

Gonadal Histopathology Following Nickel Intoxication in the Giant Gaurami *Colisa fasciatus* (Bloch and Schneider), a Freshwater Tropical Perch

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Large variety of data regarding toxic effects of nickel on mammals has been reported. Nickel refinery workers had increased mortality from lung or sinonasal cancers (Sunderman 1984). In human populations exposed to nickel and chromium compounds, chromosomal aberrations have been reported (Vainio and Sorsa 1981). Biochemical as well as histopathological alterations arising from nickel exposure in mammals have also been documented (Waltschewa et al. 1972; Clary 1975; Mathur et al. 1977). Vary scanty data is available concerning histopathological impact of nickel on fish tissues. However, to our knowledge, there are no reports available on the fish gonads of either sex encountered with nickel. The present study appears to be the first report on the topic.

Therefore, in the present investigation impact of nickel on the histological architecture of gonads of both sexes of a freshwater tropical perch, *Colisa fasciatus* have been assessed.

MATERIALS AND METHODS

Adult healthy specimens of giant gaurami, *Colisa fasciatus* (wt 5.52 ± 0.36 g; length 5.67 ± 0.28 cm) a freshwater tropical perch, were collected from local Ramgarh lake (Gorakhpur). Fish were permitted to acclimate under laboratory conditions for a period of fortnight. Fish were given food two to three times daily. Feeding was discontinued 24 hr before as well as during experimentation. Glass aquaria were filled with 15 L of tap water containing nickel sulphate ($\text{NiSO}_4 \cdot 7\text{H}_2\text{O}$) at a sublethal concentration of 64 mg/L or 2.279×10^{-4} moles/L (0.8 of 96-hr LC50) and five fish were placed in each aquarium. A control containing 15 L of tap water only along with five fish was concurrently run with each set. Media in each aquarium was renewed daily. Physico-chemical characteristics of the tap water were as follows: pH 7.57; temp. $24 \pm 1^\circ\text{C}$; dissolved oxygen content 7.6 mg L^{-1} ; hardness 172 mg L^{-1} as CaCO_3 ; electrical conductivity $100 \mu \text{ mho Cm}$. To investigate the

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impact of nickel sulphate on the gonads of both sexes following 96-hr exposure, testis and ovaries of fish from experimental as well as control aquaria were extirpated and fixed in aqueous Bouin's solution for 24 hr. Thereafter, tissues were routinely dehydrated in graded series of alcohols cleared in xylene and embedded in paraffin wax. Sections 4-6 μ m thick were cut, processed and stained with hematoxylin and eosin.

RESULTS AND DISCUSSION

Testis in C. fasciatus were paired elongated structures closely attached to the dorsal abdominal wall with the help of mesorchium. Histologically testis possess lobules of various shapes and sizes held together by thin connective tissue. Between the lobules, presence of interstitial cells and blood vessels was observed (Fig. 1 and 2). As the observations were made in the breeding period of the fish, testis were fully mature (Fig. 1) and seven stages of germ cells in the process of spermatogenesis were seen, viz. (i) sperm mother cells (ii) primary spermatogonia, (iii) secondary spermatogonia, (iv) primary spermatocytes, (v) secondary spermatocytes, (vi) spermatids, (vii) sperms (Fig. 2). Ovaries are also paired structures attached to the body wall with the help of mesovarium. At the time of the experiment the ovaries were fully mature, containing mature oocytes packed compactly thereby reducing the interspaces. Atretic oocytes were rarely observed. Few immature oocytes are also encountered (Fig. 5). Testis of the nickel exposed C. fasciatus exhibited marked degenerative alterations. There was general disorganization in the testicular lobules. Spermatogenic activity was severely reduced (Fig. 3). Germ cells in the testicular lobules were in the process of degeneration (Fig. 4). Degeneration of the lobules exhibiting collapse or rupture was apparent (Fig. 3). Blood vessels were congested. After 96-hr treatment with a sublethal concentration of nickel, ovaries presented significant alterations in the histological architecture which were exhibited by the occurrence of comparatively large inter-follicular spaces which appeared to have been formed due to the shrinkage of the oocytes. Several oocytes were observed in the process of absorption, i.e. atretic oocytes (Fig. 6).

