The Toxicity of Silver to Marine Fish

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Salinity has a remarkable effect on Ag toxicity to aquatic organisms. The free Ag⁺ ion, which is only encountered in freshwater systems in traceable quantities, is one of the most toxic commonly occurring metals with a typical 96-h LC50 of 0.1 μM to fish (Hogstrand et al., 1996). In contrast, Ag is virtually non-toxic in brackish water, due to precipitation of Ag as cerargyrite, but in seawater where dissolved AgCl complexes prevail Ag is moderately toxic. The 96-h LC50 for Ag the tidepool sculpin (Oligocottus maculosus) and seawater acclimated rainbow trout (Oncorhynchus mykiss) in seawater ranges from 4-6 μM, depending on salinity (Ferguson et al., 1995; Shaw et al., 1996). Thus, Ag, added as AgNO₃, is 50 times less toxic in seawater than in freshwater.

The mechanism of acute Ag toxicity to freshwater fish is now relatively well delineated. Ag acts at the level of the gills where it seems to non-competitively block the Na⁺/K⁺-ATPase at the basolateral membrane (Ferguson et al., 1996; Morgan et al., 1996). This blockage drastically reduces the branchial Na⁺ and Cl⁻ uptake, resulting in a large net loss of Na⁺ and Cl⁻ from the blood (Wood et al., 1996). The subsequent sequence of events during acute silver toxicity to freshwater fish can be traced back to the progressive net loss of Na⁺ and Cl⁻ across the gills. In contrast to the well described etiology of acute silver toxicity in freshwater fish, the mechanisms behind toxicity of Ag to fish in seawater is unknown. Thus, the aim of the present study was to characterize the mechanisms that lead to acute toxicity of Ag in marine fish.

The experimental approach was similar to that of Wood et al. (1996). Starry flounders (Platichthys stellatus) were collected by bottom trawl in Barkley Sound off the west coast of Vancouver Island. The fish were fitted with chronically indwelling catheters in the caudal portion of the dorsal aorta to allow repetitive blood sampling. After surgery, the fish were placed in individual 10 l plastic tubs, supplied with a continuous flow (350 ml/min) of well aerated seawater with a salinity of 32 ppt and a temperature of 12 ± 1°C. The tubes were covered by a plastic mesh to avoid visual stress. The exposure system consisted of a header tank which delivered a constant flow (3.0 l/min) of aerated sea water to a 20-l vigorously aerated mixing chamber. Silver nitrate, dissolved in distilled water and acidified with 0.05% HNO₃, was dispensed from a stock solution into the mixing chamber by a peristaltic pump at a rate of 1.0 ml/min. The silver stock solutions were kept in a darkened bottle and renewed every 48 h. The concentrations of the stock solutions were set so that the concentrations of Ag in the tubes were 2.3 μM for one group of eight fish and 9.3 μM for a second group. A third group of eight fish served as sham treated control group. Blood samples were withdrawn from experimental fish and controls before the start of the exposure and then 12, 24, 48, 96, and 144 h after onset of exposure. The blood samples were analyzed for plasma Cl⁻, plasma glucose, plasma ammonia, plasma protein, blood gasses (PₐO₂ and PₐCO₂), pH, hematocrit, hemoglobin, and lactate.

While there were few physiological disturbances in starry flounders exposed to 2.3 μM Ag, the higher concentration of Ag, 9.3 μM, was in the lethal range. Three out of eight fish exposed to 9.3 μM Ag died between the 96-h and 144-h sampling points. The increased stress in fish within this group was reflected in a rapidly increasing plasma glucose concentrations (Fig. 1). At the 1- and 2-day sampling points there were slightly elevated plasma glucose concentrations also in the group exposed to 2.3 μM of Ag, but this effect was not found later in the experiment (Fig. 1).
Plasma ammonia was the only variable that markedly changed in fish exposed to the lower concentration, 2.3 μM, of Ag (Fig. 2). The plasma ammonia level peaked within the first two days of exposure, followed by a partial recovery. A similar increase and subsequent recovery was present in the group exposed to Ag at 9.3 μM. Thus, the plasma ammonia concentration did not increase dose-dependent fashion with increasing level of Ag exposure and it was partially restored in moribound fish (Fig. 2). Recently, Webb et al. (1996) found that Ag stimulates an increase in plasma ammonia levels also in freshwater fish, which establishes elevated plasma ammonia levels as a general effect of Ag exposure in fish. However, the lack of dose-dependency of the effect, together with the fact that the plasma ammonia level was on the decline later in the experiment when fish from the 9.3 μM Ag group started to die, strongly suggest that ammonia toxicity was not the primary cause of death in Ag exposed starry flounders. However, it remains possible that ammonia could contribute to the toxicity of silver in other fish species and during conditions that impair ammonia excretion (Shaw et al., 1996).
The pattern of plasma Cl⁻ concentration changes in Ag exposed fish was characteristic of a key toxic mechanism for a lethal effect. Whereas plasma Cl⁻ in stary flounders exposed to 2.3 μM of Ag was only slightly elevated in comparison to the sham-treated control, fish exposed to 9.3 μM of Ag showed dramatically escalating plasma Cl⁻ concentrations (Fig. 3). Furthermore, the individuals that died between the 96 and 144-h sampling point exhibited critically high plasma Cl⁻ concentrations. Thus, just as in freshwater fish, the mechanism of Ag toxicity to stary flounder in seawater seems to be a disturbance in the ability to regulate Cl⁻ (and presumably Na⁺). In seawater this disturbance is manifested by a net accumulation of Cl⁻ (rather than a net loss of Cl⁻ which is the case in freshwater fish) because of the higher concentrations of Cl⁻ in seawater than in the blood. To this point, the effect of Ag resembles that of Cu; in freshwater fish exposure to waterborne Cu results in reduced plasma Cl⁻ and Na⁺ levels and in seawater fish the plasma Cl⁻ and Na⁺ levels increase during Cu exposure (Stagg and Shuttleworth, 1982; Wilson and Tyler, 1993a,b).

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References


Questions & Answers: The Toxicity of Silver to Marine Fish

Q. DOMINIC DI TORO (Hydroqual, Inc.): At the high concentrations (mg/L) of silver you must be getting silver chloride precipitation.

A. No, we don’t. This is 32 parts per thousand seawater.

Q. DI TORO: At the concentrations we’ve operated at, you would be seeing it.

A. We actually measured the silver concentration in 0.45-micron filtered water in the tubs where the fish are located. We don’t see any silver chloride precipitation. As a matter of fact, we modeled it — not at 1 milligram per liter, but at 250 micrograms per liter — and it shouldn’t precipitate.

Q. NICHOLAS FISHER (SUNY-Stony Brook): Have studies been done that have examined the toxicity of dietary silver to either marine or freshwater fish? I ask in light of the implication of the intestine in the fish. I’m not aware of any studies.

A. There will actually be a presentation by Fernando Galvez about that dietary tomorrow. I should add we have also conducted work now in freshwater at close to environmentally realistic concentrations of silver to compare how the effects that we see at these industrial concentrations work at the lower concentrations. This is the obvious question that you ask yourself, and part of it has been done. We think that we probably want to do more of that.