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MYCOTOXIN CONTAMINATION IN AQUACULTURE SYSTEMS: SOURCES, TOXICOLOGICAL EFFECTS AND IMPLICATIONS FOR FOOD SAFETY

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Abstract. Mycotoxins are toxic secondary metabolites produced by filamentous fungi that often contaminate plant-based feed ingredients used in aquaculture. The ongoing replacement of fishmeal and fish oil with cereals and oilseeds increases the risk of fish being chronically exposed to multiple mycotoxins. **This review aims** to provide a short overview of mycotoxin contamination in aquaculture systems. This review summarises the occurrence, exposure routes, toxicokinetics, and biological effects of the main regulated mycotoxins, such as aflatoxin B1 (AFB1), deoxynivalenol (DON), zearalenone (ZEN), fumonisins, and ochratoxin A (OTA), as well as emerging compounds, including beauvericin (BEA), enniatins (ENNs), and Alternaria toxins. Although environmental factors and post-harvest processes may also contribute, feed is identified as the primary contamination source. Mycotoxins show limited carry-over into fish tissues, with the liver being the main site of accumulation and biotransformation; meanwhile, muscle residues remain relatively low, yet still relevant in terms of food safety. The toxic effects are multifactorial and include oxidative stress, apoptosis, immune dysregulation, endocrine disruption and metabolic disorders. There is increasing evidence highlighting the importance of co-exposure scenarios, where additive or synergistic interactions can enhance toxicity. The study of emerging and modified ('masked') mycotoxins remains insufficient, especially in vivo, which complicates risk assessment. **Conclusions:** Improved monitoring, a better understanding of combined toxicity, and the harmonisation of regulatory limits are essential. The development of effective mitigation strategies is necessary to ensure the sustainability of aquaculture production and food safety, particularly in the context of an increasing reliance on plant-based feeds.

Keywords: mycotoxins; aquaculture; fish health; oxidative stress; carry-over; food safety; mixture toxicity; risk assessment.

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Introduction. Mycotoxins are harmful secondary metabolites produced by filamentous fungi such as *Aspergillus*, *Fusarium*, *Penicillium* and *Alternaria*. While these compounds are not essential for fungal growth, they are

synthesised under favourable environmental conditions, particularly high humidity and temperature [1, 2]. Their biosynthesis is often triggered by plant stress factors, such as drought, insect damage and mechanical injury, which facilitate fungal colonisation in both the field and during storage. Mycotoxins frequently contaminate cereals and plant-derived raw materials used in food and feed production, posing risks to human and animal health. Notable mycotoxins include aflatoxins, ochratoxin A, fumonisins, trichothecenes (e.g. deoxynivalenol and T-2 toxin), and zearalenone. Many of these exhibit hepatotoxic, nephrotoxic, immunosuppressive, teratogenic or carcinogenic properties [3]. Importantly, exposure to mycotoxins is usually chronic and occurs at low doses, which complicates toxicological assessment and risk management strategies.

Globally, it is estimated that 60–80 % of major crops contain detectable levels of mycotoxins, with approximately 20–25 % exceeding the regulatory limits set by the European Union or the Codex Alimentarius for certain compounds [4]. The European Union has set maximum levels for certain mycotoxins in food and feed (e.g. Commission Regulation (EC) No 1881/2006 and Directive 2002/32/EC for undesirable substances in animal feed), reflecting their recognised significance in terms of public health. These regulatory frameworks are continuously updated in response to new toxicological evidence and emerging contamination patterns. In recent decades, climate change, inadequate post-harvest management, and suboptimal storage conditions have contributed to the increasing prevalence of contamination [5]. Rising global temperatures and extreme weather events are expected to alter the geographical distribution of toxigenic fungi, potentially leading to an increased presence of aflatoxins and other heat-related toxins in temperate regions. In addition to well-characterised mycotoxins, modified ('masked') and emerging forms are increasingly being reported, often escaping routine detection [6]. These modified forms may be hydrolysed during digestion, thereby contributing to total dietary exposure and complicating risk assessment. Therefore, advanced analytical techniques, such as LC-MS/MS-based multi-mycotoxin platforms, are increasingly required for accurate exposure assessment.

Aquaculture is one of the fastest-growing food production sectors worldwide, experiencing a marked increase in demand for formulated feeds. The gradual replacement of fishmeal and fish oil with plant-based ingredients such as maize, wheat, barley and soybean meal has introduced new contamination risks [7]. Plant-derived feed ingredients are particularly susceptible to contamination by trichothecenes and fumonisins produced by *Fusarium* species, as well as aflatoxins produced by *Aspergillus* species [8]. Consequently, mycotoxin contamination poses a dual threat, endangering the health and productivity of fish while raising concerns about its presence in edible tissues. Chronic exposure in fish has been linked to reduced growth, an impaired immune response, oxidative stress, and histopathological changes in liver and kidney tissues. It also increases susceptibility to infectious diseases, affecting the sustainability and food safety of aquaculture [9]. Furthermore, fish can serve as sensitive bioindicators of environmental and feed-borne mycotoxin contamination, rendering aquaculture systems valuable for broader ecotoxicological monitoring.

This review aims to provide a short overview of mycotoxin contamination in aquaculture systems. Particular emphasis is placed on the occurrence of mycotoxins, their exposure pathways, toxicokinetics and mechanisms of toxicity in fish. It also aims to evaluate the transfer of both regulated and emerging mycotoxins into fish tissues, and assess the implications for fish health, aquaculture productivity and food safety. Special attention is also given to mixture toxicity, modified ('masked') mycotoxins, and emerging compounds such as beauvericin, enniatins, and *Alternaria* toxins, which are insufficiently characterised and largely unregulated. Finally, this work seeks to identify current knowledge gaps and highlight priorities for future research and regulatory development in the context of the increasing reliance on plant-based feeds in aquaculture.