Fish C. fasciatus, when exposed to nickel, exhibited extensive damage in their testis. Earlier Waltschewa et al. (1972) have reported testicular abnormalities after long term administration of nickel sulphate to rats. Rupture of the testicular lobules has been observed in the present study. Similar observations have been reported from fish exposed to arsenic (Shukla and Pandey 1984). Reduction in the spermatogenesis and degeneration of the lobules evidenced in C. fasciatus have been reported earlier following treatment of the fish with cadmium (Wani and Latey 1982) and zinc,

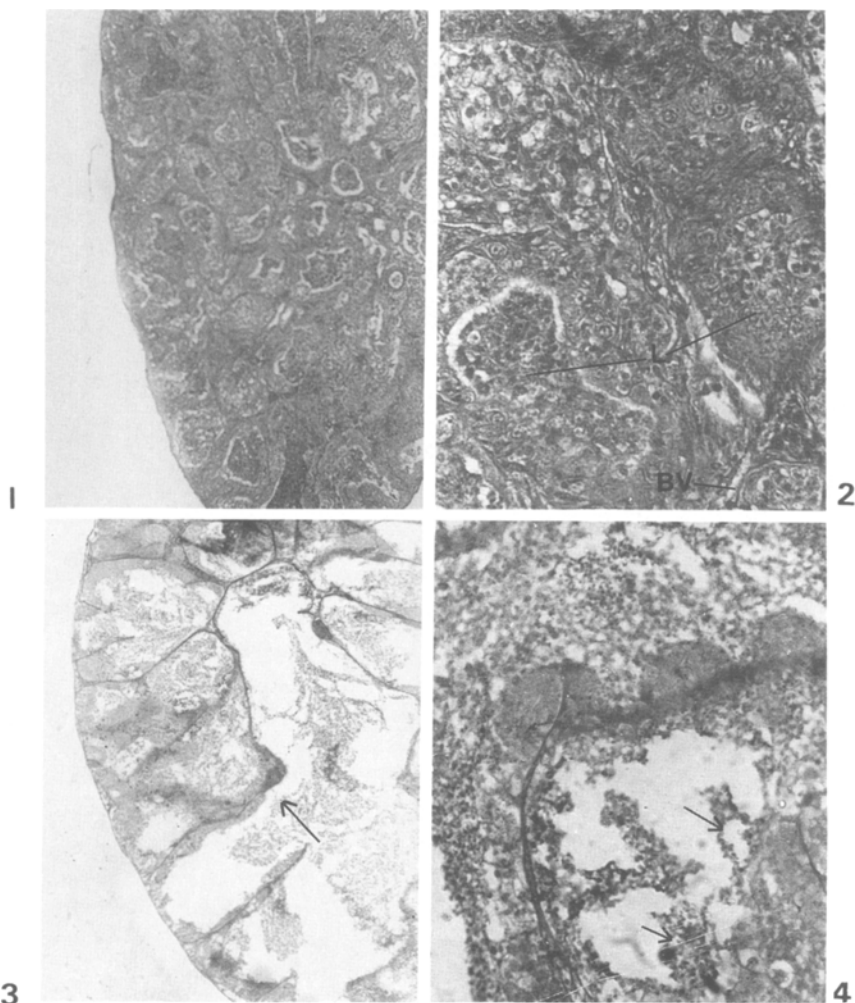


Figure 1. Photomicrograph of testis of normal C. fasciatus. HE x 160.

