

International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
ISSN Online: 2617-4707
NAAS Rating (2026): 5.29
IJABR 2026; 10(6): 1052-1054
www.biochemjournal.com
Received: 01-04-2026
Accepted: 04-05-2026

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Veterinary aflatoxicosis: A comprehensive review

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DOI: <https://www.doi.org/10.33545/26174693.2026.v10.i6m.8819>

Abstract

Aflatoxicosis is one of the most important mycotoxicoses affecting livestock, poultry, companion animals, and wildlife worldwide. It results from exposure to aflatoxins, a group of toxic secondary metabolites produced primarily by *Aspergillus flavus* and *Aspergillus parasiticus*. Contamination of feed ingredients such as maize, groundnut cake, cottonseed cake, and other cereals poses a significant challenge to animal production systems, particularly in tropical and subtropical regions. Aflatoxins adversely affect animal health by inducing hepatotoxicity, immunosuppression, growth retardation, reproductive disorders, and increased susceptibility to infectious diseases. The severity of intoxication depends on factors such as the dose and duration of exposure, species, age, nutritional status, and environmental conditions. Poultry, young animals, and certain fish species are particularly susceptible to aflatoxin toxicity. In addition to causing substantial economic losses through reduced productivity and increased mortality, aflatoxins also pose a public health concern because of their carry-over into animal-derived products such as milk, meat, and eggs. This review summarizes the etiology, occurrence, chemistry, epidemiology, and pathogenesis of veterinary aflatoxicosis, highlighting current knowledge on its mechanisms of toxicity and implications for animal health.

Keywords: Aflatoxins, aflatoxicosis, mycotoxins, livestock, poultry, hepatotoxicity, feed contamination

Introduction

Mycotoxins are naturally occurring toxic compounds produced by certain species of fungi growing on agricultural commodities before harvest, during processing, or in storage. Among the various mycotoxins identified to date, aflatoxins are considered the most significant because of their widespread occurrence, potent toxicity, and substantial impact on animal and human health (Bennett and Klich, 2003) [2]. Aflatoxins are predominantly produced by *Aspergillus flavus*, *Aspergillus parasiticus*, and, less frequently, *Aspergillus nomius* under conditions of high temperature and humidity (Klich, 2007) [9].

The term "aflatoxin" originated from the fungus *Aspergillus flavus* following the outbreak of "Turkey X disease" in England in 1960, which caused the death of more than 100,000 turkey poulters fed contaminated peanut meal (Blount, 1961) [3]. Since then, aflatoxins have been recognized as major contaminants of animal feed worldwide. Their occurrence is particularly common in tropical and subtropical countries where climatic conditions favor fungal growth and toxin production (Williams *et al.*, 2004) [15].

Veterinary aflatoxicosis refers to the disease condition resulting from ingestion of aflatoxin-contaminated feed by animals. The disease may occur in acute, subacute, or chronic forms depending on the concentration of toxin consumed and duration of exposure (Hussein and Brasel, 2001) [7]. Acute intoxication is characterized primarily by severe hepatic injury, hemorrhages, and mortality, whereas chronic exposure leads to reduced productivity, impaired immunity, poor growth performance, and reproductive dysfunction (Osweiler, 2005) [12].

The economic consequences of aflatoxicosis are extensive and include decreased feed efficiency, reduced weight gain, lower milk and egg production, veterinary treatment costs, condemnation of animal products, and restrictions on international trade. Furthermore, the conversion of aflatoxin B₁ into aflatoxin M₁ in milk creates an important food safety concern for consumers (Battacore *et al.*, 2010) [1].

Given the increasing challenges posed by climate change, globalization of feed trade, and intensification of animal production systems, aflatoxicosis continues to represent a significant threat to animal agriculture. Therefore, understanding its etiology, occurrence, and mechanisms of toxicity is essential for developing effective preventive and control strategies.

Etiology and Sources of Aflatoxins

Aflatoxins are toxic secondary metabolites synthesized mainly by *Aspergillus flavus* and *Aspergillus parasiticus*. These fungi are widely distributed in soil, decaying vegetation, stored grains, and feedstuffs. Environmental factors such as temperature between 25 °C and 35 °C, high relative humidity, insect damage, and poor storage conditions favor fungal proliferation and aflatoxin production (Klich, 2007)^[9].

The principal aflatoxins of veterinary importance include:

- Aflatoxin B₁ (AFB₁)
- Aflatoxin B₂ (AFB₂)
- Aflatoxin G₁ (AFG₁)
- Aflatoxin G₂ (AFG₂)

Among these, AFB₁ is the most prevalent and toxic compound. Metabolic derivatives such as aflatoxin M₁ and M₂ may appear in milk and other biological fluids following ingestion of contaminated feed (Battacone *et al.*, 2010)^[11].

Common feed ingredients susceptible to aflatoxin contamination include:

- Maize (corn)
- Groundnuts (peanuts)
- Cottonseed cake
- Soybean meal
- Rice bran
- Sorghum
- Wheat
- Sunflower cake
- Copra meal

Contamination may occur at multiple stages, including crop growth, harvesting, transportation, storage, and feed manufacturing. Inadequate drying, mechanical damage to grains, insect infestation, and moisture accumulation are major factors contributing to toxin development (CAST, 2003)^[5].