Routes of mycotoxin contamination in aquaculture. Mycotoxin contamination in aquaculture can occur via three main routes: (i) feed-borne contamination, which is the primary and most significant route; (ii) environmental exposure via contaminated water and sediments; and (iii) post-harvest contamination during the processing and storage of fish products. The relative contribution of each pathway depends on the production system (intensive versus extensive), feed formulation and environmental management practices.

Feed as the primary source. Commercial aquafeeds often contain cereal and plant-based ingredients that are susceptible to natural fungal colonisation. Raw materials such as maize, wheat, barley, soybean meal, and oilseed by-products are favourable substrates for toxigenic fungi, especially when drying and storage conditions are inadequate. Studies report that 75–100 % of compound feeds contain multiple mycotoxins simultaneously [9, 10]. Co-occurrence is common and can result in additive or synergistic toxic effects, even when the concentrations of individual toxins remain below regulatory limits. In freshwater species, wheat inclusion may reach 15–27 % in rainbow trout (*Oncorhynchus mykiss*) diets and up to 70 % in cyprinid feeds [11], thereby increasing exposure risk. The increasing use of plant proteins as a substitute for fishmeal in modern aquaculture further increases the likelihood of chronic, low-dose exposure to mycotoxin mixtures. Additionally, variability in the quality of raw materials across global supply chains contributes to inconsistent contamination profiles in commercial aquafeeds.

Within the EU, maximum levels for mycotoxins in aquafeeds are largely unregulated, except for the recommended limit of 10,000 µg/kg for fumonisins [12]. Guidance values also exist for certain toxins, such as deoxynivalenol (DON), zearalenone (ZEN), aflatoxin B1 and ochratoxin A, in complete feedingstuffs for fish. However, legal limits specific to aquaculture remain limited compared to those for terrestrial livestock. This regulatory gap is particularly relevant given the increasing reliance on plant-based feed ingredients in aquaculture systems. Multimycotoxin contamination, particularly involving *Fusarium*-derived toxins such as DON, ZEN, fumonisins, enniatins and beauvericin (BEA), is frequently reported [10, 13]. Emerging mycotoxins, including enniatins and beauvericin, are not routinely regulated despite being increasingly detected in aquafeeds.

Environmental and post-production exposure. Environmental contamination may occur through agricultural runoff or wastewater discharge.

DON and ZEN, for example, have been detected in surface waters [14]. Due to their chemical stability, mycotoxins can persist in aquatic environments and potentially accumulate in sediments, entering aquatic food webs. While bioaccumulation in fish tissues is generally considered to be limited for many mycotoxins, chronic low-level exposure can still occur through continuous contact with contaminated water and the ingestion of particulates. However, data on dermal or mucosal absorption in fish remain scarce. While waterborne exposure is generally considered less significant than dietary intake, it can still contribute to background contamination in intensive aquaculture systems [9].

Post-harvest contamination during the processing and storage of fish products. Post-production contamination may arise during the handling, drying, smoking or storage of fish products. Inadequate hygiene practices, high humidity and poor temperature control can encourage fungal growth on fish and processed products. Fungal genera such as *Aspergillus*, *Penicillium* and *Fusarium* have been identified in processed fish, particularly dried products stored in humid conditions [15–17]. Secondary contamination in such cases may result in the formation of aflatoxins, ochratoxin A or other storage-associated mycotoxins, which pose additional food safety concerns for consumers [9]. Ochratoxin A (OTA) is not well studied in aquatic species, but available data suggest reduced growth, poorer feed conversion, lower survival rates and haematological alterations in exposed fish [18]. Histopathological lesions in the liver and kidney have also been reported [19]. Although shrimp appear to be less sensitive in certain circumstances, higher doses and prolonged exposure can still result in adverse outcomes. This type of contamination is particularly relevant in small-scale and artisanal processing systems, where environmental control and HACCP-based procedures may be limited.

Occurrence and carry-over in freshwater fish. Freshwater fish species are particularly vulnerable to mycotoxin exposure due to the high proportion of cereal-based ingredients in their formulated diets. The main way they are exposed to mycotoxins is through chronic dietary intake, and the way they bioaccumulate them depends on their metabolism, lipid content, age, feeding regime and how long they are exposed to them for. Additionally, environmental and husbandry conditions (e.g. water temperature, dissolved oxygen and stocking density) can indirectly affect the capacity for detoxification and the overall toxicokinetics of fish [20]. Numerous experimental and field studies have investigated the transfer of aflatoxin B1 into edible muscle tissue and metabolically active organs such as the liver. The liver generally serves as the primary target organ for accumulation and biotransformation, whereas muscle tissue typically contains lower residue levels [2, 9].

Aflatoxins. Aflatoxin B1 (AFB1) is the mycotoxin that has been studied most extensively in aquatic species, largely due to its widespread occurrence, particularly in tropical regions, and its well-established hepatotoxic and carcinogenic properties [21]. Its effects have been documented in various species, including rainbow trout [22], Nile tilapia [23–25], channel catfish [26], rohu [27, 28], sea bass [21], gibel carp [29] and beluga [30].

The biological impact of AFB1 varies considerably among species and is closely linked to dietary concentration. Rainbow trout are among the most sensitive species, with tumour development reported at concentrations as low

as 0.4 µg/kg [22, 31], whereas channel catfish appear relatively resistant, exhibiting mainly reduced growth and moderate internal lesions at higher doses [31]. Similarly, European sea bass are sensitive, with acute aflatoxicosis observed at 180 µg/kg (LC₅₀) following short-term exposure [21]. In Nile tilapia and channel catfish, high dietary AFB1 levels (up to 1,880 and 10,000 µg/kg, respectively) reduce growth performance [31, 32], and mortality rates of up to 17% have been reported in tilapia at 2,000 µg/kg [33].

In addition to growth impairment, AFB1 induces a variety of clinical and pathological symptoms, such as cataracts, skin discolouration, lesions, fin erosion, abnormal swimming behaviour and reduced appetite [34]. Haematological disturbances, such as decreased erythrocyte and leukocyte counts and reduced haemoglobin levels, have also been reported [25]. At the cellular level, AFB1 toxicity is strongly associated with oxidative stress, DNA damage and disruption to protein synthesis. These factors collectively contribute to hepatocellular injury and carcinogenesis [34].

