Mycotoxin Contamination in Cattle Feeds from Agricultural Sources: Hepatotoxic and Immunosuppressive Effects, and Integrated Control Strategies

Islam alkhafaji[1], Assyaad Alyasari[2] , Eman Jawad Jabbir[1], Tuqa Sabbar Rahi[1]

[1] College of Veterinary medicine, University of Karbala, Karbala, Iraq.
[2] Agriculture college, University of Karbala, Karbala, Iraq.

Corresponding author: **Islam.j@uokerbala.edu.iq , Assyaad.a@s.uokerbala.edu.iq** , tuqa.s@uokerbala.edu.iq

Received: 9/10/2025 **Accepted:** 27/10/2025 **Published:** 15/3/2026

***Abstract*—** This review aims to summarize and evaluate recent findings on mycotoxin contamination of livestock feed, with a particular focus on its hepatotoxicity and immunosuppressive impacts. It is a comprehensive study that will help veterinarians, researchers, and agricultural stakeholders improve livestock health and feed safety. Fungal contamination of livestock feed ingredients poses a serious threat to public health and livestock productivity, as well as significant economic losses in the agricultural sector. Contamination of food crops with fungi and mycotoxins is common due to frequent contact with them, exacerbating critical health problems and leading to economic losses in the livestock sector itself, in addition to preventing the consumption of meat and dairy products due to the transfer of these toxins to humans. Commonly used feed ingredients, such as roughage, straw, corn, palm leaves, and vegetable waste, are often contaminated with aflatoxins, fumonisins, and ochratoxin A. The severity of these toxins in animals varies depending on the dose, duration of exposure, species, breed, diet, and nutritional status. In general, calves are more susceptible than older animals. These toxins are known to cause liver damage and suppress immune function, resulting in reduced feed intake, poor feed conversion, reduced weight gain, increased susceptibility to disease (due to impaired immunity), and reduced reproductive capacity. The main pathological consequences include liver damage, immunosuppression, and reduced production efficiency. Clinical indicators, including elevated liver enzyme levels and decreased immune cell counts, provide a deeper understanding of the risks associated with chronic exposure. Finally, improved agricultural practices, periodic feed testing, and veterinary interventions tailored to the specific environmental and agricultural conditions of this condition are integrated pest control strategies.

Keywords — Mycotoxins, Cattle feeds, Agricultural sources, Hepatotoxicity, Immunosuppression, Integrated control strategies

INTRODUCTION

Livestock production is vital to Iraq's agricultural economy, as cattle provide a major source of milk, meat, and revenue for rural people. Cattle nutrition in Iraq is highly reliant on plant-based diets such as maize, barley, wheat bran, and crop residue. However, these diets are extremely sensitive to fungal contamination, both before and after harvest. Poor harvesting practices, inadequate drying, and improper storage under hot and seasonally humid meteorological conditions produce suitable habitats for fungus growth and mycotoxin manufacturing, a key worry raised for the region by international agencies. (1)

Above 100 species of fungi that can soil plants and forages and form mycotoxins. Mycotoxins are mainly created by *Aspergillus, Penicillum and Fusarium genera* which attack crops in the field and may grow during storage under satisfactory conditions of temperature and humidity. (2) *Aspergillus flavus* (aflatoxins), *Fusarium verticillioides* (fumonisins) fungi are among the most frequently detected in cattle feeds, as well as *Penicillium verrucosum* (ochratoxin A). Some Iraqi studies have registered obvious contamination of maize and barley with aflatoxin B_1 and fumonisins this raises greater concerns about food safety and the resulting impact on animal health. (3)

The hepatic and immunological disturbances are the dual danger in veterinary internal medicine so after consumption infected plant various mycotoxins are absorbed by the gastrointestinal system than transported to the liver via the portal vein, where they are metabolite. Such as Aflatoxin B_1 converted into active epoxide that linked to cellular macromolecules to result hepatocellular necrosis,

oxidative stress, and leak protein synthesis (4,5). Long term contact can lead to fatty liver, abnormal enzyme activity, and reduced metabolic efficiency.

The second disturbance of mycotoxins is immunosuppressive effects. Both humoral and cell-mediated immunity can be impairing, where notice reducing antibody production and inhibiting T-lymphocyte proliferation (6). The cattle susceptibility to secondary infections can be rising due to immunosuppression, as well as vaccine efficacy, and ultimately compromises productivity also can be reducing.

