GILL MELANIZATION AND HEAVY METALS IN FRESHWATER PRAWNS

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ABSTRACT

Continuous exposure (10-12 days) of freshwater prawn Caridina rajadhari to 100-200 ppb of cadmium chloride induced gill blackening and other histopathological changes in the gills. The black pigment associated with the gills was melanin. Mercury severely affected the gill structure but there was no melanization of the gills. In Macrobrachium kistensis there was no melanization of the gills after exposure to cadmium or mercury, while other histopathological effects were identical.

Studies on toxicity and histopathological effects of copper in freshwater prawns Caridina and Macrobrachium revealed that gills of both species of prawns were severely affected by this heavy metal. Of particular interest was

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the gill blackening reaction that was observed in *Caridina* but was absent in *Macrobrachium* (Ghate and Mulherkar 1979). In the further course of investigations static acute toxicity of cadmium and mercury was determined for *Caridina rajadhari* and *Macrobrachium kistnensis* following the standard procedures described earlier. Groups of 10-15 prawns were then exposed to each heavy metal for 10-15 days to study the effect on the gills. Concentrations of cadmium chloride used in this study were 100 and 200 ppb (μg/litre) for *Caridina* as well as *Macrobrachium*. Similarly, concentrations of mercuric chloride were 5 and 10 ppb.

The static 48-h LC50 values for cadmium and mercury are given in Table 1. The results indicate that mercury was more toxic than cadmium. It was further observed that 10-12 days exposure to cadmium chloride induced gill blackening in *Caridina* (Fig. 1A) while no such effect was observed in *Macrobrachium*. Exposure to mercuric chloride did not induce gill blackening in either species of the prawns. Histological structure of the gills of *Macrobrachium* and *Caridina* is identical and it has been described previously (Ghate and Mulherkar 1979). A section of the gill from control *Macrobrachium* is illustrated in figure 1B. Each gillplate or filament was found to be made up of respiratory epithelium enclosed in a thin cuticle. Except the gill blackening observed

<table>
<thead>
<tr>
<th>Species</th>
<th>Chemical</th>
<th>48h. LC50 (in ppb)</th>
<th>95% confidence limits in bracket</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Caridina</em></td>
<td>cadmium chloride</td>
<td>630.70</td>
<td>(458.00, 689.60)</td>
</tr>
<tr>
<td>rajadhari</td>
<td>hydrated</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>mercuric chloride</td>
<td>52.75</td>
<td>(40.51, 69.02)</td>
</tr>
<tr>
<td></td>
<td>hydrated</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Macrobrachium</em></td>
<td>cadmium chloride</td>
<td>569.60</td>
<td>(362.60, 894.20)</td>
</tr>
<tr>
<td>kistnensis</td>
<td>hydrated</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>mercuric chloride</td>
<td>27.96</td>
<td>(22.65, 34.36)</td>
</tr>
</tbody>
</table>

(The LC50 values of static acute toxicity tests are in terms of the salts of the respective heavy metal and not as metal ion.)

in *Caridina* exposed to cadmium, which showed typical coating of black material on some of the gillplates, the other histological changes were same in both species of prawns. The histopathological changes included dialatation of gillplates, breaking of cuticular lining, vacuolation and necrosis of the gill epithelium. (Fig. 1C). Histochemical test, Schmorl Reaction (Bancroft, 1975), carried out on sections of black gills from cadmium-treated *Caridina*, showed that the pigment associated with gillblackening was, at least in part, melanin. Mercury treatment induced only severe vacuolation and necrosis in the gills of both species of prawns. The effects were more pronounced than that of cadmium exposure. (Fig 1D). It is interesting to note that mercury did not induce gill
blackening in *Caridina*; probably it interfered with the process of melanin synthesis. Absence of gill blackening in *Macrobrachium* exposed to both heavy metals is inexplicable.

![Image](image_url)

**Fig. 1.** A: *Caridina coimbatorensis*, with branchiopore partially removed. Note blackened margin of the branchiopore and black gills (arrow) in the experimental prawn below. (X2). B: Section of gills from control *Macrobrachium*. Note cuticle (c) and respiratory epithelium (e). (X250). C: *Caridina* gills have same structure but the gills are smaller in size; d: Section of gills from *Caridina* exposed to cadmium. Note blackening of different filaments (arrow), cuticular damage and vacuolation of respiratory epithelium. (X300). D: Section of gills from *Macrobrachium* exposed to mercury showing severe vacuolation of respiratory epithelium. (X250).

Nimmo et al. (1977) first described pathological gill blackening in marine shrimps exposed to cadmium. Their findings are very similar to our observations on copper and cadmium induced gill damage in freshwater prawns, especially *Caridina*. Couch (1977) did electron microscopic study of black gills in the same marine shrimps as used by Nimmo et al. (1977) and found amorphous
black deposits in cell cytoplasm. He did not find any melanosomes or melanocytes and hence concluded that the black deposits may be of metallic sulfides or cadmium itself. He also stated that any agent capable of causing cell death in gill tissue may elicit autolytic response leading to black gills in these animals. Our observations with mercury, however, show that despite severe damage and necrosis of gill tissue there is no gill blackening in *Cardidina*.

Lightner and Redman (1977) investigated black gills of various species of marine penaeid shrimps and for the first time conclusively demonstrated histochemically (by Schmorl Reaction) that the brown-to-black pigment associated with the inflammatory reaction in these cases is melanin. The black gills investigated were from the shrimps exposed to copper or cadmium, shrimps infected with *Fusarium* mould and even from shrimps with ascorbic acid deficiency. In fact, ascorbic acid deficiency induces widespread melanization of connective tissue, gills and even the lining of fore- and hindgut (Magarelli et al 1979). Indeed, melanin is perhaps one of the most resistant pigments and, according to Unestam and Weis (1970), it has a bacteriostatic clotting and localising function. It is also possible that the melanin is protective against heavy metals since melanin can form complexes with heavy metals (Felix et al 1978).

Black gills and various other black lesions have been described in crustaceae (Delves-Broughton and Poupard 1976; Couch 1978). The exact aetiology of black gills is uncertain in some feral marine shrimps (Rinaldo and Yevich 1974). It is probable that all such black lesions are typical inflammatory reactions involving infiltration of the haemocytes that are probably responsible for melanin formation (see Lighter and Redman 1977). In our experiments also gills of prawns showed considerable infiltration by haemocytes. Recently even feral marine crabs, *Cancer irroratus*, were reported to be showing more or less similar histopathological alteration coupled with gill blackening (Greig et al 1982), although clear-cut cause and effect relationship could not be established. It appears that melanization is perhaps a widespread protective reaction in decapod crustaceae. Future research may throw some more light on the exact mechanism of the process involved.

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NOTES

REFERENCES