In addition to general toxicity, AFB1 can adversely affect reproduction in fish. Although studies are limited, available data indicate reduced ovary weight, fecundity, and egg size in gibel carp [29], as well as a decreased gonadosomatic index, poorer sperm quality, and lower estradiol-17β levels in Nile tilapia following chronic exposure [34]. These findings suggest that AFB1 may impair endocrine regulation and reproductive success, with potential long-term consequences for population dynamics in aquaculture systems (Fig. 1).

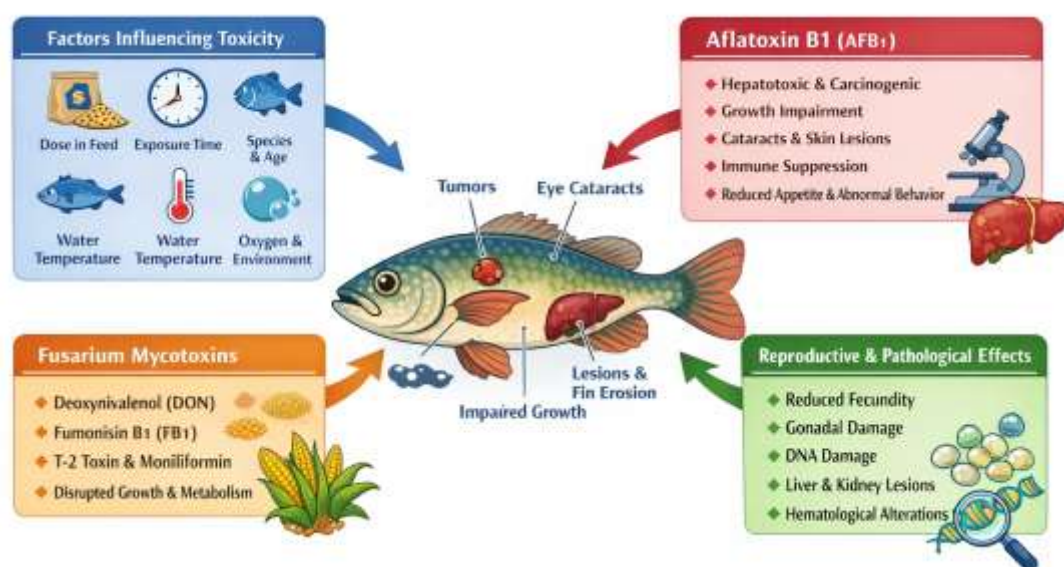


Fig. 1. Mechanisms of mycotoxin toxicity in fish: oxidative stress, metabolic disruption and physiological effects

Following exposure to contaminated feed, AFB1 has been detected in Nile tilapia (*Oreochromis niloticus*), African catfish (*Clarias gariepinus*), rohu (*Labeo rohita*) and sterlet sturgeon (*Acipenser ruthenus*) [32, 35–37]. Muscle concentrations have ranged from 0.05 to 78 µg/kg, reflecting differences in species sensitivity, exposure levels, feeding duration, and analytical methodology. Residue levels generally correlate with dietary concentration and exposure time, although depuration may occur after contaminated feed is

withdrawn [38–40]. Documented effects include hepatotoxicity, immunosuppression, reduced growth performance, oxidative stress, and histopathological liver alterations (including vacuolar degeneration and necrosis). Increased mortality has also been observed. AFB1 is metabolised in the fish liver to produce reactive intermediates, including aflatoxin M1, which can also be found in tissues and poses an additional food safety risk [41–43]. Although muscle residue levels are often low, even trace amounts are of toxicological concern due to the recognised carcinogenicity of AFB1. It has been classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC). Furthermore, differences in cytochrome P₄₅₀ activity between species may significantly impact the rate of AFB1 bioactivation and detoxification, thereby influencing residue distribution and toxicity outcomes.

Recent experimental studies have provided substantial insight into the toxicological effects of mycotoxins on aquatic organisms. These studies have particularly highlighted the importance of combined exposure scenarios, the molecular mechanisms of toxicity and mitigation strategies. The results of selected experimental studies on mycotoxin toxicity and mitigation strategies in fish are presented in Table 1.

Table 1. Selected experimental studies on mycotoxin toxicity and mitigation strategies in fish

Species / Model	Exposure type	Mycotoxins / Compounds	Key endpoints	Main findings	Study (Year)
Zebrafish (F0 and F1)	Co-exposure (<i>in vivo</i> + multi-omics)	AFB1 + epoxiconazole	Multi-organ toxicity, gene expression, transgenerational effects	Synergistic toxicity; activation of ferroptosis (liver) and apoptosis (ovary); intestinal damage; microbiota disruption; transgenerational effects in F1 embryos	Wang et al., 2025 [44]
<i>Lates calcarifer</i>	Dietary (5 weeks)	Multi-mycotoxins + histamine + rancid fats	Growth, blood biochemistry, histopathology	Reduced growth and survival; haemolysis; hyperkalemia; liver and kidney pathology; diet-specific toxicity patterns	Kwok et al., 2025 [45]
<i>Piaractus mesopotamicus</i>	Dietary + probiotic intervention	AFB1 (0-400 µg/kg)	Liver histology, antioxidant enzymes	AFB1 ≥25 µg/kg caused liver damage and enzyme suppression; probiotic additive mitigated toxicity and restored liver function	Godoy et al., 2024 [46]
Nile tilapia	Dietary (8 weeks)	AFB1 + Se nanoparticles	Oxidative stress, immunity, gene expression	AFB1 induced oxidative stress and immunosuppression; SeNPs restored antioxidant status and improved resistance to infection	Sherif and Zommara, 2024 [47]
Zebrafish embryos	Co-exposure	AFB1 + tebuconazole	LC ₅₀ , gene expression, enzyme activity	Synergistic toxicity; altered antioxidant and apoptotic pathways; AFB1 more toxic than pesticide	An et al., 2024 [48]

