Impact of Common Mixed Effluent of Sipcot Industrial Estate on Histopathological and Biochemical Changes in Estuarine Fish *Lates calcarifer*

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**Abstract:** The present study deals with the toxicity of common mixed effluent of SIPCOT Industrial Estate on histopathological changes of gills and biochemical changes in gills, hepatopancreas and muscle of an important estuarine fish *Lates calcarifer*. When fish was exposed to 10, 15 and 20% effluent concentrations, alteration in the structure of gills was observed. In fish exposed to 10% concentration, swelling, hyperplasia, hypertrophy and proliferation of chloride cells of gills were observed, but in 15% effluent, lifting up of the epithelium and lamellar fusions were seen. In 20% effluent treated fishes, disintegration of epithelial cells, desquamated epithelium, haemorrhage and complete damage of epithelial cells of both primary and secondary lamellae were observed. Overall reduction in total protein, total DNA, total RNA, glycogen content, protein bound sugars and total lipid of gills, hepatopancreas, and muscle was observed. The biochemical changes were higher in 20% effluent treated fish than that of 15 and 10% effluent treated fish. The result of the present study recommends proper dilution of the effluent before its discharge.

**Key words:** Biochemical changes, effluent, fish, histopathology

**INTRODUCTION**

Rapid industrialization in India has resulted in the substantial increase in the liquid waste (spent wash or effluent), which is traditionally discharged into open land or into nearby natural water, causing a number of environmental problems including threat to plants and animal lives and also creating problems such as surface water logging, ground water contamination and salinizing good quality land due to presence of high quality salt contents (Ramona et al., 2001). The adverse input of diverse industrial wastes has aggravated the problem of contamination, and sewage and industrial disposal has greatly enhanced the addition of heavy metals into the aquatic ecosystem. These pollutants alter the natural condition of aquatic medium that causes behavioural changes as well as morphological imbalance of aquatic organisms (Yadav et al., 2005).

According to Sivakumari et al. (2005), the estuarine pollution may lead to marine pollution, which may enter to the deep ocean, thus affecting the marine organisms, which are consumed by human beings. Thus careful management of estuary is the management of ocean and its resources. The authors further added that it is evident that a continued and systematic monitoring of the chemical environment in estuaries is necessary to understand the responses of the organisms to various stresses and to assess the degree of pollution and its variation.

Any change in water quality is rapidly reflected in fish gill structure and function, since gills are continuously exposed to ambient water. Gills are the primary sites of gas exchange, acid-base regulation and ion transfer (Randall, 1990). The gill epithelium consists mainly of three types of cells: pavement or respiratory cells, mucus cells, and chloride cells as pointed out by Laurent and Perry (1995). Pawert et al. (1998) stated that gills represent major sites for respiration; they are always in contact with water, which makes them important targets for water pollutants. The authors further stated that fish gills comprise more than half of the body surface, with an epithelial layer of only a few microns separating the interior of the fish from the external environment. As a result of this, a close association between water and blood occurs, so that the gills are strongly affected by environmental contaminants (Rombough and Garsside, 1977).

According to Christensen et al. (1972), any kind of stress not resulting in gross changes and mortality produces certain changes in fish blood characteristics.
Early detection of specific physiological abnormalities provides an indication of exposure to solutions prior to manifestation of any gross damage as opined by Gill and Pant (1981). The above authors used the biochemical changes in tissues and blood of fish to assess the chronic effects of toxicants. The impact of industrial effluents on the physiology and biochemistry of fishes has been well documented by several authors. Before the drastic cellular and systematic dysfunction manifest themselves, appropriate biochemical parameters could be used effectively as sensitive indicators (Aldrige, 1983). Therefore it appears useful to examine changes in the biochemistry of fish intoxicated with effluents to evaluate its toxicity.

