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Histopathological changes produced in the Liver and Kidney of *Channa punctatus* after Chronic Exposure to 2,3',4-Triaminoazobenzene

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Aminoazo dyes are known to be common carcinogens in mammalian animal models (MAISIN et al. 1957, BANNIKOV et al. 1973). The carcinogenic azodyes interfere with mechanisms responsible for the regulated formation of specific cellular proteins by binding themselves to the hepatic microsomes (MILLER & MILLER 1953, HULTIN 1959). RIBINOVITCH et al. (1961) have reported alkaline ribonuclease activity in rat kidney cortex and liver after the administration of Trypan-Blue and other azodyes.

Recently, DECLOITRE et al. (1975) have reported the effect of phenobarbitol on carcinogenic metabolism in rat liver caused by p-dimethylaminoazobenzene. TRUHAUT & FERRANDO (1975) have worked out physiology of some tissues of mammals after the administration of different doses of two azodyes, Amarnath and Sunset Yellow. CARRUTHERE et al. (1977) reported hepatoma induced in the rat by the administration of 3'-methyl-p-dimethyl-aminoazobenzene.

The histopathogenesis of carcinoma caused by the azodyes treatment in mammals has been worked out extensively. However, almost nothing is known of toxicity on fishes of azodyes, which are periodically drained out as residual wastes from various industries (textile etc.) using azodyes. The dyes drained out sometimes render a critical chemico-azo stress in inland water reservoirs, which cause great mortality among inhabiting fishes. The present communication deals with the histopathological effects on hepato-renal tissues in Channa punctatus after chronic exposure to 2,4-diamino, 3-aminoazobenzene (DAAB), Bismark Brown.

MATERIAL AND METHODS

Living fishes (40 to 60 g) were procured from local freshwater resources. After acclimitizing to laboratory conditions for one week, the fishes were divided into two groups each including 10 fishes. The fishes of experimental group were exposed to sublethal concentration of dye (0.0025%) by bath. The control fishes were kept in ordinary tap water. Both the aquaria of fishes of control and treatment groups were covered

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and sealed with black paper to avoid any possible photo-oxidation of the dye. The absorbency of the azo group in the dye was determined before and after the experiment. There was no detectable change in the wavelength of absorption maxima throughout the experiment and showed no structural change in the dye molecule as well as in its concentration. Fishes of both the groups were sacrificed after one month and the tissue samples of liver and kidney were fixed in Rossman's fluid, neutral formalin and chilled acetone. 5 to 7 micron thick sections were cut after paraffin embedding and were stained with haemotoxylin and eosin. The histological changes in experimental sections were studied in comparison to control sections.

