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Review

Problems and Mitigation Strategies of Trichothecenes Mycotoxins in Laying Hens Production

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Abstract: This review paper comprehensively explores the problems associated with trichothecenes mycotoxins in laying hens production and discusses mitigation strategies to address these challenges. Trichothecenes, a diverse class of mycotoxins primarily produced by *Fusarium* fungi, are examined in terms of their nature, occurrence, toxicology, and implications in poultry production. The metabolism of T-2 toxin in poultry is analyzed, highlighting the complex biotransformation pathways and the formation of metabolites with altered toxicity profiles. The most common problems of T-2 toxin in laying hens production are discussed, including adverse effects on poultry health, performance, and table egg quality. The influence of T-2 toxin on table egg quality is explored, focusing on its impact on shell integrity, yolk color, albumen consistency, and consumer acceptance. Finally, mitigation strategies for trichothecenes in laying hens production are reviewed, encompassing both pre-harvest and post-harvest interventions such as good agricultural practices, monitoring and testing programs, physical and chemical detoxification methods, nutritional supplementation, and biosecurity measures. By understanding the nature of trichothecenes mycotoxins and implementing effective mitigation strategies, producers can ensure the safety, welfare, and productivity of laying hens while maintaining the quality and marketability of table eggs for consumers.

Keywords: Mycotoxins; T-2; Fumonisin; nutrition; laying hens; poultry; eggs.

1. Introduction

The poultry industry plays a pivotal role in global food security, providing a significant source of protein in the form of eggs and meat [1]. However, the industry faces numerous challenges, among which mycotoxin contamination poses a significant threat to both poultry health and productivity [2]. Mycotoxins are toxic secondary metabolites produced by various fungi, and their presence in feed can lead to adverse effects on poultry health, performance, and even food safety. Trichothecenes, a major class of mycotoxins, are produced primarily by *Fusarium* fungi and encompass a diverse range of compounds, including T-2 toxin and fumonisin B1 [3]. In the context of laying hens production, the prevalence of trichothecene mycotoxins presents a pressing concern, warranting thorough investigation and the development of effective mitigation strategies [4].

Trichothecenes exert their toxic effects through various mechanisms, including inhibition of protein synthesis, disruption of cell membranes, and induction of oxidative stress [5,6]. Among the *Fusarium*-produced trichothecenes, T-2 toxin and fumonisin B1 are of particular importance due to their widespread occurrence and significant impact on poultry health and production [7]. T-2 toxin, a potent mycotoxin known for its immunosuppressive and cytotoxic effects, can cause severe gastrointestinal disturbances, immunosuppression, and reproductive disorders in laying hens.

Similarly, fumonisin B1, another prevalent trichothecene mycotoxin, is associated with various health issues in poultry, including hepatotoxicity, nephrotoxicity, and impaired immune function.

The presence of trichothecene mycotoxins in laying hen diets poses multifaceted challenges to producers, veterinarians, and researchers alike. Firstly, the occurrence of these toxins in feed ingredients is often unpredictable and influenced by factors such as climate, storage conditions, and agricultural practices. Additionally, the complex interactions between different mycotoxins and their synergistic or additive effects further complicate the assessment of their impact on poultry health. Moreover, the regulations governing mycotoxin levels in feed vary across regions, making it challenging for producers to implement consistent mitigation measures.

In light of these challenges, the development of effective mitigation strategies is imperative to safeguard laying hen health and ensure the production of safe and high-quality eggs. Various approaches have been proposed to mitigate the adverse effects of trichothecene mycotoxins in poultry production, ranging from pre-harvest management practices to post-harvest detoxification techniques [8,9]. Pre-harvest strategies aim to minimize mycotoxin contamination at the source by implementing good agricultural practices, such as crop rotation, proper storage, and timely harvest [10]. Additionally, the use of resistant crop varieties and biological control agents shows promise in reducing Fusarium infection and subsequent mycotoxin production in the field.

Furthermore, post-harvest interventions play a crucial role in mitigating mycotoxin contamination during feed processing and storage [11]. Physical methods, such as sorting, cleaning, and thermal processing, can help reduce mycotoxin levels in feed ingredients. Chemical detoxification agents, including binders and adsorbents, are commonly used to sequester mycotoxins in the gastrointestinal tract and prevent their absorption. Moreover, advances in biotechnological approaches, such as enzymatic detoxification and microbial biotransformation, offer novel strategies for mycotoxin degradation and detoxification [10].