In view of the above, it was felt that it would be worthwhile to study the changes in structural and biochemical changes in fish exposed to common mixed effluent from pesticide, paint, fertilizer and pharmaceutical manufacturing companies situated in SIPCOT Industrial Estate, Cuddalore, Tamil Nadu, India, that is discharged into Uppanar (Paravanar) Estuary, which would throw a clear light on the extent of changes and damage that is caused. Hence in the present work, we studied the toxic effects of common mixed effluent on histopathology of gills and biochemical changes in gills, hepatopancreas and muscle of an important estuarine fish *Lates calcarifer*.

**MATERIALS AND METHODS**

Specimens of *Lates calcarifer* were collected from Rajiv Gandhi Centre for Aquaculture (RGCA), Thirumullaivasal, Sirkali, Tamil Nadu, India, and acclimatized to laboratory conditions at Centre of Advanced Study in Marine Biology, Annamalai University, Parangipettai for fifteen days. Water was changed daily and fish were fed *ad libitum* with flour pellets and ground dried shrimp twice a day. For experimental studies, fish ranging from 7-10 cm in length and weighing 10-12 g were selected. The common mixed effluent from SIPCOT Industrial state, Cuddalore, was collected in a polythene container and stored in a refrigerator until use. Temperature and pH were noted at the point of collection itself. The physico-chemical parameters of the common mixed effluent was estimated according to APHA *et al.* (1976) and are as follows: Colour- Brown; Odour- Pungent; Dissolved Oxygen-3.2±0.02 mg l⁻¹; BOD- 13.7±2.0 mg l⁻¹; pH- 5.7±0.2; Temperature- 30.0±2.0°C; Salinity- 1.3±0.30 ppm; Total Hardness-4.0±2.0 mg l⁻¹; and Total Alkalinity-40±2.0 mg l⁻¹.

Five groups containing ten healthy fish in each were selected and introduced into the plastic tubs containing 100, 75, 50, 25, 10 and 5% effluent. The manifestation time and survival time of fish were observed following the method of Wuhrmann (1950). Based on the above studies, 10, 15 and 20% effluent concentration were selected for experimental purpose. Fish were exposed to the above said concentrations along with common control; each group had ten fish in each and the experimental setup had five replicates. At the end of one hour of effluent exposure, fish were rinsed with filtered seawater and sacrificed without anaesthesia. The organs/tissue such as gills, hepatopancreas and muscle were subsequently dissected using a sterile scalpel, and were rinsed with distilled water in order to remove the adhering body fluid. For histopathological studies, gills were fixed in Bouin’s fixative and later processed following the methods of Pearse (1968), Roberts (1978) and Humason (1979). The protein content of the samples was determined according to the method of Lowry *et al.* (1951) using crystalline Bovine Serum Albumin (BSA) as standard. Nucleic acid was extracted according to the method of Schneider (1957). Deoxy Ribonucleic Acid (DNA) was estimated by the following the protocol of Burton (1956) using highly polymerized Calf-Thymus DNA as standard. Ribonucleic Acid (RNA) was estimated according to the method of Rawal *et al.* (1977) by using yeast RNA as standard. The carbohydrates viz., protein bound sugars and glycogen was estimated by the method of Caroll *et al.* (1956) using glucose standard. Total lipids were extracted and estimated based on the procedures of Folch *et al.* (1995). The statistical significance of the samples was tested by Two Way Analysis of Variance (ANOVA) using Randomized Block Design (RBD).

**RESULTS AND DISCUSSION**

In the present study, fish survived for 1, 2 and 3 h. in 10, 15 and 20% effluent concentration, respectively, whereas in effluent concentrations of 30, 50, 75 and 100%, the fish survived for 55, 50, 40 and 30 min, respectively. Based on this observation, 10, 15 and 20% effluent concentrations were selected for experimental purpose.

