oxygen demand by the former muscles. It may be worthwhile to mention in this context that the blood pressure in the left ventricle of the chicken is higher than that of the right one. As shown in the table, compared to the duck and coot, the diving datchick has the highest myoglobin concentration both total and in terms of individual chambers. It has been shown that in porpoise, a diving marine mammal, the myoglobin concentration in the heart is far greater than in any terrestrial mammals. It should be emphasized that the difference in myoglobin concentration between the two chambers of the ventricle has a bearing on the functional dichotomy exhibited by them, as the right ventricle pumps blood to shorter distance than the left one.

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**ENZYMOLOGICAL ANOMALIES IN LIVER AND KIDNEY OF CHANNA PUNCTATUS AFTER 2', 4'-DIAMINO, 3'-AMINOAZOBENZENE (DAAB) EXPOSURE**

Several hepatotoxic-nephrotoxic dyesstuffs are known to affect enzymes. For example, administration of a mixture of dimethyl and trimethyl phanzenium chloride to rabbits causes selective necrosis of the proximal convoluted tubules with severe disturbances of kidney function and mild degenerative changes in liver. Goel and Garg recently reported hepatorenal histo-dysarchitectures among fishes induced by 2', 4'-diamino, 3'-aminoazobenzene exposure.

Many dyesstuffs particularly azodyos are known to be carcinogenic to mammals. The toxic role of azodyos is of great concern in fish management as industrial wastes from textile industries cause mortality among many palatable fishes. The present paper deals with the enzymological anomalies in the liver and kidney in *Channa* during treatment with 2', 4'-diamino 3'-aminoazobenzene (DAAB).

**Material and methods**

Live fishes (40 to 80 g) after acclimatizing to laboratory conditions for one week at 29 ± 2°C and 6.8 pH were divided into 3 batches, each consisting of 25 fishes. The fishes of 1st and 2nd batch were treated with sublethal concentrations of 0.0031 and 0.0025% of 2', 4'-diamino, 3'-aminoazobenzene (DAAB) respectively by bath (LC50 being 0.065% for 96 h). The fishes of 3rd batch served as controls and were kept in tap water. The fishes of 1st batch, were designated as those of short term (acute) exposure and enzymological analysis was made after 48 or 96 h of treatment. On the 2nd batch (chronic exposure) the enzymological analyses were carried out after 15 or 30 days. Both the aquaria of control and experimental fishes were seal-covered with black paper to avoid any possible photo-oxidation of the dye.

Acid and alkaline phosphates were assayed following the method of Morton. 5-nucleotidase and glucose-6-phosphatase were estimated according to the method of Heppel and Hillman and Swanson respectively and the activity was expressed in terms of mg of phosphorus liberated per hour per g of fresh wt. of tissue. Lipase was estimated titrimetrically following modified method of Levy. Amylase and urease were assayed by the methods of Street and Close and Halfman et al. respectively. The experiments were conducted at 29 ± 2°C and 6.8 pH. The data shown is an average of observations from 10 fishes in each case.

**Results**

The comparative data of enzymological analyses of liver and kidney of control and treated fishes are shown in Table I.

It is clearly indicated that the phosphatases (alkaline phosphatase, acid phosphatase, 5-nucleotidase and glucose-6-phosphatase) and lipase reactions were increased in the liver and kidney after acute and chronic exposure to dye significantly (P < 0.01). However, remarkable increase in urease activity was seen during chronic exposure but not during acute exposure. Amylase showed suppressed activity during dye exposure and the decline in enzyme activity was significant (P < 0.05) in liver during chronic exposure of the dye.

**Discussion**

2', 4'-diamino, 3'-aminoazobenzene induces an increase of phosphatases' activity including acid- and
### Table I