Despite the progress made in understanding trichothecene mycotoxins and their mitigation strategies, several knowledge gaps remain to be addressed. Further research is needed to elucidate the mechanisms underlying mycotoxin toxicity and explore novel approaches for their prevention and control [12]. Additionally, the development of rapid and sensitive analytical methods is essential for accurate mycotoxin detection and monitoring in feed and poultry products. Collaborative efforts between researchers, industry stakeholders, and regulatory agencies are crucial for implementing comprehensive strategies to mitigate the impact of trichothecene mycotoxins on laying hen production.

In this review paper, we aim to provide a comprehensive overview of the problems associated with trichothecene mycotoxins in laying hens production, with a specific focus on Fusarium fungi-derived toxins such as T-2 toxin and fumonisin B1. We will examine the prevalence of these mycotoxins in laying hen diets, their toxicological effects on performance, and egg quality, and suggest strategies for their mitigation. By synthesizing existing knowledge and identifying research gaps, this review aims to contribute to the development of effective strategies for ensuring the safety and sustainability of laying hen production in the face of mycotoxin challenges.

2. Trichothecenes mycotoxins: Nature, occurrence, toxicology, and implications

Mycotoxins represent a diverse group of secondary metabolites produced by various fungi, which contaminate agricultural commodities such as grains, fruits, and forages. Among the numerous classes of mycotoxins, trichothecenes stand out as one of the most significant and widely studied due to their potent toxic effects on humans and animals [13,14]. Trichothecenes are produced primarily by fungi belonging to the genera *Fusarium*, *Myrothecium*, *Trichoderma*, and *Stachybotrys*, and their occurrence in food and feed poses serious health risks and economic consequences [15].

2.1. Nature of trichothecenes

Trichothecenes are a structurally diverse group of sesquiterpenoid mycotoxins characterized by a 12,13-epoxytrichothec-9-ene nucleus, which serves as the core structure for over 200 naturally occurring derivatives [16]. These derivatives vary in their chemical structure and biological activity, exhibiting a wide range of toxicological effects on mammals, birds, and insects. Trichothecenes are classified into four main types based on their chemical structure and substitution pattern: Type A, Type B, Type C, and Type D trichothecenes [17] (Figure 1).

Type A trichothecenes, such as T-2 toxin and HT-2 toxin, contain a keto group at C-8 and various functional groups at C-3 and C-4 positions [18]. Type B trichothecenes, including deoxynivalenol (DON) and nivalenol (NIV), lack the keto group at C-8 and exhibit different substitution patterns at C-3 and C-4 positions [19]. Type C and Type D trichothecenes are less common and are primarily produced by *Myrothecium* and *Trichoderma* fungi [20], respectively.