Climate change has emerged as an additional risk factor. Elevated temperatures and drought stress predispose crops to fungal colonization and increased aflatoxin synthesis, leading to greater contamination of feed resources worldwide (Medina *et al.*, 2014)^[10].

Classification and Chemistry of Aflatoxins

Aflatoxins belong to a group of difuranocoumarin compounds derived from polyketide biosynthesis. They are chemically stable molecules capable of withstanding many feed-processing procedures, making their elimination difficult once contamination occurs (Rushing and Selim, 2019)^[14].

Based on fluorescence characteristics under ultraviolet light, aflatoxins are classified into two major groups:

B-Series

- Aflatoxin B₁

- Aflatoxin B₂

These compounds emit blue fluorescence and are commonly produced by *A. flavus* and *A. parasiticus*.

G-Series

- Aflatoxin G₁
- Aflatoxin G₂

These toxins produce green fluorescence and are predominantly synthesized by *A. parasiticus*.

Among all aflatoxins, AFB₁ exhibits the highest biological activity and carcinogenic potential. Following absorption, it undergoes biotransformation by hepatic cytochrome P450 enzymes to form reactive metabolites, particularly aflatoxin B₁-8,9-epoxide, which is responsible for most toxic effects (Guengerich *et al.*, 1998)^[6].

The physicochemical stability of aflatoxins allows them to persist during storage and processing. Consequently, contaminated feed may remain hazardous even after prolonged periods if appropriate detoxification measures are not implemented.

Epidemiology

Aflatoxicosis has been reported in nearly all domesticated animal species, including poultry, cattle, buffaloes, sheep, goats, pigs, horses, dogs, rabbits, and fish. However, susceptibility varies considerably among species and age groups (Osweiler, 2005)^[12].

Young animals are generally more vulnerable than adults because of immature detoxification mechanisms and higher metabolic demands. Poultry, especially ducklings and turkey poults, exhibit marked sensitivity to aflatoxin exposure. Swine and calves are moderately susceptible, whereas adult ruminants demonstrate relative resistance due to microbial degradation of toxins in the rumen (Hussein and Brasel, 2001)^[7].

Several outbreaks of aflatoxicosis have been documented worldwide, particularly in regions characterized by warm and humid climates. Developing countries often experience higher incidence rates because of inadequate feed storage infrastructure and limited monitoring programs (Williams *et al.*, 2004)^[15].

Risk factors associated with aflatoxin contamination include:

- High environmental humidity
- Elevated storage temperatures
- Prolonged storage periods
- Mechanical grain damage
- Insect infestation
- Drought stress before harvest
- Poor feed management practices

Seasonal variation also influences toxin occurrence, with contamination levels generally increasing during periods of high rainfall and humidity.

Pathogenesis and Mechanism of Toxicity

The toxic effects of aflatoxins are primarily mediated through metabolic activation in the liver. After ingestion, aflatoxins are rapidly absorbed from the gastrointestinal tract and transported to the liver via the portal circulation (Guengerich *et al.*, 1998)^[6].

Within hepatocytes, cytochrome P450 enzymes convert AFB₁ into highly reactive metabolites, particularly aflatoxin B₁-8,9-epoxide. This metabolite binds covalently to cellular macromolecules including DNA, RNA, proteins, and membrane phospholipids, resulting in cellular dysfunction and tissue injury (Rawal *et al.*, 2010) ^[13].

Major pathogenic mechanisms include

Hepatotoxicity

The liver is the principal target organ because it is the primary site of toxin metabolism. Hepatic injury may manifest as:

- Fatty degeneration
- Hepatocellular necrosis
- Bile duct proliferation
- Fibrosis
- Cirrhosis

Severe damage can lead to liver failure and death in acute cases.

Oxidative Stress

Aflatoxins increase the generation of reactive oxygen species (ROS), resulting in lipid peroxidation, mitochondrial dysfunction, and depletion of antioxidant defenses such as glutathione, superoxide dismutase, and catalase (Rawal *et al.*, 2010) ^[13].

Immunosuppression

One of the most important consequences of chronic aflatoxin exposure is suppression of immune function. Aflatoxins impair macrophage activity, reduce antibody production, inhibit lymphocyte proliferation, and decrease resistance to infectious diseases (Meisssonier *et al.*, 2006) ^[11].

Impaired Protein Synthesis

Binding of aflatoxin metabolites to nucleic acids interferes with transcription and translation processes. Consequently, protein synthesis declines, contributing to poor growth, reduced productivity, and tissue degeneration.

Carcinogenic and Mutagenic Effects

Aflatoxin B₁ can form DNA adducts that induce mutations and chromosomal abnormalities. Long-term exposure has been associated with hepatocellular carcinoma in several animal species and humans (IARC, 2012) ^[8].

Reproductive and Developmental Effects

Chronic intoxication may adversely affect reproductive performance by reducing fertility, impairing embryonic development, increasing embryonic mortality, and causing teratogenic effects in susceptible species (Bryden, 2012) ^[4]. Overall, the pathological manifestations of aflatoxicosis result from a combination of hepatocellular damage, oxidative stress, immunosuppression, and interference with essential metabolic processes.

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