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Species / Model	Exposure type	Mycotoxins / Compounds	Key endpoints	Main findings	Study (Year)
Grass carp	Dietary	AFB1	Immune response, gene expression	Immunosuppression via NF-κB and TOR pathways; reduced antimicrobial peptides; threshold ~29.48 µg/kg diet	He et al., 2022 [49]
Zebrafish embryos	Co-exposure	AFB1 + FB1	Development, oxidative stress, apoptosis	Combined exposure more toxic than single toxins; endocrine disruption; increased oxidative and apoptotic markers	Di Paola et al., 2022 [50]
<i>Channa argus</i>	Dietary (8 weeks)	AFB1 (0-400 ppb)	ROS/ERS signalling, inflammation, apoptosis	Dose-dependent oxidative stress; ER stress activation; apoptosis and inflammation via ROS-ERS axis	Jiao et al., 2025 [51]

A growing body of evidence suggests that exposure to multiple environmental contaminants, including mycotoxins, can result in synergistic, multi-organ toxicity. For example, Wang et al. [44] showed that exposure to a combination of aflatoxin B1 (AFB1) and epoxiconazole in zebrafish caused severe intestinal damage and altered the gut microbiota. It also activated key molecular pathways, including ferroptosis in the liver and apoptosis in the ovaries. Notably, these effects were observed in the F1 generation too, confirming transgenerational toxicity and heritable molecular alterations. Similarly, An et al. [48] reported synergistic developmental toxicity in zebrafish embryos co-exposed to AFB1 and tebuconazole, with significant modulation of antioxidant and apoptotic pathways.

The importance of mixture toxicity is also supported by Di Paola et al. [50], who showed that exposure to a mixture of AFB1 and fumonisin B1 (FB1), even at concentrations that are individually non-toxic, resulted in increased mortality, oxidative stress, apoptosis and endocrine disruption. These findings emphasise that the co-occurrence of mycotoxins poses a significantly greater risk than exposure to a single compound, a scenario that is highly relevant to real-world aquaculture conditions.

In dietary exposure studies, the chronic intake of contaminated feed has consistently been associated with impaired growth, metabolic disturbances and organ pathology. Kwok et al. [45] found that juvenile Asian seabass fed a diet containing multiple mycotoxins (aflatoxin, fumonisin, deoxynivalenol and zearalenon) showed reduced growth, haemolysis, electrolyte imbalance and histopathological lesions in their livers and kidneys. This study also highlighted the interactive effects of mycotoxins, histamines, and lipid peroxidation products, reflecting the complexity of feed contamination.

Mechanistic studies have further elucidated the role of oxidative stress and related signalling pathways in mycotoxin-induced toxicity. Jiao et al. [51] demonstrated that exposure to AFB1 in *Channa argus* triggered a cascade involving the overproduction of reactive oxygen species (ROS), endoplasmic reticulum stress (ERS), inflammation, and apoptosis via the ROS–ERS signalling axis. Similarly, He et al. [49] showed that AFB1 impairs immune

function in grass carp by modulating the NF- κ B and TOR pathways, resulting in decreased antimicrobial defence and increased susceptibility to pathogens. Taken together, these findings indicate that oxidative stress and immune dysregulation are central mechanisms underlying mycotoxin toxicity in fish.

Several studies have also explored strategies to mitigate mycotoxin-induced damage. For example, Godoy et al. [46] demonstrated that probiotic-based additives significantly alleviated AFB1-induced hepatotoxicity in *Piaractus mesopotamicus* by restoring antioxidant enzyme activity and preventing histopathological lesions. Similarly, Sherif and Zommara [47] showed that selenium nanoparticles effectively counteracted oxidative stress and immunosuppression in Nile tilapia exposed to AFB1, enhancing resistance to *Streptococcus agalactiae* infection. These studies highlight the potential of nutritional and functional feed additives as practical tools for managing mycotoxin risk in aquaculture.

The available evidence suggests that mycotoxin toxicity in fish is a multifactorial process involving oxidative stress, apoptosis, immune dysregulation, and endocrine disruption. This process is often exacerbated by exposure to multiple contaminants. However, despite significant advances, there are still major knowledge gaps regarding long-term low-dose exposure, mixture toxicity and transgenerational effects. These areas should be prioritised in future research.

Zearalenone and other regulated mycotoxins. Zearalenone (ZEN) is a mycotoxin with known oestrogenic activity that has been studied less extensively in fish, but which is linked to reproductive disturbances. In zebrafish, for example, it has been shown to reduce spawning frequency and alter fecundity across generations [52], while higher exposure levels have been associated with developmental abnormalities such as cardiac defects and skeletal deformities [53]. These effects are consistent with ZEN's role as an endocrine disruptor, capable of interfering with hormonal signalling pathways in aquatic organisms.

Low concentrations of ZEN have been detected in the livers and ovaries of rainbow trout (*Oncorhynchus mykiss*) and common carp (*Cyprinus carpio*), although levels in muscle tissue were generally minimal [54–56]. ZEN is known for its oestrogenic activity, and fish appear to be particularly sensitive to endocrine-disrupting compounds due to their reproductive physiology and continuous exposure to the aquatic environment. Reproductive impairment, an altered gonadosomatic index, reduced fecundity, and endocrine-disrupting effects have been observed [54]. Additionally, exposure in the early life stages may lead to long-term effects on gonadal development and reproductive success, even at sublethal concentrations. Other regulated mycotoxins, such as deoxynivalenol (DON) and fumonisins, are primarily associated with reduced feed intake, impaired growth, intestinal damage, and immune modulation rather than significant tissue accumulation [57]. While evidence suggests that many trichothecenes exhibit relatively low bioaccumulation potential in muscle tissue, they can still induce chronic sublethal effects that compromise fish welfare and aquaculture productivity [10].