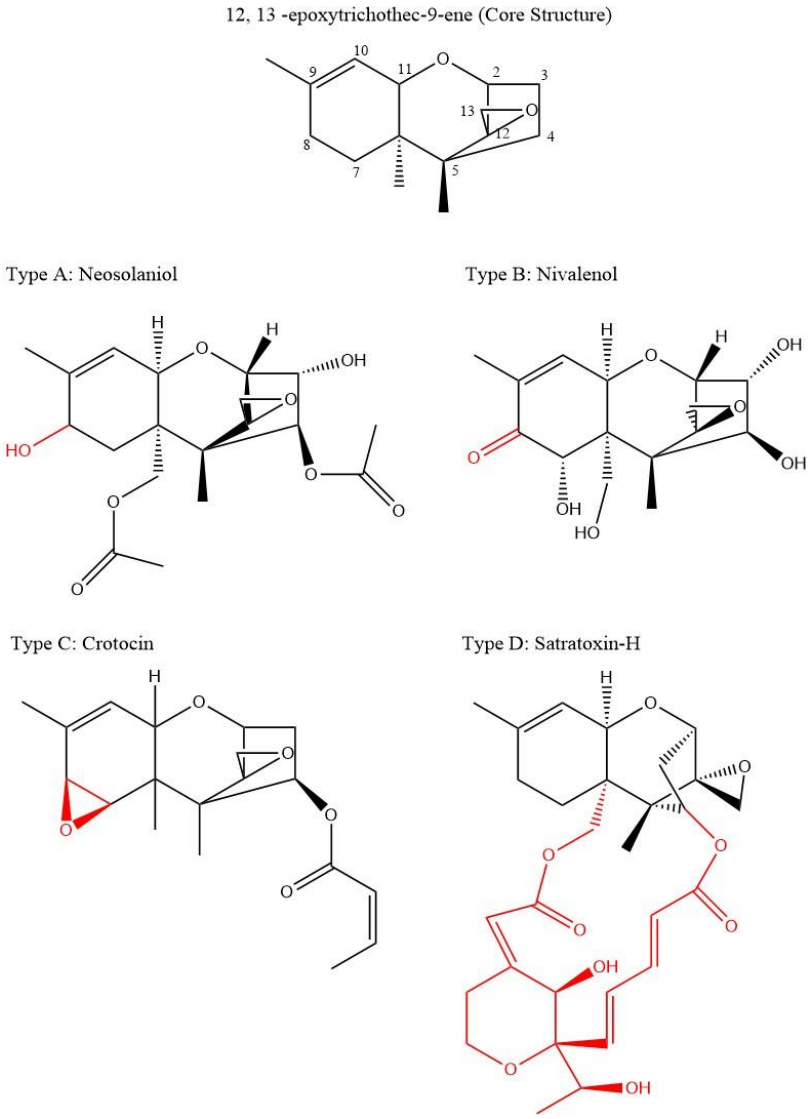

Figure 1. The core structure of all major trichothecenes with major examples from each classification type. The red color represents identifying functional groups for the classification type.

2.2. Occurrence of trichothecenes

Trichothecene mycotoxins are produced by a wide range of fungi, with species of the genus *Fusarium* being the most prevalent producers in crops. *Fusarium graminearum*, *Fusarium culmorum*, and *Fusarium sporotrichioides* are among the most important species associated with trichothecene contamination in cereal grains such as wheat, barley, maize, and rice [21]. These fungi infect plants during various stages of growth, leading to the production of trichothecene mycotoxins in infected

kernels. Factors such as temperature, humidity, and agricultural practices influence the growth and toxin production of *Fusarium* species, contributing to the variability in trichothecene contamination levels observed across different regions and cropping seasons [22].

Aside from *Fusarium* species, other fungi such as *Stachybotrys chartarum* (commonly known as black mold) produce trichothecene mycotoxins, including satratoxins and roridins, which are associated with indoor mold contamination and adverse health effects in humans [23]. Furthermore, emerging research has identified novel trichothecene-producing fungi from various environmental sources, highlighting the complexity of trichothecene ecology and the potential for new sources of contamination in food and feed [24].

Trichothecene contamination of food and feed poses significant risks to human and animal [25] health due to their stability during food processing and resistance to degradation by heat and pH. Consumption of trichothecene-contaminated grains and products can lead to acute and chronic health effects in humans, ranging from gastrointestinal disturbances to immunosuppression, neurotoxicity, and carcinogenicity [26]. In animal production systems, trichothecene exposure is associated with reduced feed intake, impaired growth performance, and increased susceptibility to infectious diseases, leading to economic losses and welfare concerns.

2.3. Toxicology of trichothecenes

The toxicological effects of trichothecene mycotoxins vary depending on factors such as chemical structure, dose, duration of exposure, and species susceptibility [27]. Type A trichothecenes, exemplified by T-2 toxin and HT-2 toxin, are among the most toxic members of the trichothecene family, exhibiting potent cytotoxic, immunosuppressive, and genotoxic effects. These mycotoxins inhibit protein synthesis by binding to the 60S ribosomal subunit, leading to the disruption of cell function and induction of apoptosis in target tissues. Moreover, T-2 toxin and its metabolites can induce oxidative stress, inflammation, and lipid peroxidation, contributing to tissue damage and organ dysfunction [28].