Histopathological studies revealed that in control fish, primary lamellae appeared normal and mucus free with well-defined secondary lamellae branched from them (Fig. 1a). In 10% effluent concentration, swelling, hyperplasia, hypertrophy and proliferation of chloride cells were observed (Fig. 1b). In 15% effluent, lifting up of the epithelium, swelling, hyperplasia, hypertrophy and proliferation of chloride cells, degenerative changes of epithelial cells and fused lamellar filaments were noticed (Fig. 1c). In 20% effluent, the gills showed necrosis, disintegration of epithelial cells, desquamated epithelium, haemorrhage and complete damage of epithelial cells of both primary and secondary lamellae (Fig. 1d).

The results of the present investigation revealed that DNA and RNA content reduced significantly in the gills,
hepatopancreas, and muscle of *L. calcarifer* exposed to effluent concentrations. The observed decrease in the levels of DNA and RNA content can be best correlated with protein reduction in the muscles exposed to different effluent concentration. Since DNA and RNA synthesis precedes protein synthesis, the reduction in their levels is very well reflected in the protein levels. The binding of metals to phenylalanine and lysine tRNA should have inhibited their propagation to ribosomes, which caused reduction in the protein content as suggested by Tulasi *et al.* (1992). In this study also decreased level of DNA and RNA were observed this may be due to the above-mentioned reason.

In the present study, glycogen content decreased in gills, hepatopancreas and muscle when exposed to various effluent concentrations. Stored glycogen content in tissues is also released by anaerobic glycolysis and utilized to meet the energy requirement under pollutant stress as stated by Heath (1987). Hinson *et al.* (1973) remarked that maximum glycogen depletion corresponds to a dramatic increase in blood glucose level in the fish *Channa punctatus* exposed to industrial pollutants. They suggested that it might be due to some of the hepatic glycogen getting converted to glucose via the intermediate glucose-1-phosphate getting and entering the circulation. The decreased glycogen content recorded in the present study may be due to the induced activation of adrenal pituitary glucocorticoid hormones which stimulate the hepatic glucose production thereby elevating the blood glucose level or it may be a physical response to meet the critical need of energy under effluent stress as suggested by the others. (Table 1).

Likewise, protein bound sugar also recorded a decline in its level in all the three tissues. Carbohydrates are considered to be the first degraded under the stress condition of animals. According to Dhavale and Masurekar, (1986), decreased level of carbohydrate constituents in tissues of toxicant exposed animals may be due to the prevalence of hypoxic condition in the tissues as a result of pollutant stress. During the hypoxic
conditions, there is increased carbohydrate metabolism to release energy resulting in the extra expenditure of carbohydrate constituents. The observed result in the present study is in accordance with the findings of Valarmathi and Azariah (2002), who have suggested that the decreased level of tissue carbohydrates in the toxicant exposed animals seemed to induce the glycogenolysis, possibly by increasing the activity of glycogen phosphorylase to meet the energy demand under stress condition or the toxicant may have an effect of glycolysis by inhibiting the activity of carbohydrate metabolism. (Table 1).

Carbohydrates in the tissues of animals exist as protein bound sugars and glycogen. Protein bound sugar is the best energy producers of the body of the living organism. Polysaccharides occur both in free-state as well as bound state along with proteins. Even though, protein is the major source of energy in animals, chemical stress causes rapid depletion of stored carbohydrates primarily in hepatopancreas and in other tissues (Vijayavel and Balasubramanian, 2006).

Lipid content is an essential organic constituent of the tissues of all animals, and plays a key role in energy metabolism. Lipids are the best energy producers of the body next to carbohydrates. The lipid content of gills, hepatopancreas, and muscle showed decreased levels in the fish exposed for 10, 15 and 20 effluent exposure. The decreased level of tissue lipid content in the present study may also be due to liver dysfunction or mobilization of glycerol or inhibition of oxidative phosphorylation. The

