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Gill lesions associated with acute exposure to ammonia

E.R. Cruz and G.L. Enriquez

The histopathological effects of the 96 hr LC50 at 20.65 ppm NH_3-N on the gills of milkfish fingerlings were examined qualitatively.

Histopathological lesions occurring in the gills of fish in all time exposures were essentially similar (Table 1) except epithelial hypertrophy which was observed only at 24 and 48 hr after treatment. A very early change was a pronounced enlargement or hypertrophy of the epithelial cells on the lamellae. Epithelial hypertrophy occurred along the entire length of the lamellae and was observed in almost all of the gills examined.

The epithelium also underwent slight hyperplasia of both the lamellar and basilamellar regions. At 72 hr, the entire interlamellar spaces became filled with hyperplastic epithelium.

In some filaments after 24 hr exposure, severe hyperplasia of the epithelium resulted in fusion of some of the lamellae at the distal portion of the filament. At 96 hr, fusion of the lamellae became more generalized along the entire length of the filament. The epithelium had proliferated beyond the tips of the lamellae.

The lamellae also underwent slight to severe epithelial lifting or detachment of the epithelial layer from the supportive cells. At 72 hr, the whole epithelial layer had completely detached from the supportive cells.

Blood-filled aneurysms or lamelar telangiectasis were also observed in most sections of gill tissue. In each of these swollen, rounded secondary lamellae, the pillar cell system was completely broken down and the epithelium enclosed a dis-organized mass of pillar cells and blood cells.

Table 1. Incidence of lesions in the secondary lamellae of the gills of milkfish (*Chanos chanos* Forsskal) fingerlings exposed to the 96 hr LC50 20.65 ppm NH_3-N over a 96 hr period. Three fish were examined per trial.

GILL LESION	24 hr			48 hr			72 hr			96 hr		
	I	II	III	I	II	III	I	II	III	I	II	III
Epithelial hypertrophy	3+ ***	3 ***	3 ***	2+ ***	1+ ***	2 ***						
Hyperplasia	3 **	2 **	3 **	2 **	2 **	2 **	3 **	2 **	3 **	1 **	2 **	1 **
Fusion			1 *	2 *			3 **	3 **	1 **	1 **	2 **	
Epithelial lifting	2 *	1 *				2 *			3 **		1 **	1 **
Lamellar telangiectasis	1 *	1 *	3 *	1 *	1 *	2 *	1 *	1 *	2 *	2 **	1 **	3 **
Disruption of the filamental system	1 *	2 *	1 *	2 **	2 **	3 **	3 ***	3 ***	3 ***	3 ***	2 ***	3 ***
Lamellar detachment		1 *	2 *		2 *	1 *			2 *	1 **	1 **	1 **
Vacuoles	1 **	2 **		1 **	2 **	1 **	2 **	3 **	3 **	1 **	2 **	2 **
Blood spaces	2 **	2 **	1 **	1 **	3 **	3 **	2 **	3 **	3 **	2 **	2 **	2 **
Mitotic figures		1 **	2 **		2 **	3 **	1 **	1 **	1 **	2 **	1 **	3 **

\+ Number of fish showing specific effect
* Rare occurrence
** Common occurrence
*** Extensive

After 24-72 hr exposure to the toxicant, one or two lamellae exhibited aneurysms. By 96 hr, multiple aneurysms or lamellar telangiectases occurred.

Disruption of the epithelium occurred at first in a few lamellae at the tip of the filaments. At 48 hr, disruption of the epithelial layer became more generalized along the entire length of the lamellae and more diffused over the gill. The basilamellar region was reduced in thickness. By 72 hr, there was complete disruption of the whole filamental system exposing the gill rays and the lamellar cell system.

Other remarkable changes noted for all time exposures were lamellar detachment, extensive vacuolation of the blood cells and presence of cells undergoing mitosis.

As in the present study, hyperplasia appears to be one of the initial reactions of the gill to various pollutants (Gardner & Yevich, 1970). The proliferation of the gill lamellae decreases the surface area of the gills, and consequently, the ability of the animal to absorb oxygen (Burrows, 1964). These may result in a reduction in the physical activities of the fish under conditions of stress or may cause afflicted fish to suffocate. Extensive proliferation of the gill epithelium was suggested as an adaptive measure to protect the gill filaments from continuous exposure to the toxicant (Eller, 1975; Smart, 1976).

Epithelial lifting was suggested as an acute inflammatory reaction (Skidmore & Tovell, 1972). The epithelial cells lining the secondary lamellae lifted away in a continuous sheet from the pillar cell system. This resulted in an increased diffusion distance from water to blood.

Aneurysms or lamellar telangiectasis was similar to those reported by Ashley (1972), Smith & Piper (1975) and Smart (1976). According to Roberts (1978), lamellar telangiectasis is a characteristic pathological change of the gill subjected to physical or chemical trauma. They are commonly found in farmed fish after pond transfer or in association with parasitic conditions, metabolic waste or chemical pollution. If there are several telangiectatic lamellae, respiratory function may be hindered, and if such fish are further traumatized, rupture and fatal haemorrhage may supervene (Roberts, 1978).

Disruption of the filamental cell system occurred due to lysis of its cells leaving the gill rays and capillaries exposed to the water. Eller (1975) and Mitchell *et al.* (1978) explained the mortalities at this stage as possibly due to asphyxiation, partial or complete loss of excretory or secretory function, or to loss of plasma electrolytes or proteins from open or exposed gill lesions.

Lamellar detachment appeared occasionally in some of the gill tissues examined. Detachment may be interpreted as weakening or necrosis of cells as a result of oxygen deficiency or poor blood circulation (Eller, 1975).

The observed increase in the number of mitotic cells in the gill of fish exposed to ammonia in the present study does not appear to have been previously described in fish poisoned with metabolic products. Increased mitotic activity was observed in gills exposed to Cadmium (Gardner & Yevich, 1970) and MeHgCl (Wobeser, 1975). Understandably, tissue repair by mitotic proliferation would occur. The apparent increase in the number of mitotic cells could be a response to epithelial damage.

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