The consequences of mycotoxins in aquaculture

Most of the mycotoxins that have the potential to reduce growth and health status of fish and other farmed animals consuming contaminated feed are produced by *Aspergillus*, *Penicillium* and *Fusarium* sp. The major classes of mycotoxins include the aflatoxins, trichothecenes, fumonisins, zearalenone and ochratoxins.

**AFLATOXINS IN FISH**

Aflatoxins are produced by *Aspergillus* fungi, which can infect many potential feedstuffs as corn, peanuts, rice, fish meal, shrimp and meat meals. Aflatoxin B₁ (AFB₁) is one of the most potent, naturally occurring, cancer-causing agents in animals. Initial findings associated with aflatoxicosis in fish include pale gills, impaired blood clotting, anemia, poor growth rates or lack of weight gain. The extent of disease, caused by consumption of aflatoxins, depends upon the age and species of the fish. Fry are more susceptible to aflatoxicosis than adults and some species of fish are more sensitive to aflatoxins than others. Rainbow trout is reported to be one of the most sensitive animals to aflatoxin poisoning; the LD₅₀ (dose causing death in 50% of the subjects) for AFB₁ in a 50g trout being 500–1000 ppb (0.5–1.0 mg/kg). The carcinogenic or toxic effects of aflatoxins in fish seem to be species specific. While Rainbow trout are extremely sensitive to AFB₁, warm water fish such as channel catfish (*Ictalurus punctatus*) are reported to be less sensitive to aflatoxins.

Although less sensitive, warm water species are still affected by aflatoxin contamination. Feeding a diet containing 10 ppm AFB₁/kg diet to channel catfish caused reduced growth rate and moderate internal lesions over a 10-week trial period. In carp, it was reported that aflatoxins are potential immuno-suppressors. A recent study indicated that feeding diets containing aflatoxins from moldy corn does not seem to affect channel catfish weight gain, feed consumption, feed efficiency, and survival. Studies on the Nile tilapia (*Oreochromis niloticus*) showed reduced growth rates when tilapias were fed diets containing 1880 ppb AFB₁. In addition, tissue abnormality or lesions in the livers of these tilapias showed the beginnings of cancer development. In another study, Nile tilapia fed diets with 0.1 ppm AFB₁ for 10 weeks had reduced growth, and fish fed diet with 0.2 ppm AFB₁ had 17% mortality. A more recent study showed that acute and sub-chronic effects of AFB₁ to Nile tilapia are unlikely if dietary concentrations are 0.25 ppm or less.
However, diets containing levels of AFB₁ higher than 0.25 ppm had lower weight gain and haematocrit count compared to a control diet. Diets containing 100 ppm AFB₁ caused weight loss and severe hepatic necrosis in Nile tilapia. Other studies have shown that tolerance levels for tilapia can vary with the production system. In green water and flow-through systems, the presence of aflatoxins at 25 to 30 ppb in the water decreased growth without any noticeable signs of mortality. However, in cage culture, concentrations of aflatoxins above 5 ppb caused an increase in mortality rates³.

**AFLATOXINS IN SHRIMP**

In marine shrimp, several studies showed that AFB₁ can cause abnormalities such as poor growth, low apparent digestibility, physiological disorders and histological changes, principally in the hepatopancreatic tissue. Nevertheless, reports on the effect of AFB₁ on shrimp are inconsistent. A study in 2003³⁴ reported that after just 7 or 10 days of consumption of diets with AFB₁ levels below 20 ppb, mortality rate was slightly higher in AFB₁-treated groups than in the control group. Histopathological findings indicated hepatothropic damage by AFB₁ with biochemical changes of the haemolymph. In another study, AFB₁ at 50–100 ppb showed no effect on growth in juvenile shrimps³⁵. However, growth was reduced when AFB₁ concentrations were elevated to 500–2500 ppb. Survival dropped to 26.32% when 2500 ppb AFB₁ was fed, whereas concentrations of 50–1000 ppb had no effect on survival³⁶. There were marked histological changes in the hepatopancreas of shrimp fed diet containing AFB₁ at a concentration of 100–2500 ppb for 8 weeks, as noted by atrophic changes, followed by necrosis of the tubular epithelial cells. Severe degeneration of hepatopancreatic tubules was common in shrimp fed high concentrations of AFB₁³⁷. According to a study in 2005³⁸, the effect of AFB₁ toxicity to shrimp results in the modification of digestive processes and abnormal development of the hepatopancreas due to exposure to mycotoxins.