Linking the agricultural and veterinary aspects of this problem, with the aim of preventing the contamination of agricultural crops with these toxins, requires an integrated approach based on improving crop management, providing good storage conditions, periodic feed analysis, and the use of mycotoxin inhibitors in ruminant feed. This relies on the efforts of agricultural scientists, veterinarians, and policymakers to protect livestock health and productivity under varying climatic and economic conditions. Therefore, this research seeks to clarify the main sources of mycotoxins in livestock feed, their hepatotoxic and immunosuppressive effects, and integrated management techniques, while providing a scientific framework for local stakeholders to mitigate the associated risks.

''Literature Review Methods''

To conduct a comprehensive and up-to-date analysis of the available literature, a systematic approach was used to identify, select, and synthesize relevant scientific studies. The methodology for this narrative review is outlined below.

- Literature Search Strategy: A comprehensive search of electronic academic databases was conducted to collect relevant literature. The main databases consulted included Google Scholar, PubMed, Science Direct, and Research Gate. These platforms were chosen for their broad coverage of veterinary and agricultural sciences and toxicology.
- Keywords: The search used a combination of keywords and Boolean operators to refine the results. The main search terms were: "mycotoxins," "aflatoxins," "fumonisins," "ochratoxins," "cattle feed," "hepatotoxicity," "immunosuppression," "cattle," "feed contamination," and "mycotoxin control strategies."
- Timeframe and Scope: The search primarily focused on literature published between 2010 and 2025 to ensure the inclusion of the latest developments and current data. However, older, closely related publications were not excluded to provide the necessary foundational context, particularly regarding established toxicological mechanisms.
- Inclusion and Exclusion Criteria: Studies were selected based on the following criteria:
- Inclusion Criteria: 1. Original research articles and review papers; 2. Studies focusing on common mycotoxins in cattle feed (e.g., aflatoxins, fumonisins, ochratoxins, and trichothecenes); 3. Research investigating hepatotoxic or immunosuppressive effects in cattle; 4. articles related to the Middle East region, including Iraqi articles; 5. publications in English.
- Exclusion criteria: (a) studies not directly related to livestock or feed; (b) articles for which full text could not be obtained; (c) non-reviewed publications.

The final selected literature was evaluated to ensure a comprehensive overview of the sources, incidence, toxic kinetics, pathological effects, and integrated management of mycotoxins in livestock**.**

"Structure of the Literature Review"

1. Sources and Occurrence of Mycotoxins in Cattle Feed

1.1. Primary Agricultural Sources of Contamination:

The vital components of cattle rations are habitually the most exposure to pollution. Corn (maize) is a main substrate for *Fusarium* species, because of contamination with deoxynivalenol (DON), zearalenone (ZEN), and fumonisins (FB1). In the same way, cottonseed and cottonseed meal are habitually soiled by aflatoxins (AFB1) produced by *Aspergillus flavus*. (7) Oilseed meals as soybean meal can also be soiled, while small grains (wheat, barley) and their associated forages and silages are common sources of DON and other trichothecenes (T-2 toxin, HT-2 toxin). (8) Silage, if poorly fermented, provides an ideal environment for fungal growth and mycotoxin production. *"see Fig. 1."*

Figure 1. Food and feed usually associated with mycotoxins. (9)

1.2. Factors Influencing Mycotoxin Production:

The initiation of contamination in the field, where abundance stress, insect damage, and extreme weather events weaken plant defenses and enable fungal colonization. Post-harvest, suitable handling is a critical safety factor. Storage at high moisture content (>14-15%), great humidity, and raised temperatures stimulates the multiplying of storage fungi and the continuous production of mycotoxins. (10)

1.3. Global Prevalence and Co-Contamination Patterns:

Mycotoxin contamination is not an infrequent problem but a continuous hazard. Global investigations dependably show that over 70-80% of feed samples have quantifiable levels of at least one mycotoxin. (2) An earnest part is co-

contamination, where many mycotoxins are found simultaneously. This is predominantly regarding as mycotoxins can have further or synergistic effects, amplifying their poisonousness even at single low concentrations. (11)

2. Absorption, Distribution and Metabolism of Mycotoxins in Ruminants:

The unique gastrointestinal physiology of ruminants significantly influences the fate and toxicity of ingested mycotoxins, involving complex interactions of absorption, distribution, and metabolism (4).