Figure 2. Photomicrograph of testis of normal C. fasciatus. HE x 640. L = Testicular lobules, BV = Blood vessel

Figure 3. Photomicrograph of the testis of C. fasciatus after 96 hr exposure to 64 ppm nickel, exhibiting lobular degeneration and rupture (arrow). HE x 640.

Figure 4. Photomicrograph of the testis of C. fasciatus, after 96 hr exposure to 64 ppm nickel, exhibiting degeneration of the germ cells in the testicular lobules (arrows). HE x 640.

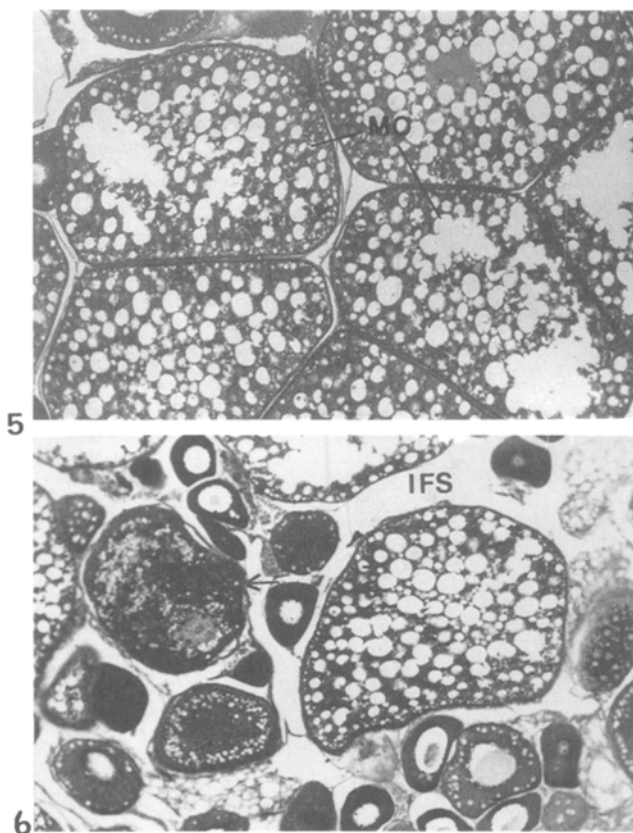


Figure 5. Photomicrograph of ovary of normal *C. fasciatus*. HE x 640. Mo = Mature oocytes

Figure 6. Photomicrograph of the ovary of *C. fasciatus*, after 96 hr exposure to 64 ppm nickel, exhibiting large interfollicular spaces (IFS) and oocytes in the process of absorption i.e. atretic oocytes (arrow). HE x 640.

copper and lead (Kumar and Pant 1984). Like C. fasciatus, reduction in the spermatogenesis and tubular degeneration have also been reported in the testis of rats exposed to nickel (Mathur et al. 1977) and pesticides (Dikshith and Datta 1972). Distension of the blood vessels engorged with erythrocytes was observed in the present study-an observation made earlier by Sangalang and O' Halloran (1973) on the testis of brooktrout exposed to cadmium. Significant histopathological alterations were observed in the ovaries of C. fasciatus when exposed to sublethal concentration of nickel. The prominent changes were - occurrence of atretic oocytes and increase in interfollicular spaces. The abnormalities encountered in the present study have also been reported by a large number of workers from fish exposed to a variety of toxicants (Saxena and Garg 1978; Wani and Latey 1982; Ram and Sathyanesan 1983; Kumar and Pant 1984; Nath 1985).

The mechanism of action of heavy metals in relation to their toxic impact on fish gonads is not well known and requires further investigation. However, various workers have attempted to describe different mechanisms of action. Kumar and Pant (1984) have attributed direct effect of heavy metals on fish gonads, besides their possible action through pituitary. Sangalang and O'Halloran (1973) have opined alterations in steroid synthesis in the case of cadmium exposed brooktrout. Ram and Sathyanesan (1983) suggested inhibition of the action of pituitary gonadotrophs and somatotrophs in Channa punctatus exposed to mercury.

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