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Health & Welfare

Nitrite toxicity affected by species susceptibility, environmental conditions

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Nitrite toxicity, relatively common in freshwater aquaculture, can lead to brown-blood disease

Although relatively common in freshwater systems, nitrite toxicity is a lesser problem in brackish and seawater culture systems.

Nitrite is an intermediate compound in the oxidation of ammonia nitrogen to nitrate by nitrifying bacteria in soil and water. It also can be a product of denitrifying bacteria in anaerobic sediment or water. Nitrite is ultimately oxidized to nitrate in the presence of dissolved oxygen. Nevertheless, aquaculture systems usually contain small nitrite concentrations below 0.1 mg/L and under certain conditions, much greater amounts.

Nitrate – the most oxidized form of inorganic nitrogen in water – is not highly toxic to aquatic animals. The concentration of nitrate-nitrogen lethal to 50 percent of test organisms in 96 hours (96-hour LC50) typically is over 100 mg/L for freshwater organisms and more than 500 mg/L for marine species. On the other hand, nitrite is considerably more toxic.

Exposure to nitrite causes gill lesions and edema in the skeletal muscles of fish, but its main effect is on respiration. When absorbed into the bloodstream, nitrite combines with hemoglobin – or hemocyanin in invertebrates – to form methemoglobin or met-hemocyanin that does not combine with oxygen.

The percentage of methmoglobin or met-hemocyanin in the blood of aquatic animals increases as the nitrite concentration in the blood increases, lessening the ability of the blood to transport oxygen to the tissues. The effect of nitrite on respiration is particularly pronounced when the dissolved-oxygen concentration is low in the culture water.

Chloride effects

The chloride concentration in water also greatly affects the uptake of environmental nitrite across the gills and into the bloodstream of aquatic animals – especially freshwater animals.

Chloride ions have the same charge and are similar in size to nitrite ions. Because of this similarity, chloride competes with nitrite for adsorption sites on the active carrier mechanism responsible for transporting environmental nitrite across the gill lamellae to the bloodstream. By blocking the uptake of

nitrite by the carrier, chloride lessens the amount of nitrite that would pass from water to the bloodstream of aquatic animals at a particular nitrite-nitrogen concentration, thereby lessening the risk of nitrite toxicity.

Nitrite toxicity

There is a large body of information on nitrite's toxicity to fish, shrimp and other aquatic organisms. The 96-hour LC50s for nitrite-nitrogen typically range 10.00-30.00 mg/L for freshwater invertebrates and 0.25-100.00 mg/L for fish. The respective ranges for marine organisms are typically 10-300 mg/L and 100-1,000 mg/L. The 96-hour LC50s for several species of aquatic animals are presented in Table 1.

Boyd, Published 96-hour LC50s, Table 1

Common Name	96-hour LC50
Freshwater	
Common carp	88.0 mg/L
Catla	117.0 mg/L
Mitten crab	25.9 mg/L
Narrow-clawed crab	29.4 mg/L
Freshwater prawn	8.6 mg/L
Cutthroat trout	0.5-0.6 mg/L
Channel catfish	7.1-44.0 mg/L
Fathead minnow	45.0-70.0 mg/L
Blue tilapia	16.0 mg/L
Largemouth bass	140.0 mg/L
Rainbow trout	0.24-11.0 mg/L
Marine	
Mud crab	41.6-69.9 mg/L
Sea bass	154.0-274.0 mg/L
European eel	84.0-974.0 mg/L
Pacific white shrimp	9.0-322.0 mg/L
Black tiger prawn	13.6 mg/L
Sea trout	980.0 mg/L

Table 1. Published 96-hour LC50s for nitrite-nitrogen in several species of aquatic animals.

Some of the variation in toxicity relates to the species' differences in nitrite susceptibility. For example, the 96-hour LC50 in Pacific white shrimp at 35-ppt salinity is 322.0 mg/L, while at the same salinity, the value for black tiger prawn is only 14.0 mg/L. At a chloride concentration of 22.0 mg/L, the 96-hour LC50 for channel catfish is 7.5 mg/L, while for fathead minnows, it is 70.0 mg/L.

Water quality effects

Much of the variation in data from toxicity tests results from differences in the water quality conditions under which animals are exposed to nitrite. The LC50 tends to decrease with increasing temperature. For example, in a study with seabass, the LC50 declined from 274 mg/L at 17 degrees C to 154 mg/L at 27 degrees C. This relationship is not surprising, because at a higher temperature, organisms need more oxygen, and nitrite interferes with oxygen transport in the bloodstream.

Salinity also influences nitrite toxicity. The 96-hour LC50 of nitrite-nitrogen for European eels increased from 84 mg/L in freshwater to 974 mg/L in water with 36-ppt salinity. In Pacific white shrimp, LC50s rose from 61 mg/L at 15-ppt salinity to 322 mg/L at 35 ppt. This effect no doubt resulted from an increase in chloride concentration in response to greater salinity and provided protection against nitrite toxicity.

Coldwater species are much more sensitive to nitrite than are warmwater species. For example, LC50s for rainbow trout are four or more times lower than those for channel catfish.

The LC50 values from nitrite toxicity tests for aquaculture species are difficult to interpret because of the various factors that affect them. Moreover, aquaculturists want to avoid negative effects of nitrite on growth and increased susceptibility to diseases that occur at much lower concentrations than the LC50.

Safe concentrations for continuous exposure of aquatic animals to nitrite and other common toxins often are estimated as 0.05 or 0.10 of the 96-hour LC50. Based on reported LC50s, safe concentrations of nitrite-nitrogen in freshwater range 0.0125-0.5000 mg/L for coldwater fish and 0.5000-2.5000 mg/L for invertebrates and warmwater fish. For marine organisms, the ranges in safe concentrations are higher – 0.5 to 15.0 mg/L for invertebrates and 5.0 to 50.0 mg/L for fish.

Brown-blood disease

Nitrite toxicity is not a common problem in brackishwater and seawater systems, but it is relatively common in freshwater. One symptom of nitrite toxicity is easily recognized: The blood of fish or shrimp will be brown in color as a result of the elevated bloodstream concentrations of methemoglobin or met-hemocyanin.

As a result, nitrite toxicity commonly is referred to as brown-blood disease. The severity of brown-blood disease varies with nitrite concentration, dissolved-oxygen concentration and other factors. It is interesting to note that fish suffering brown-blood disease quickly recover when transferred to water with a low nitrite concentration.

Controls

Dissolved-oxygen concentrations below 3 mg/L for warmwater species and below 5 mg/L for coldwater species encourage oxidation of nitrite to nitrate in the water and at the sediment-water interface. They also provide a margin of safety for nitrite-stressed animals that are more susceptible to low dissolved-oxygen levels.

In channel catfish culture in the United States, farmers typically apply sodium chloride to ponds each year to maintain chloride concentrations of 50-100 mg/L that avoid brown-blood disease. This procedure is highly effective and could be used in other types of freshwater aquaculture.

Nitrite is relatively easy to measure, and fairly accurate results can be obtained with inexpensive test kits. In freshwater aquaculture, pond managers who measure elevated nitrite concentrations in waters of ponds or other culture systems can apply sodium chloride to increase the chloride concentration.

A chloride concentration 20 times greater than that for nitrite-nitrogen concentration will completely counteract nitrite toxicity in channel catfish and probably most other freshwater species. Sodium chloride treatment does not appear to be feasible in brackishwater and marine aquaculture.

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