Recent studies have significantly advanced our understanding of the toxicological effects of major *Fusarium*-derived mycotoxins, particularly ZEN, DON and fumonisins, as well as emerging compounds, in aquatic organisms.

These investigations have revealed a variety of toxicity mechanisms, including neurotoxicity, oxidative stress, immune system disruption and impaired growth and regeneration, which are often exacerbated by exposure to multiple toxins (Table 2).

Table 2. Recent studies on zearalenone (ZEN), deoxynivalenol (DON), fumonisins and emerging mycotoxins in fish

Species / Model	Mycotoxin(s) / Exposure	Experimental design	Key endpoints	Main findings	Study (Year)
Zebrafish larvae	ZEN	Behavioural + <i>in silico</i> + docking	Neurotoxicity, motor function	Reduced locomotion; neuroinflammation via TP53, AKT1, CASP3, MAPK3, NF- κ B pathways	Xu et al., 2025 [58]
Zebrafish larvae	ZEN (0-1.5 μ M)	Developmental exposure	Regeneration, immunity, ROS	Impaired regeneration; neutrophil dysregulation; oxidative stress; apoptosis	Luo et al., 2025 [59]
Zebrafish embryos	ZEN + hydroxytyrosol	Detoxification study	Development, apoptosis, gene expression	Hydroxytyrosol mitigated ZEN toxicity; reduced apoptosis and cell cycle arrest (p53/FoxO)	Zhang et al., 2024 [60]
Zebrafish + marine fish embryos	ZEN	Metabolomics (HRMAS NMR)	Energy metabolism, oxidative stress	Mitochondrial dysfunction; altered metabolites; oxidative stress biomarkers identified	Annunziato et al., 2023 [61]
Zebrafish embryos	ZEN + trifloxystrobin	Co-exposure	Enzymes, gene expression	Synergistic toxicity; oxidative, immune and endocrine disruption	Cang et al., 2023 [62]
Zebrafish fibroblasts	DON	<i>In vitro</i> mechanistic study	Cell cycle, proliferation	Ribosome binding; cell cycle arrest; impaired DNA replication; mitophagy activation	Duan et al., 2025 [63]
Zebrafish embryos and adults	DON + bacteria	Toxicity + bioremediation	Survival, ROS, histology	DON reduced survival and induced oxidative stress; bacteria showed detoxification potential	Yao et al., 2024 [64]
Rainbow trout	DON + <i>Fusarium</i> toxins	Dietary (8 weeks)	Growth, histology	Additive (not synergistic) toxicity; reduced growth and energy retention	Koletsis et al., 2023 [65]
Gilthead seabream	ENNБ + FB1	Dietary (28 days)	Metabolism, neurotoxicity	ENNБ more toxic; lipid/protein degradation; AChE inhibition (mixture)	Mello et al., 2025 [66]
Grass carp	FB1 (0-8.05 mg/kg)	Dietary (30 days)	Growth, intestine, oxidative stress	Impaired growth; intestinal damage; apoptosis; oxidative stress (Keap1/Nrf2 disruption)	Chen et al., 2025 [67]
Zebrafish embryos	FB1 derivatives	<i>In vivo</i> + binding study	Toxicity ranking	Palmitoyl-FB1 derivatives more toxic than FB1; higher protein binding affinity	Csenki et al., 2023 [68]

ZEN is increasingly recognised as a multisystem toxicant that affects endocrine, neural, and immune pathways. Xu et al. [58] demonstrated that ZEN

exposure significantly reduced the locomotor activity of zebrafish larvae, indicating neurotoxicity. Mechanistically, this effect was associated with the activation of neuroinflammatory pathways involving key molecular targets such as TP53, AKT1, CASP3, MAPK3, and NF- κ B. Complementary molecular docking results confirmed strong binding affinities between ZEN and these proteins, supporting the idea of direct molecular interactions underlying neurotoxicity.

Concurrently, Luo et al. [59] revealed that ZEN disrupts tissue regeneration by altering neutrophil dynamics and inducing oxidative stress. Increased ROS production, apoptosis, and impaired antioxidant defence were found to be associated with delayed fin regeneration and developmental abnormalities. Taken together, these findings suggest that immune dysregulation and redox imbalance are central to ZEN-induced developmental toxicity.

Metabolomics-based evidence further supports these mechanisms. Annunziato et al. [61] identified conserved metabolic alterations in both freshwater and marine fish embryos, including mitochondrial dysfunction, membrane disruption, and impaired energy metabolism. These metabolic signatures could be used as early biomarkers of ZEN exposure in aquaculture and environmental monitoring.

Importantly, mitigation strategies are being explored. Zhang et al. [60] demonstrated that hydroxytyrosol effectively counteracts ZEN-induced developmental toxicity by modulating apoptosis and cell cycle pathways (the p53/FoxO axis), thereby restoring normal growth and behaviour. This highlights the potential of natural antioxidants as protective agents against mycotoxin-induced damage.

Studies on co-exposure confirm that exposure to a combination of mycotoxins and other contaminants can result in enhanced or synergistic toxic effects. For example, Cang et al. [62] reported that the combination of ZEN and the fungicide trifloxystrobin significantly altered the expression of antioxidant enzymes, endocrine markers, and genes related to the immune system in zebrafish embryos. Gene expression changes were notably more pronounced under co-exposure conditions than with individual compounds, emphasising the importance of mixture-based risk assessment approaches.

DON toxicity is primarily associated with the inhibition of protein synthesis and cell proliferation. Duan et al. [63] demonstrated that DON binds directly to ribosomes in zebrafish fibroblasts, leading to impaired DNA replication, cell cycle arrest, and disrupted mitosis. Furthermore, the activation of autophagy and mitophagy pathways indicates a cellular stress response to ribotoxic damage. Rainbow trout are highly sensitive to DON, exhibiting reduced growth, feed intake, and nutrient utilisation even at low concentrations [69], whereas channel catfish demonstrate greater tolerance, with minimal effects observed even at higher dietary levels [70]. DON exposure is often linked to reduced growth performance and feed efficiency [69, 71], though some studies report no significant impact on weight gain [72, 73]. This suggests variability in species responses and experimental conditions.