1. Absorption: The process begins in the gastrointestinal tract. Mycotoxins are primarily absorbed in the rumen and the intestines. The rate and extent of absorption vary significantly depending on the specific mycotoxin's chemical properties, the composition of the diet, and the health of the rumen epithelium. Lipophilic mycotoxins, such as aflatoxins and ochratoxins, are generally more readily absorbed than hydrophilic ones.

2. Distribution: Once absorbed, mycotoxins enter the bloodstream and are distributed throughout the body via the portal vein, with the liver being the primary target due to its role as the first organ encountered post-absorption (12). The lipophilicity of many mycotoxins allows them to cross cell membranes and distribute to various tissues, including the kidneys, lungs, and immune organs, where they can exert their toxic effects.

3. Metabolism: Metabolism is a critical determinant of mycotoxin toxicity and occurs in two main sites:

- **Ruminal Mtabolism:** The ruminal microflora can metabolize certain mycotoxins, sometimes leading to detoxification. For example, deoxynivalenol (DON) can be partially converted to the less toxic de-epoxy DON (DOM-1). However, this microbial detoxification is often incomplete, strain-dependent, and can be compromised by high-concentrate diets that alter rumen function and reduce its detoxification capacity (13, 14).
- **Hepatic Metabolism:** The liver is the major site for the biotransformation of absorbed mycotoxins. This process can have dual outcomes:
  - **Bioactivation:** Some mycotoxins are metabolized into more toxic compounds. The most prominent example is Aflatoxin B_1 (AFB_1), which is activated by hepatic cytochrome P450 enzymes (e.g., CYP3A4) into the highly reactive and carcinogenic aflatoxin-8,9-epoxide. This metabolite is responsible for DNA damage and hepatocellular necrosis (15).
  - **Detoxification:** Conversely, the liver can also conjugate mycotoxins (e.g., via glucuronidation or glutathione conjugation) to form water-soluble, less toxic metabolites that are prepared for excretion **(**16).

The balance between activation and detoxification pathways in the liver, influenced by species, health status, and nutritional factors, ultimately determines the overall hepatotoxic and systemic impact of mycotoxin exposure. Furthermore, the carry-over of toxins like AFB1 into milk as Aflatoxin M1 (AFM1) is a direct result of this absorption and metabolism, posing a significant public health risk (17).

3. Hepatotoxic Effects: Mechanisms, Clinical Presentation, and Diagnosis:

The liver, as the primary detoxification organ, is a major target for many mycotoxins being particularly hepatotoxic.

3.1. Pathological outcomes of hepatotoxicity

A detailed understanding of the general mechanisms of hepatotoxicity is fundamental to elucidating the clinical and pathological features of liver injury caused by toxins. The liver's central role in the uptake, biotransformation, and biliary excretion of xenobiotics is crucial for systemic detoxification but also predisposes it to damage. (15) This is because hepatocytes are the first line of defense against ingested toxins, which arrive directly from the gastrointestinal tract via the portal venous system. (16)

While the general mechanisms of hepatotoxicity provide a foundational understanding, the specific pathological outcomes vary significantly depending on the mycotoxin involved.

- **Aflatoxin B_1 (AFB_1):** Its hepatotoxicity is primarily mediated by its bioactivation via cytochrome P450 enzymes (particularly CYP3A4 and CYP1A2) into the highly reactive exo-AFB_1-8,9-epoxide. This metabolite forms covalent adducts with cellular DNA and proteins, leading to DNA damage, mutations, and inhibition of protein synthesis. A crucial consequence is the significant depletion of glutathione (GSH), which exacerbates oxidative stress and lipid peroxidation, ultimately triggering apoptosis or necrosis of hepatocytes. (18) Chronic exposure disrupts lipid metabolism, leading to fatty liver and, potentially, hepatocellular carcinoma.
- **Fumonisins (FB_1):** The primary mechanism of fumonisin toxicity is the competitive inhibition of the enzyme ceramide synthase. This disruption in sphingolipid metabolism leads to an accumulation of bioactive sphingoid bases (like sphinganine) and a depletion of complex sphingolipids. The altered sphinganine-to-sphingosine ratio is a key biomarker. This imbalance promotes oxidative stress, disrupts cell membrane integrity, and induces apoptosis in hepatocytes, contributing to liver damage. (19)
- **Ochratoxin A (OTA):** While its primary target is the kidney, OTA also induces hepatotoxicity. Its mechanism involves inhibition of mitochondrial ATP production, leading to oxidative stress via the generation of reactive oxygen species (ROS). OTA also inhibits protein synthesis by competing with phenylalanine in the phenylalanyl-tRNA synthetase reaction. Furthermore, it can form DNA adducts, contributing to its genotoxic and carcinogenic potential. (20) *"see Fig. 2. and table .1."*