Type B trichothecenes, including DON and NIV, are primarily known for their effects on the gastrointestinal tract and immune system. DON, in particular, acts as a potent inhibitor of protein synthesis by binding to the ribosomal peptidyl transferase center, leading to the induction of ribotoxic stress response and activation of pro-inflammatory pathways [29]. Acute exposure to high doses of DON can cause feed refusal, vomiting, and diarrhea in animals, while chronic exposure to low doses is associated with immunosuppression, reproductive disorders, and reduced performance [30].

In addition to their direct toxic effects, trichothecenes can exert synergistic or additive interactions with other mycotoxins and environmental stressors, exacerbating their impact on human and animal health. For example, the co-occurrence of trichothecenes with aflatoxins, ochratoxins, or zearalenone in contaminated feed can potentiate their toxic effects and increase the risk of adverse health outcomes [31]. Furthermore, factors such as age, sex, genetics, and nutritional status influence individual susceptibility to trichothecene toxicity, highlighting the importance of risk assessment and management strategies tailored to specific populations.

2.4. Implications of trichothecenes in agriculture and food safety

Trichothecene contamination of agricultural commodities poses significant challenges to food safety, international trade, and regulatory compliance [32]. The presence of trichothecene mycotoxins in food and feed exceeds permissible limits set by regulatory agencies in many countries, leading to the rejection of contaminated shipments and economic losses for producers and exporters. Moreover, the complex nature of trichothecene toxicity and the potential for synergistic interactions with other mycotoxins necessitate comprehensive risk assessment and management strategies to ensure the safety and quality of food and feed products [33].

In animal production systems, trichothecene contamination of feed contributes to reduced productivity, increased disease incidence, and impaired welfare of livestock and poultry [34]. Laying hens exposed to trichothecene-contaminated feed may exhibit decreased egg production,

poor egg quality [35], and increased susceptibility to infectious diseases such as coccidiosis and Newcastle disease (Figure 2).

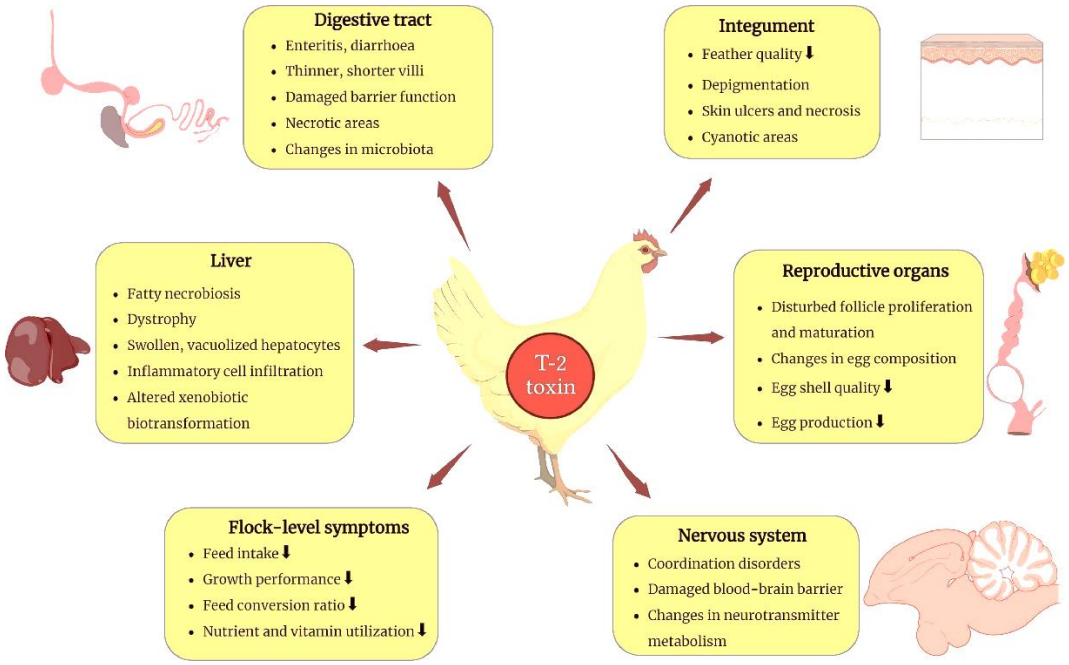

Figure 2. A detailed summary of the major pathological findings in poultry resulting from exposure to T-2 toxin [36].