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Table 1: Biochemical changes in organs/tissue of estuarine fish *Lates calcarifer* when exposed to different concentrations of common mixed effluent of SIPCOT Industrial Estate.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Toxicants</th>
<th>Gills</th>
<th>Hepatopancreas</th>
<th>Muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (g M⁻¹ wet tissue)</td>
<td>Control</td>
<td>297.30±0.709&lt;sup&gt;a&lt;/sup&gt;</td>
<td>457.62±0.761&lt;sup&gt;a&lt;/sup&gt;</td>
<td>381.40±0.675&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>10 %</td>
<td>167.68±0.212&lt;sup&gt;b&lt;/sup&gt;</td>
<td>217.54±0.673&lt;sup&gt;b&lt;/sup&gt;</td>
<td>266.50±0.636&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-24.88)</td>
</tr>
<tr>
<td>15 %</td>
<td>141.44±0.743&lt;sup&gt;b&lt;/sup&gt;</td>
<td>182.70±0.644&lt;sup&gt;b&lt;/sup&gt;</td>
<td>227.60±0.129&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-40.33)</td>
</tr>
<tr>
<td>20 %</td>
<td>117.44±0.615&lt;sup&gt;b&lt;/sup&gt;</td>
<td>121.50±0.644&lt;sup&gt;a&lt;/sup&gt;</td>
<td>188.50±0.667&lt;sup&gt;a&lt;/sup&gt;</td>
<td>(-50.58)</td>
</tr>
<tr>
<td>DNA (g M⁻¹ wet tissue)</td>
<td>Control</td>
<td>83.34±0.615&lt;sup&gt;a&lt;/sup&gt;</td>
<td>103.30±0.731&lt;sup&gt;a&lt;/sup&gt;</td>
<td>95.72±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>10 %</td>
<td>73.30±0.644&lt;sup&gt;b&lt;/sup&gt;</td>
<td>83.30±0.660&lt;sup&gt;b&lt;/sup&gt;</td>
<td>83.30±0.660&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-12.98)</td>
</tr>
<tr>
<td>15 %</td>
<td>65.30±0.745&lt;sup&gt;b&lt;/sup&gt;</td>
<td>75.40±0.718&lt;sup&gt;b&lt;/sup&gt;</td>
<td>77.30±0.682&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-19.20)</td>
</tr>
<tr>
<td>20 %</td>
<td>40.40±0.636&lt;sup&gt;b&lt;/sup&gt;</td>
<td>54.30±0.644&lt;sup&gt;b&lt;/sup&gt;</td>
<td>51.40±0.644&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-46.30)</td>
</tr>
<tr>
<td>RNA (g M⁻¹ wet tissue)</td>
<td>Control</td>
<td>94.20±0.687&lt;sup&gt;a&lt;/sup&gt;</td>
<td>124.40±0.771&lt;sup&gt;a&lt;/sup&gt;</td>
<td>109.30±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>10 %</td>
<td>85.40±0.709&lt;sup&gt;b&lt;/sup&gt;</td>
<td>113.20±0.729&lt;sup&gt;b&lt;/sup&gt;</td>
<td>94.12±0.712&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-13.87)</td>
</tr>
<tr>
<td>15 %</td>
<td>73.40±0.765&lt;sup&gt;b&lt;/sup&gt;</td>
<td>92.10±0.702&lt;sup&gt;b&lt;/sup&gt;</td>
<td>81.20±0.708&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-25.62)</td>