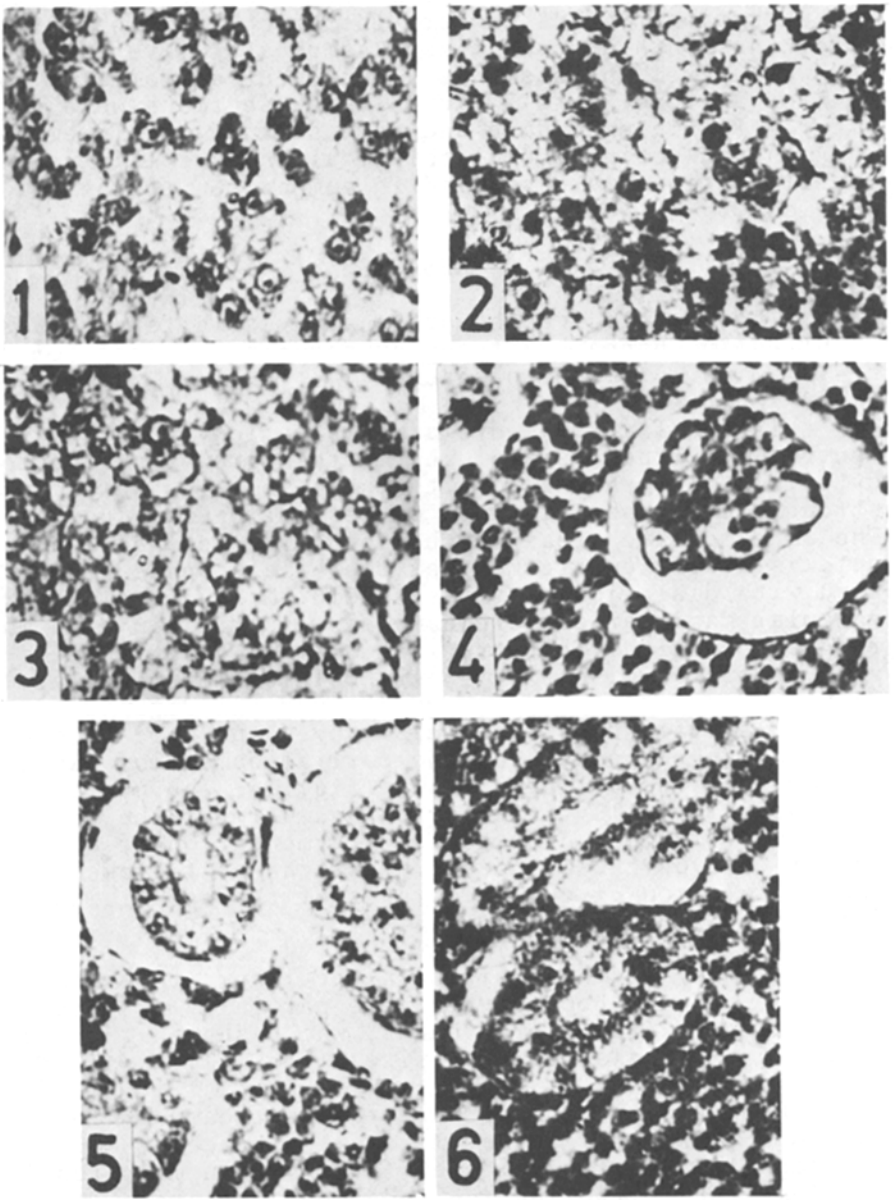
RESULTS

The histological hepatorenal dysarchitecture was resulted in Channa punctatus by chronic exposure to 2,4-diamino, 3-aminoazobenzene.

In the liver the dye administration caused the centrolobular necrosis of the certain groups of hepatocytes. In the central zone the hepatic cells were strongly eosinophilic, more or less irregular in shape. The intermediate areas of the lobular zones showed characteristically enlargement of the bilary space associated with dialation of the sinusoid. Many interlobular vascular capillaries were damaged resulting in a haemorrhagic condition. The cytoplasm of many hepatic cells got vacuolated disposing their nuclei excentrically. Many hepatocytes were seen under hydropic degeneration. The hepatocytic cords were disarrayed and were irregularly arranged because of the partial damage of hepatic connective tissue binding them (Fig. 1). The nuclei in some hepatic cells got enlarged and were at the merge of damage. In most hepatocytes fragmentation of enlarged nuclei (karyopycnosis) occurred which was followed by the dissolution of cell membranes of the hepatic cells (Fig. 2).

At many necrosed sites in the liver, the remaining scars of the hepatocytes were partially or completely replaced by the fibral bands, which showed the occurrence of cirrhosis at these sites (Fig. 3).

Renal damage in response to dye treatment resulted in the shrinkage of glomeruli and the space in Malpighian capsular body was widened (Fig. 4). In many renal tubules, the epithelium too underwent shrinkage, thus departing the cells from the basement membrane. At many places the coalescing and then dissolution of basement membranes of two adjacent renal tubules was also observed (Fig. 5). The cytoplasm of the epithelial cells



Figures showing histopathological lesions in the liver (1-3) and kidney (4-6) of Channa exposed to 2,4-diamino, 3-aminoazobenzene.

renal tubules was found to be clear and under hydropic degeneration. In many tubules the adjacent epithelial cells underwent coalescing by the dissolution of intercellular membranes. There were no well defined nuclei in most of these epithelial cells of the renal tubules. In many of these cells nuclei underwent pycnosis (fragmentation), while in others even fragments disappeared. Brush borders of almost all the renal tubules were deformed and damaged as such the renal tubules became insignificant so far as their function was concerned (Figs. 5 & 6). Thus, more or less complete damage of most renal tubules, including damage of epithelium and brush borders, and shrinkage of glomeruli, was resulted by the long-term azodye treatment (Fig. 6).

DISCUSSION

2,4-Diamino, 3-aminoazobenzene exposure resulted characteristic deformative changes in the histology of liver and kidney. In the liver it resulted significant changes like enlargement of biliary spaces, disarraying of hepatocytes' cords and vacuolization of cytoplasm with excentric enlarged nuclei at the merge of damage in many hepatocytes. MILLER & MILLER (1953) have shown progressive microscopic alterations in the liver of rats fed on 3'-methyl-4-dimethylaminoazobenzene and 4'-fluoro-4-dimethylaminoazobenzene, which are more or less identical to our results. Vacuolization of cells and disarraying of hepatocytic cords were reported to occur in response to endrin exposure by SASTRY & SHARMA (1978). CHRISTIE & LEPAGE (1961) as well observed enlargement of size and variation in the diameter of the nuclei of hepatocytes and renal tubule epithelial cells in animals after feeding them with dimethylnitrosamine. The dialation of the bile canaliculi and diminution of the size and number of the microvilli in the liver during thioacetamide intoxication were demonstrated by ROUILLER & SIMON (1962). Damage of renal tubules in the kidney of Colisa fish during lindane treatment has been revealed by VERMA et al. (1975). The fragmentation of nuclei in liver and renal tubule epithelial cells during the present study are confirmed by the findings of CARRUTHERE et al. (1977). According to GINZLER (1946) the administration of di- and trimethyl phenazonium chloride results selective necrosis of the proximal convoluted tubules with severe disturbances of kidney function and mild degenerative changes in the liver.

The exposure of fish to sublethal concentration of azodye used, though has not been able to provoke tumors in liver and kidney as shown by STEWART & SNELL (1957), but renders necrotropic deformative changes in these tissues. These changes are probably due to a disturbance in the enzyme pattern of the organ. According

to BERENBOM et al. (1955) most of the enzymes, oxidative enzymes especially, decrease with time during dye intoxication and lead to necrotropic changes in tissues. Our unpublished results confirm the above explanation for the necrotropic changes as the activity of lactic dehydrogenase in liver and kidney decreased substantially during DAAB chemico-azo stress. GOEL et al. (1978) have also shown suppressed alkaline phosphatase reaction in liver and kidney of Channa during congo red intoxication.

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