Furthermore, the transfer of trichothecene residues from feed to animal-derived products such as eggs and meat raises concerns regarding their potential impact on human health and consumer safety.

3. Metabolism of T-2 toxin in poultry

The metabolism of T-2 toxin in poultry is a complex process influenced by various factors including species differences, route of exposure, dose, and duration of exposure [37]. T-2 toxin, a potent Type A trichothecene mycotoxin produced by *Fusarium* fungi, poses significant health risks to poultry due to its cytotoxic, immunosuppressive, and genotoxic properties. Understanding the metabolism of T-2 toxin in poultry is crucial for assessing its toxicological effects, developing effective mitigation strategies, and ensuring the safety of poultry products for human consumption.

Upon ingestion, the T-2 toxin undergoes extensive metabolism in the gastrointestinal tract, liver, and other tissues, leading to the formation of metabolites with altered chemical and biological properties. The metabolism of T-2 toxin in poultry involves several phase I and phase II biotransformation reactions mediated by various enzymes, including cytochrome P450 enzymes, glutathione S-transferases, and sulfotransferases [38].

Phase I metabolism of T-2 toxin involves the conversion of the parent compound into intermediate metabolites through oxidation, reduction, and hydrolysis reactions. Cytochrome P450 enzymes, particularly cytochrome 3A and cytochrome 1A, play a critical role in catalyzing the oxidative metabolism of T-2 toxin to form hydroxylated metabolites such as HT-2 toxin, neosolaniol, and other less characterized metabolites. These hydroxylated metabolites exhibit varying degrees of toxicity and may contribute to the overall toxicity of T-2 toxin in poultry [39].

In addition to oxidative metabolism, T-2 toxin can undergo reduction reactions mediated by reductases, leading to the formation of metabolites such as T-2 triol and T-2 tetraol. These reduced metabolites are often considered less toxic than the parent compound but may still exhibit cytotoxic and immunosuppressive effects in poultry.

Phase II metabolism of T-2 toxin involves conjugation reactions with endogenous molecules such as glutathione, sulfate, and glucuronic acid, leading to the formation of water-soluble metabolites that are more readily excreted from the body [40]. Glutathione S-transferases catalyze the conjugation of T-2 toxin with GSH to form glutathione conjugates, which are subsequently metabolized to mercapturic acid derivatives and excreted in the urine and feces. Sulfotransferases and UDP-glucuronosyltransferases catalyze the conjugation of T-2 toxin with sulfate and glucuronic acid, respectively, leading to the formation of sulfate and glucuronide conjugates that are excreted via bile and urine.

The metabolism of T-2 toxin in poultry can be influenced by various factors including age, sex, genetics, and nutritional status [41]. Young animals, particularly chicks and poults, are often more susceptible to T-2 toxin toxicity due to their immature metabolic pathways and reduced detoxification capacity compared to adult birds [42]. Furthermore, factors such as concurrent exposure to other mycotoxins, the presence of underlying diseases, and stressors such as heat stress or transportation can modulate the metabolism and toxicity of T-2 toxin in poultry.

The toxicological effects of T-2 toxin and its metabolites in poultry are diverse and may manifest as gastrointestinal disturbances, immunosuppression, hematological alterations, and organ damage. Acute exposure to high doses of T-2 toxin can cause feed refusal, diarrhea, hemorrhagic lesions, and mortality in poultry, while chronic exposure to lower doses may result in growth retardation, reduced egg production, and increased susceptibility to infectious diseases [43]. The immunosuppressive effects of T-2 toxin can impair the poultry's ability to mount an effective immune response against pathogens, leading to increased incidence and severity of infections.

4. The most often problems of T-2 toxin in laying hens production

The presence of T-2 toxin in laying hens production poses significant challenges to both poultry health and egg production. T-2 toxin, a potent Type A trichothecene mycotoxin produced by *Fusarium* fungi, exerts a range of toxicological effects that can negatively impact laying hens' performance, welfare, and the quality of eggs produced. Understanding the problems associated with T-2 toxin in laying hens production is crucial for implementing effective mitigation strategies and safeguarding both animal and consumer health [44].