</tr>
<tr>
<td>20 %</td>
<td>65.40±0.718&lt;sup&gt;b&lt;/sup&gt;</td>
<td>82.30±0.745&lt;sup&gt;b&lt;/sup&gt;</td>
<td>73.30±0.793&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-32.87)</td>
</tr>
<tr>
<td>Glycogen (g M⁻¹ wet tissue)</td>
<td>Control</td>
<td>31.40±0.787&lt;sup&gt;a&lt;/sup&gt;</td>
<td>58.50±0.752&lt;sup&gt;a&lt;/sup&gt;</td>
<td>58.50±0.809&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>10 %</td>
<td>28.50±0.742&lt;sup&gt;a&lt;/sup&gt;</td>
<td>28.30±0.787&lt;sup&gt;a&lt;/sup&gt;</td>
<td>53.10±0.687&lt;sup&gt;a&lt;/sup&gt;</td>
<td>(-12.47)</td>
</tr>
<tr>
<td>15 %</td>
<td>25.40±0.642&lt;sup&gt;b&lt;/sup&gt;</td>
<td>42.20±0.665&lt;sup&gt;b&lt;/sup&gt;</td>
<td>39.20±0.682&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-32.92)</td>
</tr>
<tr>
<td>20 %</td>
<td>23.40±0.667&lt;sup&gt;b&lt;/sup&gt;</td>
<td>42.20±0.665&lt;sup&gt;b&lt;/sup&gt;</td>
<td>39.20±0.682&lt;sup&gt;b&lt;/sup&gt;</td>
<td>(-32.92)</td>
</tr>
<tr>
<td>Protein bound sugar</td>
<td>Control</td>
<td>12.50±0.705&lt;sup&gt;a&lt;/sup&gt;</td>
<td>36.30±0.751&lt;sup&gt;a&lt;/sup&gt;</td>
<td>27.20±0.694&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>(µg M⁻¹ wet tissue)</td>
<td>10 %</td>
<td>13.30±0.702&lt;sup&gt;a&lt;/sup&gt;</td>
<td>23.30±0.723&lt;sup&gt;a&lt;/sup&gt;</td>
<td>28.60±0.814&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>15 %</td>
<td>9.30±0.702&lt;sup&gt;a&lt;/sup&gt;</td>
<td>19.60±0.793&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.50±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>20 %</td>
<td>7.70±0.793&lt;sup&gt;a&lt;/sup&gt;</td>
<td>15.40±0.702&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.30±0.729&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total Lipid (µg M⁻¹ wet tissue)</td>
<td>Control</td>
<td>156.30±0.511&lt;sup&gt;a&lt;/sup&gt;</td>
<td>253.30±0.687&lt;sup&gt;a&lt;/sup&gt;</td>
<td>337.20±0.702&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>10 %</td>
<td>147.30±0.702&lt;sup&gt;a&lt;/sup&gt;</td>
<td>253.30±0.687&lt;sup&gt;a&lt;/sup&gt;</td>
<td>312.30±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>15 %</td>
<td>131.40±0.681&lt;sup&gt;a&lt;/sup&gt;</td>
<td>190.40±0.771&lt;sup&gt;a&lt;/sup&gt;</td>
<td>297.30±0.687&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>20 %</td>
<td>127.30±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
<td>173.20±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
<td>285.30±0.708&lt;sup&gt;a&lt;/sup&gt;</td>
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</table>

