presented by the affected birds. This may include anti-inflammatory drugs, antibiotics to control secondary infections, and intravenous fluid therapy for rehydration (40).

Prevention and Control:

Prevention and control of mycotoxin contamination in poultry feed require a multi-faceted approach involving various strategies at different stages of production. Here are some commonly employed preventive and control measures:

Good Agricultural Practices (GAPs):

Implementing good agricultural practices during crop cultivation can help reduce the risk of mycotoxin contamination. This includes proper field management, crop rotation, timely harvesting, and effective weed control. Maintaining optimal moisture levels and preventing insect infestation can also minimize fungal growth and mycotoxin production(41).

Good Manufacturing Practices (GMPs):

Adhering to good manufacturing practices during feed production and processing is crucial for reducing mycotoxin contamination. This includes regular equipment cleaning and maintenance, proper storage conditions, and appropriate monitoring of raw materials and finished feed for mycotoxin levels (42).

Mycotoxin Testing and Monitoring:

Regular testing and monitoring of feed ingredients and finished feed for mycotoxin contamination can help identify potential risks and ensure timely intervention. Analytical techniques such as high-performance liquid chromatography (HPLC) or enzyme-linked immunosorbent assay (ELISA) can be employed for mycotoxin analysis (43).

Feed Ingredient Sourcing:

Selecting high-quality feed ingredients from reliable sources can help minimize the risk of mycotoxin contamination. Suppliers should provide certificates of analysis or other documentation to verify the quality and mycotoxin levels of the ingredients (44).

Mycotoxin Binders and Detoxifiers:

Mycotoxin binders and detoxifiers can be added to the feed to mitigate the adverse effects of mycotoxins. These substances bind to mycotoxins in the gastrointestinal tract, preventing their absorption and reducing their negative impact on bird health. Examples of binders and detoxifiers include activated carbon, bentonite clay, yeast cell walls, and certain plant extracts (45).

Proper Storage and Handling:

Appropriate storage conditions, including maintaining optimal temperature, humidity, and ventilation, are crucial for preventing fungal growth and mycotoxin production during feed storage. Implementing proper handling procedures, including segregation of contaminated and uncontaminated feed, can also help minimize the spread of mycotoxins (46).

Conclusion:

In conclusion, mycotoxicosis can have significant influences on the health, productivity, and profitability of domestic birds. Understanding the impacts of mycotoxin contamination and implementing effective prevention and mitigation strategies are essential for maintaining the welfare and productivity of poultry populations.

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جوانب حول تأثيرات التسمم الفطري في الطيور الداجنة: مراجعة

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الخلاصة

يعد التسمم الفطري مصدر قلق كبير في صناعة الدواجن في جميع أنحاء العالم، ويحدث بسبب تناول السموم الفطرية، وهذه السموم هي نواتج أيض ثانوية سامة تنتجها الفطريات التي عادة ما تلوث الأعلاف الحيوانية. وتهدف هذه المراجعة إلى استعراض التاثيرات المختلفة للتسمم الفطري على الطيور الداجنة. وكذلك مناقشة السموم الفطرية الرئيسية التي تؤثر على الدواجن، بما في ذلك الأفلاتوكسينات، والأوكراتوكسينات، والتريكوثيسينات، والزيرالينون، والفومونيزينات، ومصادر تلوث العلف بهذه السموم، مثل عوامل ما قبل الحصاد وما بعد الحصاد وعوامل التخزين. بالإضافة إلى ذلك، تسلط المراجعة الضوء على تأثير التسمم الفطري على صحة الطيور، بما في ذلك تثبيط جهاز المناعة، واضطرابات الجهاز الهضمي، وانخفاض النمو والتطور، واضطرابات التكاثر، فضلاً عن آثاره على جودة البيض ونسبة الفقس. علاوة على ذلك، عرض استراتيجيات وقائية وإدارية، مثل تنفيذ الممارسات الزراعية الجيدة، وإدارة الأعلاف ومراقبة الجودة، واستخدام المواد الرابطة ومثبطات للسموم الفطرية، والمواد المضافة إلى الأعلاف والمكملات الغذائية، والتدخلات البيطرية. تؤكد المراجعة على أهمية المبادئ التوجيهية التنظيمية وبرامج المراقبة لضمان سلامة الأغذية والتخفيف من حدة المشكلات المتعلقة بالسموم الفطرية. وعموما، فإن فهم تأثيرات التسمم الفطري في الطيور الداجنة أمر ضروري ومهم لوضع وتنفيذ تدابير فعالة لمنع التلوث بالسموم الفطرية لتعزيز صحة وإنتاجية الدواجن.

الكلمات المفتاحية: الطيور الداجنة, mycotoxins ,التلوث, الصحة.