One of the primary problems of T-2 toxin in laying hens production is its adverse effects on poultry health [42]. T-2 toxin is known to be cytotoxic, immunosuppressive, and genotoxic, leading to a range of physiological and pathological effects in laying hens [39]. Acute exposure to high doses of T-2 toxin can cause severe gastrointestinal disturbances, including feed refusal, diarrhea, and hemorrhagic lesions in the gastrointestinal tract [45]. These gastrointestinal effects can result in reduced feed intake, impaired nutrient absorption, and weight loss in laying hens, ultimately impacting their growth and performance.

Chronic exposure to lower doses of T-2 toxin can lead to immunosuppression, making laying hens more susceptible to infectious diseases such as coccidiosis, infectious bronchitis, and Newcastle disease [46]. T-2 toxin suppresses the poultry's immune response by inhibiting lymphocyte proliferation, cytokine production, and antibody formation, compromising their ability to fight off pathogens and increasing the risk of disease outbreaks. Furthermore, T-2 toxin-induced immunosuppression can exacerbate the severity of existing infections and delay the recovery of affected birds, leading to economic losses for producers and reduced welfare for laying hens [47].

Another significant problem of T-2 toxin in laying hens production is its impact on egg quality and safety. T-2 toxin can accumulate in the ovaries and oviducts of laying hens, leading to the deposition of toxin residues in eggs [48]. Consumption of T-2 toxin-contaminated eggs poses risks to human health, as T-2 toxin and its metabolites can be transferred to consumers through egg consumption. T-2 toxin residues in eggs may exceed regulatory limits set by food safety authorities, leading to the rejection of contaminated eggs and economic losses for producers [48].

Moreover, T-2 toxin contamination of eggs can affect their quality attributes, including shell quality, yolk color, and albumen consistency. T-2 toxin has been shown to impair calcium

metabolism and reduce shell thickness in laying hens, resulting in increased susceptibility to eggshell fractures and microbial contamination. Additionally, T-2 toxin can alter the color and viscosity of egg yolks and albumen, making them less appealing to consumers and affecting the marketability of eggs [49].

The economic implications of T-2 toxin contamination in laying hens production are substantial, affecting both producers and consumers. Producers incur costs associated with reduced egg production, impaired feed efficiency, increased mortality, and veterinary treatments for T-2 toxin-related health problems in laying hens [44]. Furthermore, the rejection of T-2 toxin-contaminated eggs by food safety authorities and retailers leads to financial losses for producers and impacts consumer confidence in the safety and quality of poultry products.

Mitigating the problems of T-2 toxin in laying hens production requires a multi-faceted approach that addresses both the prevention of mycotoxin contamination and the management of contaminated feed and poultry [50]. Implementing good agricultural practices such as crop rotation, proper storage, and timely harvest can reduce the risk of Fusarium fungal infection and subsequent T-2 toxin contamination in feed ingredients. Additionally, monitoring and testing programs can help identify and mitigate T-2 toxin contamination in feed and poultry products, ensuring compliance with regulatory standards and consumer safety requirements [51].

Furthermore, post-harvest interventions such as physical, chemical, and biological detoxification methods can be employed to reduce T-2 toxin levels in contaminated feed. Physical methods such as sorting, cleaning, and thermal processing can remove or destroy T-2 toxin-contaminated grains and feed ingredients [10]. Chemical detoxification agents such as binders and adsorbents can sequester the T-2 toxin in the gastrointestinal tract of laying hens, preventing its absorption and reducing its toxic effects. Biological detoxification methods such as enzymatic degradation and microbial biotransformation offer promising strategies for the degradation and detoxification of T-2 toxin in feed and poultry products [10].

5. The influence of T-2 toxin on table egg quality

The influence of T-2 toxin on table egg quality is a critical concern in poultry production, as it directly impacts consumer safety, marketability, and economic viability. T-2 toxin, a potent Type A trichothecene mycotoxin produced by *Fusarium* fungi, can contaminate poultry feed and subsequently accumulate in eggs, leading to adverse effects on egg quality attributes such as shell integrity, yolk color, albumen consistency, and overall consumer acceptance [52]. Understanding the influence of T-2 toxin on table egg quality is essential for implementing effective mitigation strategies and ensuring the safety and marketability of poultry products [53].