Values are expressed as mean±S.E. of five individual observations, Values in parentheses are per cent change over control, - : indicates per cent decrease over control. Values are significant at p<0.05 level, *: values are significantly different among organs/tissue, **: values are significantly different among effluent concentrations.
above results revealed that the fluctuations of biochemical constituents were higher in the tissues of fish exposed to effluent concentrations (Table 1).

Gilbert and O’Connor (1970) reported that lipids are vital to embryogenesis, providing two thirds of energy by oxidation. Agarwal (1992) observed that an elevated level of serum cholesterol in *Channa punctatus* exposed to mercuric chloride effluent might be an indication of liver damage, which normally esterifies cholesterol, and excrete a part of it with the bile. Janderko and Kossmann (1965) demonstrated that blood cholesterol elevation might be due to the inhibition of the activity of enzymes of lipid metabolism such as lipoprotein lipase by toxic effluents leading to a retarded clearance of lipids from blood. In the present study also, the decreased total lipid content might be due to liver damage and/or inhibition of activity of enzymes of lipid metabolism.

Examination of tissues after death from fish and other aquatic organisms may serve to identify the cause of death and possibly the causative agent as opined by Meyers and Hendricks (1985). Gill lesions can be divided into two groups i.e., the direct deleterious effects of the irritants (Temmink et al., 1983) and the defence responses of the fish (Morgan and Towell, 1973). Separation of epithelial cells and necrosis were reported in brook trout, *Salvelinus fontinalis* exposed to acute toxic levels of toxicant (Daye and Garside, 1976). Jagoe and Haines (1983) noted shortened and thickened secondary lamellae, primary lamellar swelling, droplets of mucus and fused adjacent secondary lamellae in Sunapee trout, *Salvelinus alpinus oquassa* subjected to pH 4.0 Hyperplasia, shortened and thickened respiratory lamellae, fused adjacent primary lamellae were observed by Leino et al. (1987) in pell dace, *Semotilus margarita* and fathead minnows, *Pimephales promelas* exposed to low acid pH of 5.16. Scherl and Hoffmann (1988) found fused secondary lamellae and hyperplasia in fish, brown trout, *Salmo trutta* exposed to pH 3.0 and 4.9.

In the present study, rupture and necrosis of gill epithelium of the effluent may be due to direct deleterious effect of the effluent. However, hyperplasia, hypertrophy, lamellar fusion and mucus secretion and sloughing of gills may be defence responses of the fish to the effluent toxicity. The above findings may recall the work of Mallatt (1985), who reported that rupture and necrosis of the gill epithelial cells occur in acute toxic conditions caused by low pH. The intimate contact of gill with polluted water may lead to alterations in the normal gill epithelium (Jayanatha et al., 1984), Lloyd (1960), Brown (1968) and Skidmore and Towell (1972) reported that many noxious compounds and ions have been shown to damage the respiratory epithelium of gills.

Lamellar fusion could be protective as it diminishes the amount of vulnerable gill surface area in fish (Mallatt, 1985). Fusion of secondary lamellae and swelling of primary and secondary lamellae increases the diffusion distance (Tietge et al., 1988) and reduced surface area (Smith and Haines, 1995). Fusion of primary lamellae at the distal end and thickened and shortened secondary lamellae observed in the present study may be involved in reducing the impact of effluent toxicity in the present case supporting the observation of the above authors.

Munshi and Singh (1971) and Jagoe and Haines (1983) suggested that the mechanism by which cells recognize and aggregate with one another at the cell surface is still unknown. The authors further stated that the term ‘mutual recognition’ in a cell population, ‘surface coding’ and ‘preferential affinities’ have been used to explain such mechanisms; it could be explained that alterations in the charge of glycoproteins coating the lamellae due to the presence of sialic acid residues of mucin at low pH, favour attraction between the cells of adjacent lamellae causing fusion. A similar reason may be attributed for the fusion of primary and secondary lamellae in the present study too.

Jagoe and Haines (1983) found swelling of primary and secondary lamellae, fusion of adjacent secondary lamellae, increased mucus production, progressive loss of micro-ridge pattern, secondary lamellae appeared thickened and shortened with extremely rough surface and considerable mucus in brook trout, *Salvelinus fontinalis* exposed to pH 4.5 and 5.0 for 456 hr. A similar observation was made by Tandjung (1982) and Segner et al. (1988) in brown trout, *Salmo trutta*. Daye and Garside (1976) observed hypertrophy and separation of epithelial cells from the supporting pillar cells in brook trout, *Salvelinus fontinalis* in chronic acid pH (pH 5.5) treatment and this condition greatly increased the diffusion distance (water-blood distance). Skidmore and Towell (1972) observed that toxicants appear to cause loss of adhesion between the epithelial cell and the underlying pillar cell system accompanied by a collapse of the structural integrity of the secondary lamella. Wood et al. (1988) reported that thickening of the lamellar epithelium increased diffusive distance of the gill. The thickening of the gill epithelium (*via* cell hypertrophy) is sometimes considered to be an indicator of cell degeneration and eventually necrosis (Tietge et al., 1988; Peuranen et al., 1993). The lifting and hypertrophy of cells greatly increases the diffusion distance (water-blood distance) (Ingersoll et al., 1990). Schmidt et al. (1999) observed epithelial cell lifting, eosinophilic granular cytoplasm, epithelial hypertrophy, mild curvatures of the primary and secondary lamellae, and occasionally epithelial hyperplasia and leukocyte infiltration of gill epithelium changes and also showed elevated gill indices in brown trout, *Salmo trutta* in diluted sewage plant effluents (62 to 205%) exposure.