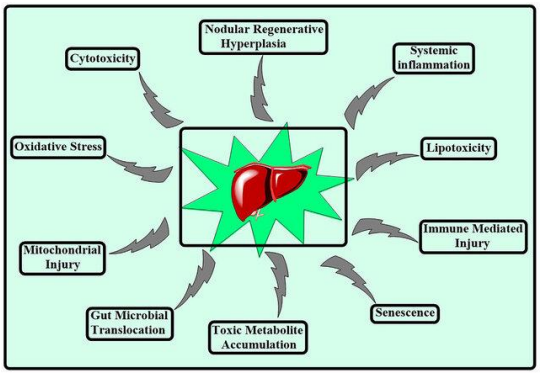

Figure 2. Factors which affect the hepatic cells and cause damage of hepatocytes. (21**)**

3.2. Clinical Manifestations and Pathological Lesions:

Plant- and fungal-associated hepatotoxicity in cattle manifests through a combination of acute and chronic clinical presentations, often with overlapping features that complicate diagnosis. Acute hepatotoxicity typically presents with in appetence, lethargy, decreased milk production, jaundice, and secondary (hepatogenous) photosensitization, especially in cattle exposed to high ultraviolet radiation without access to shade. Within hours of toxin ingestion, unpigmented skin regions may develop erythema, exudation, ulceration, and necrosis. (22,23)

In severe cases, hepatic encephalopathy may ensue, characterized by behavioral abnormalities such as depression, ataxia, aggression, muscle tremors, and terminal convulsions, particularly in the terminal stages of liver failure. (24) Chronic hepatotoxicity is commonly induced by toxins such as pyrrolizidine alkaloids, aflatoxin B1, phomopsins, and sporidesmin, and presents as ill-thrift, reduced feed intake, poor weight gain, and possibly jaundice or encephalopathy in advanced cases. (25,26)

Sudden death without preceding clinical signs, particularly when affecting large numbers of animals, suggests exposure to potent toxins such as microcystin from cyanobacteria, carboxyparquin from *Cestrum parqui*, or acute bovine liver disease (ABLD) toxins. (27)

Pathologically, hepatic damage is categorized into cholestatic injury, necroinflammation, steatosis, fibrosis/cirrhosis, and vascular lesions. Cholestatic injury—exemplified by pithomycotoxicosis—shows gross lesions including bile duct prominence, gallbladder distension, and green-yellow liver discoloration due to bile accumulation. (28) Microscopically, bile canaliculi dilation, hepatocyte bile pigment, Kupffer cell activity, biliary hyperplasia, and portal mononuclear infiltration are frequently observed. Gallbladder wall edema and cholecystitis may also be present. (29) Recognition of these integrated clinical, biochemical, and pathological findings is essential for accurate diagnosis and targeted management of hepatotoxic syndromes in cattle. “see Fig. 3.”

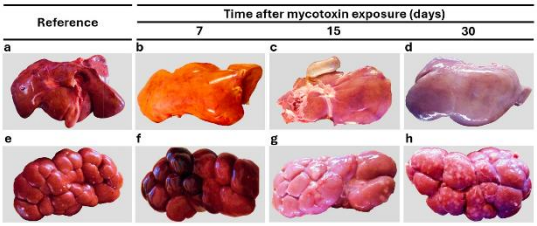

Figure 3. Characteristic morphological alterations in the liver. (a) Liver without gross lesions and with characteristic coloration. Nonexposed group. (b) Liver with yellowish discoloration and friable consistency. (c) Liver with multifocal hemorrhages and friable consistency. (d) Liver with pale discoloration and hemorrhagic foci. (30)

Biochemically, hepatotoxic injury leads to elevated hepatocellular (AST, GLDH) and cholestatic (GGT, ALP) enzymes, reflecting damage to both hepatocytes and cholangiocytes. (31) Toxins targeting hepatocytes—such as cycasin, carboxyparquin, and furanosesquiterpenoid oils—cause more pronounced hepatocellular enzyme elevation, while those affecting cholangiocytes—like sporidesmin and aflatoxin B1—primarily elevate cholestatic enzymes. (32)