One of the primary effects of T-2 toxin on table egg quality is its impact on shell integrity and strength (Figure 3). T-2 toxin has been shown to interfere with calcium metabolism in laying hens, leading to reduced shell thickness and increased susceptibility to eggshell fractures. Calcium is essential for the formation of eggshells, and any disruption in calcium absorption or utilization can compromise shell quality [54]. Studies have demonstrated that T-2 toxin exposure can result in thin, fragile eggshells that are prone to breakage during handling, transportation, and storage [55]. Eggshell fractures not only reduce the aesthetic appeal of eggs but also increase the risk of microbial contamination and spoilage, thereby affecting their shelf life and consumer safety [55].

In addition to shell quality, T-2 toxin can also affect the color and consistency of egg yolks and albumen. Yolk color is an important quality attribute that influences consumer perception and preference for eggs. T-2 toxin exposure has been associated with alterations in yolk color, resulting in paler yolks with reduced pigmentation. This can impact the visual appeal of eggs and may lead to consumer dissatisfaction [48]. T-2 toxin can affect the viscosity and gelation properties of albumen, resulting in changes in albumen consistency and texture. Eggs with abnormal albumen consistency may be perceived as lower quality and may not meet consumer expectations for freshness and taste.

Figure 3. Effects of mycotoxins on table eggshell quality.

Moreover, T-2 toxin contamination of eggs can lead to off-flavors and odors, further diminishing their sensory quality and consumer acceptance. T-2 toxin and its metabolites can impart unpleasant tastes and aromas to eggs, making them unpalatable or undesirable for consumption. These off-flavors and odors may result from the interaction of T-2 toxin with lipids and proteins in egg yolks and albumen, leading to the formation of volatile compounds that contribute to off-flavors and odors. Consumer studies have shown that eggs with off-flavors and odors are often rejected or perceived as inferior in quality, highlighting the importance of mitigating T-2 toxin contamination to preserve the sensory attributes of table eggs [56].

6. Mitigation strategies for trichothecenes in laying hens production

Mitigation strategies for trichothecenes in laying hens production are crucial for safeguarding poultry health, ensuring egg safety, and maintaining the economic viability of poultry operations [57]. Trichothecenes, a class of mycotoxins primarily produced by *Fusarium* fungi, pose significant challenges to laying hens production due to their toxicological effects on poultry health and egg quality [58]. Implementing effective mitigation strategies involves a multi-faceted approach that addresses both pre-harvest and post-harvest factors, focusing on preventing mycotoxin contamination in feed ingredients and minimizing the impact of contaminated feed on laying hens [59].

Pre-harvest mitigation strategies aim to reduce the risk of trichothecene contamination in feed ingredients by implementing good agricultural practices and crop management techniques [60]. Crop rotation, tillage practices, and weed control can help minimize *Fusarium* fungal infection and subsequent mycotoxin production in field crops such as maize, wheat, barley, and soybeans. Additionally, selecting resistant crop varieties and planting them at optimal times can reduce the vulnerability of crops to fungal infection and mycotoxin contamination. Proper storage facilities and practices, including adequate ventilation, temperature control, and moisture management, are essential for preventing post-harvest mold growth and mycotoxin production in stored grains and feed ingredients [60].

Monitoring and testing programs are essential components of pre-harvest mitigation strategies, allowing producers to assess the risk of mycotoxin contamination in feed ingredients and make informed decisions regarding feed formulation and usage. Regular sampling and analysis of feed ingredients for mycotoxin levels can help identify contaminated batches and guide the implementation of appropriate mitigation measures [61]. Additionally, the use of predictive models and risk assessment tools can help anticipate potential mycotoxin outbreaks [62] and inform preventive measures to minimize their impact on laying hens production.

Post-harvest mitigation strategies focus on minimizing the impact of trichothecene-contaminated feed on laying hens' health, performance, and egg quality. Physical methods such as sorting, cleaning, and thermal processing can help remove or destroy mycotoxin-contaminated grains and feed ingredients, reducing the risk of exposure to laying hens [10]. However, these methods may not be effective in eliminating mycotoxins, particularly heat-stable compounds such as T-2 toxin, necessitating additional mitigation measures.