Brown et al. (1990) and Peuranen et al. (1993) observed that hypertrophied gill tissue exhibited an
Several authors have reported alterations in protein content of gills, hepatopancreas, and muscle when fish are exposed to pollutant stress. The reduction in protein content might be due to the blocking of protein synthesis or protein denaturation or interruption in the TCA cycle for energy production during stress condition.

Considering the above facts, the present study concludes that the effect of common mixed effluent of SIPCOT Industrial Estate has heavy impact on the histopathological and biochemical parameters of the fish. Hence the present study recommends proper dilution of the effluent before its discharge.

**CONCLUSION**

Since the *Lates calcarifer* is considered to be one of the chief edible fishes in this region, a continuous assessment with respect to the discharge of effluents into the water bodies are need of the hour. If not treated properly, it will therefore affect the food chain including human. It is of interest to note that a change in severe gill alteration was observed in all treatments but the alteration was less in 10% concentration compared to 25%. That the fish gills show various changes in the gills this may be due to stress cum toxic responses of the exposed fish. In the present study recommends to avoid such impact in the aquatic environment, a proper dilution management is needed before they are discharged.

**Significant of the study:** Since the *Lates calcarifer* is considered to be one of the chief edible fishes in all over the world, a continuous assessment with respect to the discharge of effluents into the water bodies are need of the hour. If not treated properly, it will therefore affect the food chain including human. It is of interest to note that the reduction in protein content under effluent stress noticed in the present study may be attributed to the utilization of amino acids in various catabolic reactions. Therefore, the present study was aimed to evaluate the effect of mixed effluent of SIPCOT Industrial Estate on growth and biochemical parameters of *Lates calcarifer*.

The reduction in protein content under effluent stress noticed in the present study may be attributed to the utilization of amino acids in various catabolic reactions. By the process of transamination and deamination, ketoacids are synthesized, which serve as precursors for the maintenance of carbohydrate metabolism to meet the energy requirements during pollutant stress. The reduction in protein content might be due to the blocking of protein synthesis or protein denaturation or interruption in the amino acid synthesis by metals (Jha, 1991). The food utilization decreases when the animals are under stress condition, which leads to the depletion of protein content in tissues. The reduction in protein content indicates that the tissue protein undergoes proteolysis, which results in the production of free amino acids and used in the TCA cycle for energy production during stress condition.
Tulasi et al. (1992). In this study also decreased level of DNA and RNA were observed this might be due to the above-mentioned reason. The decreased glycoprotein content recorded in the present study may be due to the induced activation of adrenal pituitary glucocorticoid hormones which stimulate the hepatic glucose production thereby elevating the blood glucose level or it may be a physical response to meet the critical need of energy under effluent stress as suggested by the others. During the hypoxic conditions, there is increased carbohydrate metabolism to release energy resulting in the extra expenditure of carbohydrate constituents. In the present study also, the decreased total lipid content might be due to liver damage and/or inhibition of activity of enzymes of lipid metabolism. In the present study, rupture and necrosis of gill epithelium of fish may be due to direct deleterious effect of the effluent. A change in severe gill alteration was observed in all treatments but the alteration was less in 10% concentration compared to 20%. That the fish gills show various changes in the gills this may be due to stress cum toxic responses of the exposed fish. In the present study recommends to avoid such impact in the aquatic environment, a proper dilution management is needed before they are discharged.

REFERENCES