Further *in vivo* studies confirm its detrimental effects. Yao et al. [64] reported reduced survival rates, intestinal damage, and increased oxidative stress in zebrafish exposed to DON. However, the application of *Achromobacter*

sp. P-9 showed promising biodegradation potential, suggesting that bioremediation strategies could mitigate DON toxicity in aquaculture systems. Koletsi et al. [65] demonstrated that DON and other *Fusarium* toxins have an additive negative effect on the growth performance and feed efficiency of rainbow trout, with no clear evidence of synergistic interactions. These findings suggest that chronic exposure to multiple mycotoxins may cumulatively impair productivity, even in the absence of strong synergism.

Fumonisin B1 (FB1) primarily affects intestinal integrity and sphingolipid metabolism. Chen et al. [67] demonstrated that dietary FB1 impairs growth, disrupts intestinal barrier function, induces oxidative stress, and activates apoptosis in grass carp. These effects were associated with the inhibition of the Keap1/Nrf2 antioxidant pathway and alterations in tight junction proteins, indicating compromised intestinal health and reduced nutrient absorption.

Fumonisin B1 has been shown to disrupt sphingolipid metabolism and act as a cancer promoter in rainbow trout, while also impairing growth and haematological parameters in species such as Nile tilapia and common carp. Similarly, T-2 toxin and MON negatively affect growth, haematological indices and organ integrity in various fish species [74–76]. Many *Fusarium* toxins mechanistically interfere with protein synthesis, lipid metabolism, and intestinal integrity, leading to reduced nutrient absorption and systemic physiological stress.

Emerging evidence also highlights the importance of less-studied mycotoxins. For example, Mello et al. [66] demonstrated that enniatin B (ENNB) had a stronger toxic effect than FB1 on gilthead seabream, causing metabolic disruption and neurotoxicity (acetylcholinesterase inhibition). This finding emphasises the importance of including emerging mycotoxins in regulatory frameworks and risk assessments. Furthermore, Csenki et al. [68] demonstrated that modified fumonisin derivatives (e.g. palmitoyl-FB1) are significantly more toxic than the parent compound, likely due to increased protein binding affinity. This emphasises the importance of considering modified ('masked') mycotoxins in exposure and toxicity evaluations.

These studies collectively demonstrate that mycotoxin toxicity in fish is a multidimensional process involving oxidative stress, inflammation, apoptosis, metabolic disruption, and impaired physiological functions, which is strongly modulated by co-exposure and environmental context. Emerging evidence also emphasises the importance of modified mycotoxins, mixture toxicity and mitigation strategies, all of which are underrepresented in current regulatory frameworks and require further investigation.

Mixtures, emerging toxins and knowledge gaps. Most experimental designs have employed single-toxin fortification under controlled laboratory conditions. However, naturally contaminated feeds typically contain complex mixtures of mycotoxins. This discrepancy limits the ecological and practical relevance of many toxicokinetic and toxicodynamic findings. The potential for additive or synergistic interactions among co-occurring toxins in freshwater species are not well characterised. Furthermore, data on emerging, modified ('masked') or conjugated mycotoxins in freshwater fish tissues are scarce, limiting the ability to conduct a comprehensive risk assessment for consumers [9, 2]. In particular, the role of intestinal microbiota in the

biotransformation of masked mycotoxins is an emerging factor that still needs to be understood better in terms of its influence on internal exposure.

Occurrence in marine fish and products. Compared with freshwater species, fewer studies have addressed the occurrence of mycotoxins in marine aquaculture. However, concerns have been raised about long-term exposure and potential tissue residues due to the rapid growth of marine fish farming and the increasing use of plant-based ingredients in marine aquafeeds. As in freshwater systems, dietary intake remains the primary exposure route. While this shift towards plant-based feed formulations in marine aquaculture reflects global sustainability trends, it simultaneously increases the risk of introducing mycotoxins into production systems that were previously less exposed.

Regulated mycotoxins. Concentrations of AFB1 of up to 4.25 µg/kg were detected in the muscle of European sea bass (*Dicentrarchus labrax*) following experimental dietary exposure [21]. In Atlantic salmon (*Salmo salar*), deoxynivalenol (DON) was distributed across tissues (with concentrations of up to 28.6 µg/kg in the liver), while ochratoxin A (OTA) mainly accumulated in the liver and kidneys [77]. These findings confirm that the liver remains the primary target organ for bioaccumulation and biotransformation, whereas muscle residues are generally lower, yet still of concern to consumers from a toxicological perspective. Species-specific differences in lipid content and metabolic capacity may further influence residue distribution patterns in marine fish.

Market surveillance studies generally report low detection frequencies in commercially available marine fish. However, some investigations have identified OTA levels exceeding 1 µg/kg. Although maximum residue limits (MRLs) for mycotoxins in fish muscle are not harmonised at the EU level, the presence of detectable residues highlights the need for ongoing monitoring, particularly in intensively farmed species. The lack of harmonised regulatory thresholds for fish products complicates risk assessment and may lead to inconsistencies in the evaluation of food safety across different regions.

Chronic dietary exposure to mycotoxins in marine fish has been associated with reduced growth performance, oxidative stress, hepatocellular alterations, and modulation of immune responses, even in the absence of high tissue accumulation [2,9]. While such subclinical effects may not be immediately apparent, they can significantly impact production efficiency and disease resistance in aquaculture systems.

Emerging mycotoxins. Enniatins (ENNA, ENNA1, ENNB and ENNB1) and beauvericin (BEA) have been found in European sea bass, gilthead sea bream (*Sparus aurata*) and Atlantic salmon. ENNB often exhibited the highest prevalence, with tissue concentrations reaching 119 µg/kg in certain cases [78, 79]. These cyclic hexadepsipeptide compounds are mainly produced by *Fusarium* species and are characterised by ionophoric activity, which can disrupt cellular ion homeostasis. Their lipophilic nature may explain their deposition in fatty tissues, such as those found in salmon. Preliminary toxicological evidence suggests potential cytotoxic, immunomodulatory, and pro-oxidative effects; however, data on these effects in marine fish are limited [9,2]. Furthermore, their frequent co-occurrence with regulated mycotoxins raises concerns about combined toxicity and interactive effects that are not yet fully understood.