Chemical detoxification agents such as binders and adsorbents are commonly used to mitigate mycotoxin contamination in feed [63]. These agents work by binding to mycotoxins in the gastrointestinal tract of laying hens, and other animal species as well, preventing their absorption and reducing their toxic effects [64]. Binders such as activated carbon, bentonite clay, and hydrated sodium calcium aluminosilicate (HSCAS) have been shown to effectively reduce the bioavailability of mycotoxins in poultry, improving feed safety and performance. Additionally, the use of mycotoxin-degrading enzymes and microbial biotransformation products offers promising strategies for detoxifying contaminated feed and reducing mycotoxin levels in laying hens [2].

Nutritional strategies such as dietary supplementation with antioxidants, vitamins, and minerals can help mitigate the adverse effects of mycotoxin exposure on laying hens' health and performance [65]. Antioxidants such as vitamin E, vitamin C, and selenium can help reduce oxidative stress and inflammation induced by mycotoxins, protecting the poultry's immune system and organ function. Additionally, dietary supplementation with mycotoxin-binding agents such as yeast cell walls, mannan oligosaccharides, and beta-glucans can help improve gut health, nutrient absorption, and overall resilience to mycotoxin exposure [66].

Biosecurity measures are also essential components of post-harvest mitigation strategies, aiming to prevent the introduction and spread of mycotoxin-producing fungi in poultry facilities [67]. Strict hygiene practices, including cleaning and disinfection of feed storage areas, equipment, and facilities, can help minimize the risk of mold growth and mycotoxin contamination. Proper management of feed storage and distribution can help prevent cross-contamination of feed batches and minimize the exposure of laying hens to mycotoxin-contaminated feed [68].

Mitigation strategies for trichothecenes in laying hens production involve a comprehensive approach that addresses both pre-harvest and post-harvest factors [69]. Preventing mycotoxin contamination in feed ingredients through good agricultural practices, monitoring, and testing programs is essential for minimizing the risk of exposure to laying hens. Additionally, post-harvest mitigation measures such as physical, chemical, and nutritional interventions, along with biosecurity measures, are critical for minimizing the impact of mycotoxin-contaminated feed on poultry health, performance, and egg quality [11]. By implementing these strategies effectively, producers can ensure the safety, welfare, and productivity of laying hens and maintain the quality and marketability of table eggs for consumers.

7. Conclusion

The T-2 toxin in laying hens production is multifaceted and encompasses issues related to poultry health, egg quality, and food safety. T-2 toxin contamination can impair laying hens' health and performance, increase the risk of disease outbreaks, and affect the quality and safety of eggs produced. Addressing these problems requires a comprehensive approach that focuses on preventing mycotoxin contamination, monitoring and testing for T-2 toxin levels, and implementing effective mitigation strategies to ensure the safety and sustainability of laying hens production.

The metabolism of T-2 toxin in poultry is a complex process involving phase I and phase II biotransformation reactions that result in the formation of metabolites with altered toxicity and excretion profiles. Understanding the metabolism of T-2 toxin in poultry is essential for assessing its toxicological effects, developing effective mitigation strategies, and ensuring the safety of poultry products for human consumption. Further research is needed to elucidate the metabolic pathways of T-2 toxin in poultry and identify biomarkers of exposure and toxicity that can be used for risk assessment and monitoring purposes. Additionally, efforts to minimize T-2 toxin contamination in

feed and improve poultry management practices are critical for reducing the risk of T-2 toxin exposure and protecting poultry health and welfare.

The influence of T-2 toxin on table egg quality is a significant concern in poultry production, as it can affect shell integrity, yolk color, albumen consistency, and sensory attributes of eggs. T-2 toxin contamination can lead to thin, fragile eggshells, paler yolks, abnormal albumen consistency, off-flavors, and odors, reducing the marketability and consumer acceptance of table eggs.

Mitigating T-2 toxin contamination requires comprehensive strategies that address both pre-harvest and post-harvest factors, with a focus on preventing mycotoxin contamination in feed ingredients and implementing effective detoxification methods to ensure the safety, quality, and marketability of table eggs.

Conflicts of Interest: The authors declare no conflict of interest.

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