In ruminants, ALT lacks diagnostic value due to naturally low hepatocellular concentrations, and AST elevations should be interpreted alongside CK to rule out muscle damage. (33) Chronic hepatic dysfunction may result in decreased BUN, hypoproteinemia, anemia of chronic disease, and hyperammonemia leading to encephalopathy. Additionally, GGT may remain elevated for months, even when clinical signs subside, and serum bile acids may offer greater sensitivity in ambiguous cases (34). *"As in table .1."*

Table 1. Common Mycotoxins in Cattle Feeds and Their Effects

Mycotoxin	Fungal Source	Primary Target Organ	Main Mechanism of Toxicity (Key Biochemical Lesion)	Clinical Signs & Pathological Lesions	Diagnostic Indicators (in cattle)	Common Feed Sources
Aflatoxin B₁	*A. flavus*	Liver	Bio activation to AFB₁-8,9-epoxide; DNA & protein adduct formation; Glutathione depletion; Severe oxidative stress; Inhibition of protein synthesis.	Jaundice, Hepatogenous photosensitization, Fatty liver, Reduced milk yield, Ill-thrift, Increased susceptibility to infections.	↑AST, ↑GGT, ↑GLDH, ↑Bile Acids; Hypoalbuminemia; Prolonged clotting time.	Maize, Cottonseed, Peanuts
Fumonisin B₁	*F. verticillioides*	Liver, Lungs, Kidneys	Inhibition of ceramide synthase; Disruption of sphingolipid metabolism (↑sphinganine/sphingosine ratio); Oxidative stress; Apoptosis.	Poor weight gain, Pulmonary edema (in high doses), Hepatocyte necrosis, Immunosuppression.	↑Liver enzymes (AST, GLDH); Altered sphinganine/sphingosine ratio in serum or tissues.	Corn, Maize silage
Ochratoxin A	*P. verrucosum*	Kidneys, Liver	Inhibition of mitochondrial ATP production; Oxidative stress (ROS generation); Inhibition of protein synthesis; DNA adduct formation.	Immunosuppression, Anorexia, Reduced growth, Nephrotoxicity (polyuria, polydipsia), Mild liver damage.	↑Urea, ↑Creatinine (primary); Mild ↑GGT; Proteinuria; Glucosuria.	Barley, Wheat, Oats
Deoxynivalenol (DON)	*Fusarium* spp.	Gastrointestinal, Immune System	Inhibition of protein synthesis (binds to ribosomes); Induction of pro-inflammatory cytokines ("ribotoxic stress response").	Feed refusal, Vomiting, Diarrhea, Immunosuppression, Necrosis of gastrointestinal mucosa.	Non-specific; Diagnosis relies on feed analysis. Clinical signs are primary indicators.	Wheat, Barley, Corn

4. Immunosuppressive Effects: Impacts on Herd Health:

Immunosuppression is arguably the most economically damaging effect of mycotoxins, as it increases susceptibility to a wide range of diseases.

4.1. Impact on Innate and Adaptive Immunity:

Mycotoxins like DON and AFB1 suppress the immune system through multiple pathways. They inhibit protein synthesis in immune cells, impairing the production of antibodies, cytokines, and complement proteins. (35) They reduce the proliferative response of T-lymphocytes and B-lymphocytes to mitogens and antigens, weakening the adaptive immune response. Furthermore, they impair the phagocytic and killing capacity of macrophages and neutrophils, crippling the first line of defense (innate immunity). (36)

4.2. Consequences for Cattle Health and Productivity

This immunosuppression manifests as:

A. **Increased incidence of infectious diseases** like mastitis, metritis, and bovine respiratory disease (BRD) due to reduced resistance to pathogens.