Similarly, data on *Alternaria* toxins, such as alternariol (AOH) and alternariol monomethyl ether (AME), in marine fish are extremely limited. Given the increasing presence of *Alternaria*-derived metabolites in plant-based feeds, further research is needed to assess their potential for bioaccumulation and metabolic transformation, as well as the implications for the safety of marine aquaculture products [80,81]. Future studies should also address the transfer of these emerging toxins along the marine food chain and their potential implications for human dietary exposure.

Toxicological profile of emerging mycotoxins. Although there is little in vivo data on fish, extensive in vitro studies provide valuable insight into the toxicity mechanisms of emerging mycotoxins. Most of the available evidence originates from mammalian cell culture models. While these models are not fully representative of aquatic species, they enable the identification of conserved cellular targets, such as mitochondria, oxidative stress pathways, and DNA integrity mechanisms. However, it is important to note that interspecies differences in xenobiotic metabolism, particularly in liver enzyme systems, may significantly influence the extrapolation of these findings to fish. These compounds are often found together in feed, raising concerns about their combined toxicity. This co-occurrence reflects real exposure scenarios in aquaculture and highlights the need for toxicological assessments of mixtures rather than single compounds.

Beauvericin (BEA). Beauvericin is a cyclic hexadepsipeptide that is primarily produced by *Fusarium* species. It exhibits strong cytotoxicity in multiple cell lines, including Caco-2, HepG2, Jurkat T-cells, dendritic cells, and ovarian cells [82-84]. IC₅₀ values generally fall within the low micromolar range (1–12 µM), indicating considerable biological potency. The mechanisms of action include the generation of reactive oxygen species (ROS), lipid peroxidation, apoptosis, cell cycle arrest and mitochondrial dysfunction [85–87]. BEA acts as an ionophore, forming complexes with cations (e.g. Ca²⁺, Na⁺ and K⁺), thereby disrupting cellular ion homeostasis and mitochondrial membrane potential. The EFSA concluded that BEA is not directly genotoxic; the observed DNA damage appears to be a consequence of oxidative stress and indirect mechanisms [88]. Nevertheless, its high lipophilicity may favour bioaccumulation in lipid-rich tissues, which is particularly relevant for certain fish species. However, uncertainties remain regarding chronic low-dose exposure and potential immunomodulatory effects, particularly in scenarios involving co-exposure to other *Fusarium* toxins.

Enniatins (ENNs). Structurally related to BEA, the enniatin family (ENNA, ENNA1, ENNB and ENNB1) exhibits dose- and time-dependent cytotoxicity in intestinal, hepatic, immune and neural cell models [89–91]. ENNA1 generally exhibits the greatest potency. Reported IC₅₀ values range from 2 to 12 µM depending on the cell type and exposure duration. Mechanisms include ROS production, apoptosis induction and disruption of ion homeostasis [92,93]. Like BEA, enniatins exhibit ionophore properties, facilitating transmembrane cation transport and leading to mitochondrial depolarisation, ATP depletion, and activation of the intrinsic apoptotic pathway [94]. Furthermore, evidence suggests that they may modulate inflammatory signalling pathways and impair epithelial barrier integrity in intestinal models [95]. Their ability to cross biological membranes efficiently may enhance their systemic distribution

following dietary exposure. Despite increasing detection in food and feed, maximum levels have not yet been specified in EU legislation due to limited *in vivo* toxicity data and insufficient exposure assessment.

***Alternaria* toxins (AOH and AME).** These toxins, produced by *Alternaria* species, exert cytotoxic effects in both tumour and normal cell lines, including HepG2, Caco-2, GES-1 and PC-3 cells [96–98]. AME is often more potent than AOH. Mechanisms involved include DNA strand breaks, topoisomerase inhibition, activation of the p53-mediated DNA damage response, and oxidative stress [98, 99]. Both AOH and AME have demonstrated genotoxic potential *in vitro*, including clastogenic effects and interference with DNA repair processes. This raises concerns regarding long-term carcinogenic risk [97–99]. Furthermore, there is some suggestion of their potential interaction with endocrine signalling pathways, but this remains insufficiently characterised. However, human bioaccumulation data for *Alternaria* toxins are currently unavailable. Similarly, *in vivo* data in fish are extremely limited, preventing robust characterisation of the risk to aquatic species and consumers. Further toxicokinetic, bioaccumulation and mixture toxicity studies are needed to clarify their safety profile in aquaculture contexts. In particular, integrating omics-based approaches (e.g. transcriptomics and metabolomics) into studies could provide deeper insight into their mechanisms of action.

The toxicological relevance and regulatory oversight of mycotoxins detected in aquaculture can differ substantially depending on whether they are classified as regulated, emerging, or *Alternaria*-derived compounds. The following comparative overview summarises current knowledge gaps, the regulatory status, and evidence of carry-over into fish tissues within the context of the European Union (Table 3).

Table 3. Comparative overview of regulatory status, occurrence, carry-over, and toxicological evidence of regulated and emerging mycotoxins in aquaculture

Aspects	Regulated mycotoxins (AFB1, OTA, DON, ZEN)	Emerging mycotoxins (BEA, ENNs)	<i>Alternaria</i> toxins (AOH, AME)
Regulatory status (EU)	Partially regulated	Not regulated	Not regulated
Occurrence in aquafeed	Frequent	Increasingly reported	Limited data
Carry-over to fish	Documented	Species-dependent	Insufficient data
Toxicity evidence	<i>In vivo</i> and <i>in vitro</i>	Mostly <i>in vitro</i>	Mostly <i>in vitro</i>
Genotoxicity	AFB1: carcinogenic	Limited evidence	DNA damage mechanisms reported
Risk assessment	Partial	Insufficient data	Insufficient data

It highlights the imbalance between the increasing detection of emerging mycotoxins and the limited availability of toxicological and regulatory frameworks that address them.

