The environment is currently polluted by thousands of chemicals or xenobiotics introduced into the environment by man to meet the demands of modern era. The pollution is continuous and alarming influx to aquatic environment worldwide from both naturally occurring and anthropogenic resources. The polluted water may lead to the destruction of the beneficial species either directly by effecting the aquatic forms of life or indirectly through breaking the biological food chains such as fish and their habitat and behavioural pattern. The fish as a bio indicator of aquatic medium, plays an important role in the monitoring of water pollution because of sudden death of fish indicates heavy pollution and the effects of exposure to sub lethal levels can be measured in the terms of biochemical, physiological and histological responses of the fishes.

**MATERIALS AND METHODS**

Living specimen of *Colisa fasciatus* were collected from local fresh water resources and acclimatized in laboratory conditions for a minimum period of seven days before experimentation. Visibly healthy fishes were selected and treated with 0.1% KMnO4 solution and divided into five batches. One batch was kept in water and was used as control. The remaining four batches were kept in acute and chronic concentrations of metanil yellow and bismark brown. Water was replaced periodically and black paper was used to prevent any possible photo-oxidation of the dyes. The fishes of all batches were sacrificed at 48 hrs. and 96hrs. (acute exposure) and 15 days and 30 days ( chronic exposure ). The blood was collected from cut caudal vein and was allowed to clot at room temperature and then centrifuged at 2000 rpm. The `t` test of Fisher was used to calculate the significance of data.

**RESULTS AND DISCUSSION**

Significant alteration in succinic dehydrogenase activity was observed in the blood of *Colisa fasciatus* exposed to metanil yellow and bismark brown (Table: 1). The decreased activity in the blood serum of *Colisa fasciatus* was found to be -18.72%, - 25.29%, - 43.74% and -64.89% in response to metanil yellow and -20.70%, -37.89%, - 54.63% and -71.64% in response to bismark brown upon acute (T1 and T2) and chronic (T3 and T4) exposures respectively. All the results were statistically highly significant (P < 0.01).

Succinic dehydrogenase is a mitochondrial respiratory enzyme which catalyze the conversion of succinate to fumerate in citric acid cycle. Kreb's cycle function in the inner membrane of mitochondria, is a major pathway for the generation of ATP molecules. SDH is a primary enzyme in the oxidative catabolism of sugars and as such is used effectively as a marker of mitochondrial abundance and activity. Decreased succinic dehydrogenase activity in the liver of *C. batrachus* was reported upon exposure to congo red and bismark brown. Significant decrease in SDH activity was reported in liver, brain and gills of *C. batrachus* intoxicated with endosulfan. Decreased SDH activity was reported in brain, gills, intestine and kidney of *C. punctatus* exposed to the sub lethal concentrations of three heavy metals mercury, nickel and chromium. Inhibited SDH activity was reported in gills, intestine, liver, kidney, muscles and blood of *H. fossilis* under the stress of lead nitrate. Decrease in SDH activity was found in liver and muscles of *C. punctatus* exposed to cycloart- 24-en- 3 B- ol
Table-1. Alteration in the activity of succinic dehydrogenase (sdh) induced by metanil yellow and bismark brown in the blood of colisa fasciatus. values are mean +s. e. of nine observations each)

<table>
<thead>
<tr>
<th>DYES</th>
<th>C</th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>T4</th>
</tr>
</thead>
<tbody>
<tr>
<td>4’AASA</td>
<td>2.222</td>
<td>1.806</td>
<td>1.660</td>
<td>1.250</td>
<td>0.780</td>
</tr>
<tr>
<td>DAAB</td>
<td>2.222</td>
<td>1.762</td>
<td>1.380</td>
<td>1.008</td>
<td>0.630</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DYESTreatment</th>
<th>Metanil yellow</th>
<th>Bismark brown</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 = 48 Hrs</td>
<td>T3 = 15 Days</td>
<td></td>
</tr>
<tr>
<td>T2 = 96 Hrs</td>
<td>T4 = 30 Days</td>
<td></td>
</tr>
</tbody>
</table>

from Euphorbia royleana latex 12. SDH activity also increased to 107%, 118% in muscles and 105% and 120% in liver of C. punctatus after 96 hrs. treatment to the sub lethal doses of Stem bark extract of Croton tiglium13. SDH activity depleted in muscles, liver and brain while the enzyme elevated in kidney and intestine of C. punctatus when treated with carbamate pesticide sevin14. Sub lethal exposure of cypermethrin after 96 hrs caused significant depletion in the activity of SDH in muscles, liver, gonadal and nervous tissues of fresh water teleost C. fasciatus15.

Significant inhibition in SDH activity was observed in the liver, muscles and gills of L. rohita when exposed to sub lethal concentration of sodium cyanide16. Rapid depletion was seen in SDH activity in liver, muscles, gills and kidney of L. rohita treated with lethal and sub lethal doses of endosulfan and fenvalerate17.

The decrease in SDH activity can be associated with the inhibition of mitochondrial respiratory mechanism or dearrangement in ultra-structure, architectural intigrity and permeability of mitochondria18. This prevents the transfer of electrons to molecular oxygen, resulting in the inhibition of SDH activity and shifting the aerobic metabolism to anaerobiosis.

REFERENCES