TABLE I

Isolation of retinyl ester from the intestines of Channa gachua *after administration of β-carotene*

No. of fish used	Total amount of β-carotene administered (μg)	Time between administration and killing (h)	Total amount of β-carotene recovered (μg)	Total amount of retinyl ester formed (μg)
2	1160	4	33	52
5	1740	4	24·8	80·2
4	2320	4	696	116·4
1	580	5	22	65·9
4	2320	5	51·2	83·5
1	580	6	..	40·6
6	3480	6	756	178·5
7	4060	6	864	68·5
8	4640	6	584	145

predominant fish could convert β-carotene into retinoic acid and occasionally into retinol, it was considered worthwhile to study the metabolism of β-carotene in *C. gaucha* a retinol rich freshwater fish in the same way as was done in the case of *H. fossilis*.

Channa gachua like other mammalian and avian species can convert β-carotene into retinol. Here we fail to isolate retinoic acid which may form from β-carotene in the intestines of β-carotene administered fish. From these experiments it can be seen that *C. gachua* behaves like most animal species. The exception found in *H. fossilis* which appears to lack of retinaldehyde reductase to have a lot of aldehyde oxidase and not so much enzymes catabolising retinoic acid. There may be every possibility of formation of retinoic acid, but probably it has much less aldehyde oxidase; and the small amount of retinoic formed is rapidly catabolised, so it was not easy to detect in the intestinal extracts after the administration of β-carotene in *C. gachua*.

Although it cannot be generalised from the present and the previous studies that retinol predominant fish can convert β-carotene into retinol, yet we think that like other mammalian and avian species β-carotene may act as best precursor in retinol predominant freshwater fish. Further it can be concluded from the present studies that fish are capable of conversion of β-carotene into either retinol or retinoic acid, but are unable to convert β-carotene into dehydroretinol.

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EFFECT OF ZINC ON ADENOSINE TRIPHOSPHATASE ACTIVITY IN THE GILLS OF *CHANNA FUNCTATUS* (BLOCH)

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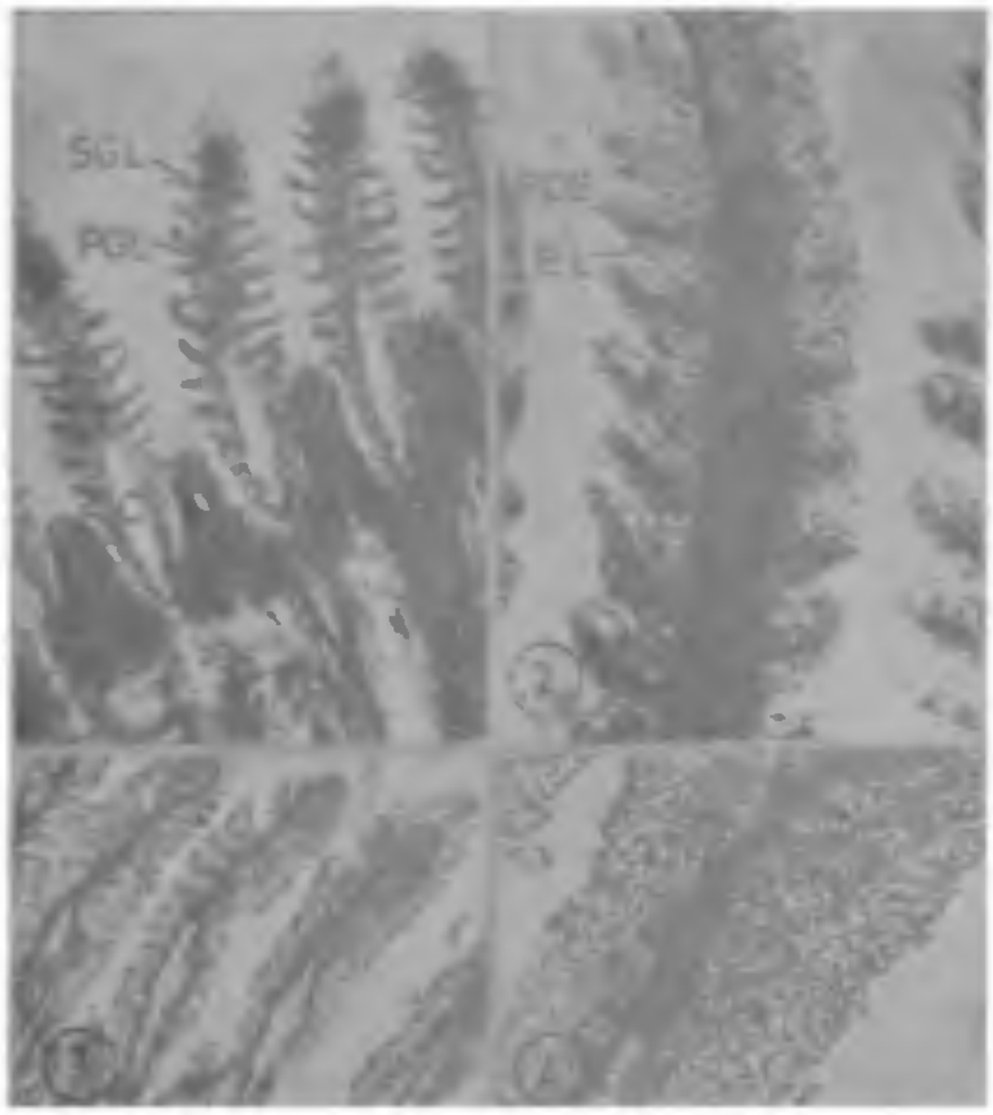
SOME of the freshwater reservoirs in and around Udaipur receive zinc and other electrolytes from industrial effluents[1]. The toxic effects of zinc ions on edible fishes are poorly understood[2]. Further, very little is known about the zinc toxicity induced enzymological changes in the vital organs such as gills[3].