Mycotoxins such as aflatoxin B1, ochratoxin A, deoxynivalenol and zearalenone are regulated within the European Union, where maximum or guidance levels have been set for them in food and feedstuffs. AFB1 is strictly regulated due to its well-established carcinogenicity, while guidance values exist for DON, ZEN and fumonisins in feedstuffs, including fish feed. In contrast, emerging mycotoxins such as beauvericin and enniatins, as well as *Alternaria* toxins such as AOH and AME, are not currently regulated at the EU level due to

a lack of toxicological and exposure data [2, 9]. This regulatory gap highlights the urgent need for risk assessment frameworks that are harmonised and incorporate emerging contaminants and their combined effects in aquaculture systems.

Conclusions. Contamination of aquaculture feed and fish products by mycotoxins represents an evolving and multifaceted food safety challenge. The growing global dependence on plant-based feed ingredients has exacerbated the risk of exposure to multiple mycotoxins in both freshwater and marine aquaculture systems. Feed remains the main source of contamination, especially given the progressive replacement of fishmeal and fish oil with cereal- and oilseed-derived components.

The carry-over of regulated mycotoxins, especially aflatoxin B₁, ochratoxin A, deoxynivalenol and zearalenone, into fish tissues has been documented. However, residue levels are highly variable and are influenced by species, exposure duration, feed composition and metabolic capacity. The liver is consistently identified as the primary target organ, whereas muscle residues are generally lower, yet still relevant for assessing consumer exposure.

Emerging mycotoxins, including beauvericin and enniatins, are prevalent in aquafeeds and are occasionally detected in fish tissues. However, they remain unregulated at EU level. Their lipophilic and ionophoric properties, combined with evidence of cytotoxicity, oxidative stress induction and mitochondrial dysfunction, raise concerns regarding chronic low-dose exposure and potential mixture effects.

Currently, toxicological evidence is dominated by *in vitro* studies with limited *in vivo* validation in fish models. Long-term feeding trials, toxicokinetic investigations (absorption, distribution, metabolism and excretion) and mixture toxicity assessments are urgently needed to provide robust data for risk characterisation. The occurrence of masked or modified mycotoxins in fish tissues and their potential reconversion to parent toxins during digestion remain largely unexplored, constituting a major research gap.

The absence of harmonised maximum residue limits for mycotoxins in fish and fish products makes regulatory oversight more difficult. Although guidance values exist for certain mycotoxins in feed, the absence of specific limits for edible fish tissues creates a regulatory gap within the farm-to-fork continuum. To support future risk assessments and regulatory decision-making, harmonised monitoring strategies, validated multi-mycotoxin analytical methods (e.g. LC-MS/MS and HRMS) and integrated surveillance programmes are urgently required.

In light of the projected growth in global aquaculture, it is crucial to systematically investigate the occurrence of mycotoxins, their toxicokinetics, their potential for bioaccumulation, and their cumulative dietary exposure, in order to safeguard fish health, ensure the sustainability of the industry, and protect consumers. A coordinated approach that integrates feed control, environmental monitoring, toxicological research and regulatory harmonisation will be critical for the effective, long-term management of risks in the aquaculture sector.

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ЗАБРУДНЕННЯ МІКОТОКСИНАМИ В СИСТЕМАХ АКВАКУЛЬТУРИ: ДЖЕРЕЛА, ТОКСИКОЛОГІЧНІ ЕФЕКТИ ТА НАСЛІДКИ ДЛЯ БЕЗПЕКИ ХАРЧОВИХ ПРОДУКТІВ

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Анотація. Мікотоксини – це токсичні вторинні метаболіти, що продукуються мікроскопічними (пліснявими) грибами та часто контамінують рослинні компоненти кормів, які використовуються в аквакультурі. Постійна заміна рибного борошна та риб'ячого жиру зерновими й олійними культурами підвищує ризик хронічного впливу на рибу множинних мікотоксинів. Цей огляд має на меті надати стислий аналіз проблеми контамінації

мікотоксинами в системах аквакультури. У роботі узагальнено дані щодо поширення, шляхів надходження, токсикокінетики та біологічних ефектів основних регульованих мікотоксинів, таких як афлатоксин В1, дезоксиніваленол, зеараленон, фумонізиди та охратоксин А, а також нових сполук, зокрема боверицину, енніатинів і токсинів роду *Alternaria*. Хоча фактори довкілля та технологічні процеси також можуть сприяти контамінації, корм визначено як основне джерело забруднення. Мікотоксини характеризуються обмеженим транспортуванням у тканини риб, при цьому печінка є основним місцем їх накопичення та біотрансформації; водночас рівень залишків у м'язах залишається відносно низьким, але все ще важливим з точки зору безпеки харчових продуктів. Токсичні ефекти мають багатофакторний характер і включають оксидативний стрес, апоптоз, порушення імунної регуляції, ендокринні порушення та метаболічні розлади. Зростає кількість доказів, що підкреслюють важливість комбінованого впливу, за якого адитивні або синергічні взаємодії можуть посилювати токсичність. Дослідження нових і модифікованих («маскованих») мікотоксинів залишається недостатнім, особливо в умовах *in vivo*, що ускладнює оцінку ризику. **Висновки:** Покращення моніторингу, глибше розуміння комбінованої токсичності та гармонізація нормативних обмежень є необхідними. Розробка ефективних стратегій мінімізації ризиків є ключовою для забезпечення сталого розвитку аквакультури та безпеки харчових продуктів, особливо в умовах зростаючої залежності від рослинних кормів.

Ключові слова: мікотоксини; аквакультура; здоров'я риб; оксидативний стрес; кумуляція в тканинах; харчова безпека; токсичність сумішей; оцінка ризику

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