**OCHRATOXINS**

Ochratoxins are a group of secondary metabolites produced by fungal organisms belonging to *Aspergillus* and *Penicillium* genera. Ochratoxin A (OA) is the most abundant of this group and is more toxic than other ochratoxins. Very few studies have been conducted to determine the effect of ochratoxins in fish species. In juvenile channel catfish, diets containing levels of 1 to 8 ppm of OA resulted in the development of toxic responses. Significant reduction in body weight gain was observed after only 2 weeks in fish fed diets containing 2 ppm of ochratoxin A or above³⁹. After 8 weeks body weight gain was significantly reduced for fish fed diets containing 1 ppm OA or above. Additional toxic responses included poorer FCR for fish fed diets with 4 or 8 ppm OA, and lower survival and hematocrit count for fish fed the 8 ppm OA diet. Severe histopathological lesions of liver and posterior kidney were observed after 8 weeks for catfish fed diets containing levels of OA of 4 and 8 ppm⁴⁰. In growing rainbow trout the oral LD₅₀ of ochratoxin A has been determined to be 4.67 ppm.

**CYCLOPIAZONIC ACID (CPA)**

Cyclopiazonic acid (CPA) is a mould toxin produced by several species of *Aspergillus* and *Penicillium* fungi. CPA, a neurotoxin frequently found in association with aflatoxins, has been found to be more toxic to channel catfish than aflatoxins and is more frequently found than aflatoxins in feedstuffs in the southern United States. A dietary level of 100 ppb CPA significantly reduced growth, and 10,000 ppb caused necrosis of gastric glands. The minimum dietary concentration that caused a reduction in growth rate was 100 ppb for CPA as compared with 10,000 ppb for AFB₁⁴¹.

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pressing growth and/or causing histopathological lesions, this fish survived mycotoxins levels up to 150 ppm. Reduction on the percentage of survival of channel catfish was observed for diets containing 240 ppm FB1.

Studies on the effect of FB1 in carp indicated that long-term exposure to 0.5 and 5.0 mg per kg body weight is not lethal to young carp, but can produce adverse physiological effects. The primary target organs of FB1 in the carp are kidney and liver. Other changes subsequent to fumonisin exposure that have been reported for carp include scattered lesions in the exocrine and endocrine pancreas, and inter-renal tissue, probably due to ischemia and/or increased endothelial permeability.

**TRICHOTHECENES**

Trichothecenes are a group of mycotoxins produced by certain fungi of the genus *Fusarium* that infect the grains, wheat by-products and oilseed meals used in the production of animal feeds. The type A-trichothecene T2-toxin produced by the fungus *Fusarium tricinctum* proved lethal to rainbow trout at a dietary concentration near 6 mg/kg body weight, however, fed rainbow trout T2-toxin at 15 ppm of diet and found that the main effects were reduced feed consumption, reduced growth, lower hematocrit, and lower blood hemoglobin.

Results from 2003 demonstrated that T2-toxin is toxic to juvenile channel catfish. Reductions in growth rate were observed after 8 weeks for fish fed diets containing levels of T2-toxin ranging from 0.625-5.0 ppm, compared to a control diet. Significantly poorer feed conversion ratio was found only for the highest level of T2-toxin (5 ppm). The survival of fish fed T2-toxin at 2.5 and 5 ppm was significantly lower than that of the control fish.

A recent study with channel catfish indicate that disease resistance of juvenile channel catfish was reduced when fed feedborne T-2 toxin, resulting in significantly greater mortality when challenged with *Edwardsiella ictaluri* compared to a control group.

In carp, the injection of T-2 toxin did not significantly change the activity of enzymes in carp liver, although a tendency for reduction was noted.

Deoxynivalenol (DON), also known as vomitoxin, and other type B trichothecenes are produced by *Fusarium sp.* and can be an important contaminant of wheat. Deoxynivalenol levels of 0.2, 0.5, and 1.0 ppm in the diet significantly reduced body weight and growth rate in white shrimp *Litopenaeus vannamei*. However, the effects of 0.2 and 0.5 ppm DON were manifested at later stages of growth, and 0.2 ppm DON affected only growth rate and not body weight. Feed conversion ratio and survival of shrimp fed diets containing 0.2, 0.5, and 1.0 ppm DON were not significantly different from those of shrimp fed the control diet (0.0 ppm DON).

Reduced weight gain has also been noted in rainbow trout fed DON-contaminated feeds and feed refusal has been found to occur in fish fed with diets containing more than 20 ppm DON. For rainbow trout, a dietary level of 1–12.9 ppm resulted in reduced growth and feed efficiency showed that rainbow trout had sensitive taste acuity for DON and reduced their feed intake as the concentration of DON increased from 1 to 13 ppm of diet; the fish refused to consume the diet with a DON concentration of 20 ppm.

References 1–45 are available on request.