B. **Vaccination failure**, as the animal fails to mount a proper protective antibody response following vaccination.

C. **Reactivation of latent infections** (e.g., bovine herpesvirus) and prolonged recovery times from illness.

5. Integrated Management Strategies for Risk Mitigation:

A single approach is insufficient; an integrated strategy from field to feed bunk is essential. *"see Fig. 4."*

5.1. Pre-Harvest Prevention Strategies:

This includes using fungal-resistant crop varieties, implementing good crop rotation and tillage practices, and effective insect control to minimize fungal entry points. (37) Biocontrol using a toxigenic strains of *A. flavus* to competitively exclude toxigenic strains has shown success in reducing AFB1 in crops. (38)

5.2. Post-Harvest and Storage Management:

Proper drying of grains to below 14% moisture and maintenance of clean, dry, and well-ventilated storage facilities are crucial to prevent post-harvest fungal growth. (39)

5.3. Detoxification and Interventional Strategies

When contamination occurs, several interventions can be employed:

- **Physical Methods:** Sorting, cleaning, and thermal processing can reduce but not eliminate mycotoxins.
- **Adsorbing Agents (Mycotoxin Binders):** These are added to feed to reduce bioavailability. Aluminosilicates (e.g., HSCAS ranges from **1% to 0.33% of the feed dry weight**) are effective against AFB1 but less so against other mycotoxins. (40) Yeast cell wall components (e.g., glucomannans in cattle feed range from **0.1% to 0.5% of dry matter**,) (41) and bacterial polymers -based additives are **0.1–0.3% of dry feed matter** in livestock diets, depending on the product and contamination level as broader spectrum of activity. (42)
- **Biological Detoxification:** This is an emerging field using specific enzymes or microorganisms (e.g., *Eubacterium* BBSH 797 for trichothecenes) to biotransform mycotoxins into non-toxic metabolites. (43)

5.4. Monitoring and Risk Assessment:

Regular feed testing using analytical methods (HPLC, LC-MS/MS) or rapid screening kits (ELISA) is essential for monitoring contamination levels and making informed management decisions. (44)

Figure 4: Control methods of mycotoxin contamination in grain and oil crops. (45)

''Future Perspectives and Conclusions''

Mycotoxin contamination in cattle feed represents a critical intersection between agricultural practices and veterinary health. Evidence from recent studies confirms that the combined hepatotoxic and immunosuppressive effects of mycotoxins significantly reduce animal productivity and compromise disease resistance. The persistence of contamination, often due to inadequate storage and climatic stress, underscores the need for continuous surveillance and preventive management. Integrating pre-harvest control, rapid feed diagnostics, and biological detoxification methods offers the most practical path for mitigation. Future directions should emphasize the use of innovative biocontrol agents and AI-based predictive systems to identify contamination risks early and optimize intervention strategies. This holistic approach is essential to ensure safe feed production, enhance animal welfare, and protect public health**.**

REFERENCES

1) Food and Agriculture Organization of the United Nations (FAO). (2020). "The Near East and North Africa Regional Overview of Food Security and Nutrition: Tackling challenges from climate change and conflicts

2) Mekuanint Gashaw (2015). Review on Mycotoxins in Feeds: Implications to Livestock and human health:

Journal of Agricultural Research and Development Vol. 5(3). pp. 0137-0144.

3) Moayad S. H. (2023). Detection Aflatoxin on the Imported Maize (Zea mays L) Using HPLC in the Areas of Baghdad. South Asian Research Journal of Biology and Applied Biosciences, 5(3), 55 59.

4) Lach, M., & Kotarska, K. (2024). Negative Effects of Occurrence of Mycotoxins in Animal Feed and Biological Methods of Their Detoxification: A Review. Molecules, 29(19), 4563.

5) Sui, Y., Lu, Y., Zuo, S., Wang, H., Bian, X., Chen, G., Huang, S., Dai, H., Liu, F., & Dong, H. (2022). Aflatoxin B_1 Exposure in Sheep: Insights into Hepatotoxicity Based on Oxidative Stress, Inflammatory Injury, Apoptosis, and Gut Microbiota Analysis. Toxins, 14(12), 840.

6) Ramona M. O, Nicodim F., Cosmina M. B. and George Cosmin N. (2025)."Mycotoxins in Broiler Production: Impacts on Growth, Immunity,Vaccine Efficacy, and Food Safety" Toxins, 17(6),126.

7) Eckard, S., Wettstein, F. E., Forrer, H. R., & Vogelgsang, S. (2011). Incidence of Fusarium species and mycotoxins in silage maize. Toxins, 3(8), 949-967.

8) Kosicki, R., Błajet-Kosicka, A., Grajewski, J., & Twaruzek, M. (2016). Multiannual mycotoxin survey in feed materials and feedingstuffs. Animal Feed Science and Technology, 215, 165-180.