*Showing enzymology of liver and kidney of control and treated fishes*

<table>
<thead>
<tr>
<th>Enzymes</th>
<th>Control</th>
<th>Acute exposure 96 hours</th>
<th>Chronic exposure 30 days</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LIVER</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alkaline phosphatase*</td>
<td>$2.30 \pm 0.36$</td>
<td>$3.05 \pm 0.79$</td>
<td>$4.99 \pm 1.27$</td>
</tr>
<tr>
<td>Acid phosphatase*</td>
<td>$2.59 \pm 0.01$</td>
<td>$3.22 \pm 0.072^b$</td>
<td>$5.08 \pm 0.36^b$</td>
</tr>
<tr>
<td>5-nucleotidase*</td>
<td>$2.10 \pm 0.02$</td>
<td>$1.65 \pm 0.03^b$</td>
<td>$2.40 \pm 0.023^b$</td>
</tr>
<tr>
<td>Glucose-6-phosphatase*</td>
<td>$2.66 \pm 0.005$</td>
<td>$8.16 \pm 0.008^b$</td>
<td>$9.57 \pm 0.627^b$</td>
</tr>
<tr>
<td>Lipase (units/g)</td>
<td>$1.51 \pm 0.74$</td>
<td>$5.45 \pm 0.40$</td>
<td>$10.20 \pm 0.362^b$</td>
</tr>
<tr>
<td>Amylase (units/g)</td>
<td>$4.47 \pm 0.50$</td>
<td>$4.15 \pm 0.003$</td>
<td>$2.70 \pm 0.051^a$</td>
</tr>
<tr>
<td>Urease (units/g)</td>
<td>$1.16 \pm 0.004$</td>
<td>$1.68 \pm 0.002^b$</td>
<td>$4.56 \pm 0.009^b$</td>
</tr>
<tr>
<td><strong>KIDNEY</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alkaline phosphatase*</td>
<td>$1.12 \pm 0.53^*$</td>
<td>$2.30 \pm 0.24^b$</td>
<td>$4.63 \pm 0.146^b$</td>
</tr>
<tr>
<td>Acid phosphatase*</td>
<td>$3.06 \pm 0.736$</td>
<td>$3.90 \pm 1.01$</td>
<td>$4.80 \pm 0.266$</td>
</tr>
<tr>
<td>5-Nucleotidase*</td>
<td>$1.50 \pm 0.066$</td>
<td>$3.25 \pm 0.001^b$</td>
<td>$4.70 \pm 0.381^b$</td>
</tr>
<tr>
<td>Glucose-6-phosphatase*</td>
<td>$1.40 \pm 0.06$</td>
<td>$10.80 \pm 0.13^b$</td>
<td>$11.62 \pm 0.058^b$</td>
</tr>
<tr>
<td>Lipase (units/g)</td>
<td>$1.15 \pm 0.10$</td>
<td>$4.31 \pm 0.312^b$</td>
<td>$8.33 \pm 0.84^b$</td>
</tr>
<tr>
<td>Amylase (units/g)</td>
<td>$5.78 \pm 0.00$</td>
<td>$4.32 \pm 0.55$</td>
<td>$4.20 \pm 1.10$</td>
</tr>
<tr>
<td>Urease (units/g)</td>
<td>$3.13 \pm 0.010$</td>
<td>$3.94 \pm 0.009^b$</td>
<td>$8.89 \pm 0.12^b$</td>
</tr>
</tbody>
</table>

All values are mean ± S.E. of 10 determinations (animals).

* Activity expressed in mg of phosphorus liberated per hour per g fresh tissue.

$a, P < 0.05$, $b, P < 0.01$.

Alkaline phosphatase and glucose-6-phosphatase. However, 5-nucleotidase in liver shows a decrease during acute exposure reviving to almost normal during chronic exposure. Maximum increase has been recorded in case of glucose-6-phosphatase which shows increased activity by four times. Increased alkaline phosphatase reaction has also been reported by Firminger\(^1\) after azodye intoxication. However, Mulay and Firminger\(^1\) have reported the acid phosphatase activity varying from low to normal values during azodye poisoning. The increase of phosphatases' activity is considered to be resulted because of tissue inflammatory reactions of DAAB toxin, and these results are supported by similar findings of Schmidt and Schmidt\(^1\). Increase of many liver specific enzymes during decumarol poisoning has been shown by Wrobiewski and Manso\(^1\). However, Kaneko et al.\(^3\) and Onoe et al.\(^12\) have shown the variable activity of GS\(\text{Pase}\) with histological alteration of hepatocytes during 3'-methyl, 4'-diaminoazobenzene (MDAB) intoxication. According to them the enzymatic activity decreases during the first phase which begins to increase again parallel to the proliferation and maturation of the renewed hepatocytes. Our results for lipase activity are more or less similar to the findings of Kaneko et al.\(^3\). Like phosphatase, urease in both the tissues worked out has increased indicating the increased breakdown of urea in response to DAAB treatment. Elevated level of phosphatases, amylase, urease in the plasma of *Channa* under chemo-azo stress of 2', 4'-diamino, 3'-aminoazobenzene has also been reported by Goel and Garg\(^4\).
Financial assistance for the present study from UGC, New Delhi, is thankfully acknowledged.

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6. Hafmann, Ed. and Schmidt, W., Biochem. Z., 1953, 324, 125.

The Unusual Occurrence of the Male of Pseudochioropinus narcinae on the Gill Filaments of Narcine timlei

Minute and short-lived, the lernaeopodid male is usually seen near the female genital apertures and only very rarely attaches itself to the host tissues. The female Pseudochioropinus narcinae (Pillai) is parasitic on the gill arches of Narcine timlei (Bloch and Schn.). Recently we obtained, along with several females, a few males two of which were unusually found attached directly to the gill filaments of the host, a very rare phenomenon. The morphology of the male of P. narcinae has already been described.

These males along with the gill filaments were fixed in 10% neutral-buffered formalin, processed and paraffin sections cut at 5-7 μ were stained with iron haematoxylin and cosin for the present study.

Both the males were found attached to the lateral margin of the primary gill filaments by their powerful maxillipeds (Fig. 1) which pierced through the epithelium (capping tissue) and the underlying thick connective tissue thereby establishing a firm grip with the aid of the disto-medial process and the sub-chela (Fig. 2). In both the males the second maxillae, though subchelate, were not seen attached to the gill tissue.

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Fig. 1. A portion of the gill filament of Narcine timlei with the attached male of P. narcinae. (1 : gill filament.)

Literature pertaining to the pathology of infection by lernaeopodid copepods is meagre and is limited to the works of Friend, Hoshina and Suenaga, Kabata, and Kabata and Couvans. None of these describes the nature of damage caused by a male lernaeopodid. In the present instance the damage is purely mechanical caused by the maxillipeds and the tissue affected is the epithelium wherein a portion of the capping tissue with mucous cells is destroyed thereby exposing the underlying connective tissue (Fig. 3). Hypertrophy of the connective tissue is not evident. The erosion of the capping tissue, though not severe in itself, may become “open gates” (Kabata and Couvans) for secondary microbial infection by fungi and bacteria.