The present work reports the changes in the histochemical activity and distribution of adenosine triphosphatase (ATPase) in the gills of an edible teleost *Channa punctatus* (Bloch).

Fish weighing 2·4 g with total length of 58 mm were collected locally and acclimatised to laboratory regimen for 10 days. They were challenged with 56 ppm of Zn for 8 and 24 hr of exposures. Controls



FIGS. 1–4. Photomicrographs of T.S. of gill filaments of C. *punctatus* showing the ATPase activity. Fig. 1. Control fish (× 40). Fig. 2. At 56 ppm of Zn after 8 hr of exposure (× 100). Figs. 3–4. After 24 hr of exposure (× 40; × 400). PGL: Primary gill lamellae; SGL: Secondary gill lamellae; PCS: Pillar cell system; EL: Epithelial layer; GAR: Gill arch region; and C: Cartilaginous cell.)

were also run simultaneously under identical conditions. Gills of control and Zn-challenged fish were extracted and washed several times in chilled water to remove blood and mucus. They were fixed in ice-cold neutral formalin (10% at 4° C) for 6–12 hr. Frozen sections (10–15 μM) were processed according to Padykula and Herman[4–6].

Comparison of ATPase activity in the gill lamellae and secondary gill lamellae of control and Zn-treated *C. punctatus* showed variations. In the control fish, the cartilaginous cells were ATPase negative, but intense and uniform enzyme reaction was observed in the gill filament, secondary lamellae, gill-arch region, pillar cell system and epithelial layers of secondary gill lamellae (Fig. 1).

Zinc caused histological deformities at 56 ppm after 8 hr of exposure and increase in ATPase activity was observed in gill filament and to some extent also in the secondary gill lamellae and pillar cell system. The epithelial layers in all the secondary gill lamellae were separated from the pillar cell system and showed intense ATPase activity (Fig. 2).

After 24 hr of exposure, the histopathological conditions were accentuated in the secondary gill lamellae along with an increase in enzyme activity. The cartilaginous cells of the primary gill lamellae also showed increased ATPase reaction (Figs. 3–4).

ATPase activity in the gills of untreated fish was normal as compared to Zn-treated ones where it was intense. This may be due to the increased stress induced by Zn toxicity leading to suffocation and "knocking off" of the basal metabolic rate of the gill epithelium. In the control fish, the degree of ATPase staining represents threshold concentration of this enzyme necessary for the normal functioning of gills. That many metallic ions including zinc are considered "enzymicidal" in nature[6] is not in agreement with the present finding.

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TRANSUTERINE MIGRATION OF THE EMBRYO IN THE BAT, *MINIOPTERUS SCHREIBERSII FULIGINOSUS* (HODGSON)

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TRANSUTERINE migration of the embryo has been reported to occur in several eutherian mammals[1]. Among bats this phenomenon has been noticed as an exception in some species[2–4] and as a normal feature in a few species[5–8]. In the former case, as noticed in *Myotis lucifugus lucifugus*[4], while ovulation may occur from either ovary, blastocyst implantation takes place invariably in the right uterine cornu, and in the latter case, as represented by most species of *Miniopterus*[4–8], ovulation occurs invariably from the left ovary but the conceptus is invariably carried in the right uterine cornu. Baker and Bird[8], however, reported that in *Miniopterus australis*, whereas pregnancy was carried in the right uterine cornu in most cases, there were three specimens in which the embryo was in the left