9) Fernandes, T., Ferrão, J., Chabite, I., Guina, J., Garrine, C., & Bell, V. (2021). Impact of Toxigenic Fungi: An Economic and Health Obstruction to Wellness in Mozambique. European Journal of Agriculture and Food Sciences, 3(5),

10) Awuchi, Chinaza Godswill; Awuchi, Chibueze Gospel; Ukpe, Aniefiok Emem; Asoegwu, Chisom Rachael; Uyo, Chijioke Ndukwe; Ngoka, Kelechi Enyinnaya (2020). Environmental Impacts of Food and Agricultural Production: A Systematic Review. European Academic Research. 8 (2): 1120 – 1135.

11) Zhu, Z., Cheng, H., Wang, J., Ma, J., Wang, J., Wang, H., Zhou, X., & Yang, J. (2025). Occurrence of co-contamination and interaction of multi-mycotoxins in dairy cow feed in China 17(3), 137.

12) Abidin ZU and A Khatoon, (2012). Ruminal microflora, mycotoxin inactivation by ruminal microflora and conditions favouring mycotoxicosis in ruminants: A Review. Inter J Vet Sci, 1(1): 37-44.

13) Ogunade, I. M., Martinez-Tuppia, C., Queiroz, O. C. M., Jiang, Y., Drouin, P., Wu, F., Vyas, D., & Adesogan, A. T. (2018). Silage review: Mycotoxins in silage: Occurrence, effects, prevention, and mitigation. Journal of Dairy Science, 101(5), 4034–4059.

14) Upadhaya, S. D., Park, M. A., & Ha, J. K. (2010). Mycotoxins and their biotransformation in the rumen: A review. Asian-Australasian Journal of Animal Sciences, 23(9), 1250-1260 .

15) Wang, L., Shao, Z., Wang, X., Lu, W., & Sun, H. (2025). Xenobiotic-induced liver injury: Molecular mechanisms and disease progression. Ecotoxicology and Environmental Safety, 303, 118854.

16) Jordi Y. Zhou . (2023). Innate immunity and early liver inflammation. Frontiers in Immunology, 14, 1175147.

17) Iha, M. H., Barbosa, C. B., Okada, I. A., & Trucksess, M. W. (2016). Mycotoxins in Bovine Milk and Dairy Products: A Review. Journal of Food Science, 81(3), R544–R552.

18) Jaeschke, H., Gores, G. J., Cederbaum, A. I., Hinson, J. A., Pessayre, D., & Lemasters, J. J. (2002). Mechanisms of hepatotoxicity. Toxicological Sciences, 126(1), 6–35.

19) Stoev, S. D. (2017). Foodborne mycotoxicoses, risk assessment and underestimated hazard of masked mycotoxins and joint mycotoxin toxicity or interaction. Environmental Toxicology and Pharmacology, 55, 1-10.

20) Kőszegi, T., and Miklós P (2016). Ochratoxin A: Molecular interactions, mechanisms, and toxicity. Environmental Toxicology and Pharmacology, 15;8(4):111.

21) Thakur, S., Kumar, V., Das, R., Sharma, V., & Mehta, D. K. (2024). Biomarkers of hepatic toxicity: An overview. Current Therapeutic Research, 100, 100737.

22) Patel, K.A., Bhoi D.B. and Raval J.K. (2022). Photosensitization and its management in Livestock Journal of Livestock Science (ISSN online 2277-6214) 13: 188-193.

23) Melissa. S and Yonaira .C (2013). "Hepatic encephalopathy: etiology, pathogenesis, and clinical signs" Compend Contin Educ Vet;35(6): E1-8; quiz E9.

24) Vladimir. Po., Olga. P., Anastasia K. and Ludmila.A. (2024). Etiology and pathogenesis of non-infectious hepatopathy in cattle under industrial production. E3S Web of Conf., VOL 510: 01026.

25) Lohse, J. C., Paredes, E., González, C. M., Koene, M., & Mageed, M. (2018). Pyrrolizidine alkaloid toxicosis and hepatic encephalopathy in horses in Easter Island, Chile. Austral Journal of Veterinary Sciences, 50(2), 107–110.

26) EFSA Panel on Contaminants in the Food Chain (CONTAM). (2020). Scientific Opinion on the risks to public health related to the presence of aflatoxins in food and feed. EFSA Journal, 18(3), 6037.

27) Eve M. Manthorpe,1 Ian V. Jerrett, Grant T. Rawlin, Lucy Woolford (2021). Clinical and pathologic features of acute bovine liver disease in Australia. Journal of Veterinary Diagnostic Investigation, Vol. 33(5) 875–883.

28) Michelle S. Mostrom (2024). Facial Eczema in Animals. Veterinary Diagnostic Laboratory ToxicologyReviewed/Revised Nov 2021 | Modified Sept 2024

29) Manthorpe, E. M., Jerrett, I. V., Rawlin, G. T., & Woolford, L. (2020). Plant and fungal hepatotoxicities of cattle in Australia, with a focus on minimally understood toxins. Toxins, *12*(11), 707.

30) de Luna-López, M. C., Valdivia-Flores, A. G., Rangel-Flores, E. J., & Ortiz-Muñoz, R. (2025). Time-Dependent Changes in Performance, Biochemistry, and Histology in Dairy Calves with Acute Aflatoxicosis. Vet. Sci. , 12(3), 273.

31) Mary, V. S., Valdehita, A., Navas, J. M., Rubinstein, H. R., & Fernández-Cruz, M. L. (2015). Effects of aflatoxin B1, fumonisin B1 and their mixture on the aryl hydrocarbon receptor and cytochrome P450 1A induction. Food and Chemical Toxicology, 75, 104–111.

32) Hadžimusić, N., & Hadžijunuzović-Alagić, Dž. (2025). Liver enzyme profiles in high-yielding dairy cows: Indicators of metabolic health. Sciences of Europe, (161), 54–61.

33) Kalugniy, I. I., Markova, D. S., Yashin, A. V., Prusakov, A. V., Ponamarev, V. S., & Andreeva, N. L. (2021). Diagnosis of hepatopathy in Holstein cattle with metabolic disorders. IOP Conference Series: Earth and Environmental Science, 723(2),

34) Merck Veterinary Manual. (2023). Hepatic function tests. In The Merck Veterinary Manual. Merck & Co., Inc.https://www.merckvetmanual.com/clinicalpathology-and-procedures/liver-function tests/hepatic-function-tests

35) Pestka, J. J. (2010). Toxicological mechanisms and potential health effects of deoxynivalenol exposure. Archives of Toxicology, 84(9), 663-679.

36) Oswald, I. P., Marin, D. E., Bouhet, S., Pinton, P., Taranu, I., & Accensi, F. (2005). Immunotoxicological risk of mycotoxins for domestic animals. Food Additives & Contaminants, 22(4), 354-360.

37) Mshelia, L. P., Selamat, J., & Samsudin, N. I. P. (2020). Strategies for controlling and decontaminating mycotoxins in foods and feeds: A review. Food Control, 116.

38) Khan, R., Ghazali, F. M., Mahyudin, N. A., & Samsudin, N. I. P. (2021). Biocontrol of Aflatoxins Using Non-Aflatoxigenic Aspergillus flavus: A Literature Review. Toxins, 13(2), 154.

39) Magan, N.; Aldred, D. (2007) Post-harvest control strategies: Minimizing mycotoxins in the food chain. Int. J. Food Microbiol., 119, 131–139.

40) ngh, R., Saini, A. K., Dhial, K., & Pathak, A. (2024). Effectiveness of hydrated sodium calcium aluminosilicate (HSCAS) in mitigating the adverse effects of aflatoxin on in vitro rumen fermentation of wheat straw. Indian Journal of Veterinary Science and Biotechnology, 20(5), 121–125.

41) Jouany, J. P. (2005). How yeast cell wall components can alleviate mycotoxicosis in animal production and improve the safety of edible animal products. Journal of Animal and Feed Sciences, 14(2), 1–11

42) Kholif, A. E., Anele, A., & Anele, U. Y. (2024). Microbial feed additives in ruminant feeding. MS Microbiology, 10(3), 542–571.

43) Saricaoglu, B., Subaşı, B. G., Karbancioglu-Guler, F., Lorenzo, J. M., & Capanoglu, E. (2023). Phenolic compounds as natural microbial toxin detoxifying agents. Toxicon, 222,

44) Muñoz-Solano, B., Lizarraga Pérez, E., & González-Peñas, E. (2024). Monitoring mycotoxin exposure in food-producing animals (cattle, pig, poultry, and sheep). Toxins, 16(5), 218.

45) Zhang C, Qu Z, Hou J, Yao Y. (2024). Contamination and Control of Mycotoxins in Grain and Oil Crops. Microorganisms.;12(567):567.