Summary
And
Conclusions
1. Human population explosion, unplanned urbanization, deforestation, profit-oriented capitalism and technological advancement have inadequately introduced pollution into the aquatic environment. Out of all the pollutants causing pollution of aquatic environment, heavy metals are predominant. Some of these metals are essential in limited quantities to normal growth of organisms. Even essential metals are toxic if the concentration exceeds the normal requirement. Chromium belongs to the first series in the transitional elements (VI-B), plays dual role, as an essential metal and as a potential toxic element. It is spewed into the atmosphere from the leather tanning industry and also with the burning of fossil fuel. Lead belongs to the subgroup IV-A and man is using it in different form from ancient times. Lead is entering the aquatic environment by dumping of effluents of various lead consuming industries, surface run off and aerosol precipitation. Fishes represent one of the largest populations of aquatic life and serve as proteins food supplement for many people worldwide. Protection of these animals from the deleterious effects of metals is possible only by analyzing the response patterns of them to various concentrations at different periods of exposure. The present study has been taken up with this view, in which some of the biochemical and histological responses in the selected organs like the gill, liver and muscle of an edible freshwater fish, *Cyprinus carpio*, exposed to sublethal concentrations of chromium and lead were analyzed.

2. For the evaluation of sublethal concentrations of chromium and lead the LC$_{50}$s were determined to the fish by dose response curves and are
subsequently verified by Dragstedt and Behren’s method. As the period of exposure is considerably important in evaluating the toxicity of a metal, 96 h exposure was preferred in determining the LC$_{50}$s with a view that the effects of either chromium or lead become consistent with in this period. The 96 h LC$_{50}$s obtained were 24.90 mg/l and 17.18 mg/l respectively for chromium and lead. The values indicated that the fish were more sensitive to lead than chromium. Various symptoms of poisoning like irritability, hyperexcitability, mucus secretion and restless movements were observed, especially in the fish exposed to acute concentrations of lead. Slow sluggish and short jerky movements, surfacing and gulping of air with loss of equilibrium were noticed in the fish exposed to chromium. No significant ethological changes were observed in lower concentrations of chromium, but in lead some hyperexcitable movements were noticed. Thus an approximate concentration of the metal in the ambient medium can be analyzed based on the behavioral patterns of fish.

3. Some aspects of carbohydrate metabolism were studied in different organs of the fish exposed to the sublethal concentrations of chromium and lead, 2.5mg/l, 1.7mg/l respectively, at different periods of exposure, 1, 7, 15 and 30 days. The blood glucose level initially decreased at day 1 of exposure of the fish to the sublethal concentration of either chromium or lead. But on further exposure of fish to chromium the blood sugar level increased and ultimately reached to normalcy at day 30. Where as in lead exposure fish the blood sugar level progressively increased from day 1 to day 30. The
glycogen levels though insignificantly decreased at days 15 and 30 in the liver and days 1 and 15 in muscle, with the corresponding increase in the activity of glycogen phosphorylase, they restored normalcy at day 30 in the fish exposed to chromium; whereas progressive glycogenolysis was observed in the liver and muscle of the fish at all the days of exposure to the lead. Thus significant hyper-glycema was seen in the lead exposed fish but not in chromium exposed ones. The break down of glycogen in the fish exposed to lead could be due to the neuro endocrinal imbalance induced by the lead ions.

4. Supporting the changes observed in glucose and glycogen levels, the SDH activity insignificantly increased from days 7 to 30, except the decrease at day 1 in the gills, kidney, liver and muscle of the fish exposed to chromium. Added to it the LDH activity also increased over time to exposure in the organs of chromium intoxicated fish. Further, the pyruvate levels increased initially at day 1 and 7 which was followed by its decrease at days 15 and 30. But the lactate levels increased progressively over time of exposure. The over all results indicated the elevation of both anaerobic glycolysis and oxidative metabolism in the organs of the fish exposed to chromium stress to meet the energy demands required for the activation of compensatory mechanism in order to adapt to the tolerable concentration of chromium. Such a situation was not observed in lead exposed fish due to stepwise decrease in SDH activity in the organs from day 1 to day 30 followed by the accumulation of high concentrations of pyruvate and lactate. It appears that
the fish might be derived the energy mostly by the elevation of anaerobic glycolysis, as evidenced by the increase in LDH activity. The decrease in SDH activity would be due to interference of the lead ions with the active sites of this enzyme. Thus it is seen that though lead stimulated glycogenolysis leading to the production of glucose, this has not been fully utilized by the animal for the energetic purposes due to the suppressed oxidative pathway. The non-availability of sufficient energy to the active organs of the fish could result in the failure of metabolic compensation to the imposed toxic stress.

5. Metabolic disorders, in the fish exposed to the sublethal concentrations of chromium and lead were confirmed by the histological changes observed in gills, liver and muscle of the fish. In the sublethal concentrations of chromium, though a few pathological changes were seen, which are mild in degree in organs of fish during the initial days of exposure i.e., at day 7, a significant structural reorganization was seen on further exposure of the fish. The increase in oxidative and glycolytic cycles might have facilitated the protein synthetic abilities for the structural reorganization in the sublethal concentrations of chromium. Whereas in lead exposed ones the gills exhibited confirmational degenerative changes such as swellings at the base of secondary gill lamellae, hypertrophy, hyperplasia and necrosis. Similarly the liver and muscle of fish also exhibited the degenerative changes like the disarray of liver cords, dilation of sinusoids with coagulated blood, heavy vacuolation in liver tissue, thinning down and
breakdown of muscle fibrils etc. All these changes in the organs of the fish confirmed structural disruption by lead ions on prolonged exposure.

6. On the whole, based on the biochemical and histological responses of the fish, *Cyprinus carpio* to the sublethal concentration of chromium and lead it can be inferred that the adaptive ability of the fish to heavy metals is not only dependent on the concentration of the metal to which it is exposed but also the nature of the metal and the length of exposure. Eventhough the sublethal concentration of chromium initially suppressed some of the biochemical activities in the organs of the fish and disturbed their structural integrity, on prolonged exposure these animals could resist the chromium stress by bringing the necessary biochemical changes leading to the repair of the loss structural integrity. So, *Cyprinus carpio* can tolerate the lower concentration of the chromium to a considerable degree and even it can adapt to such concentrations with out suffering lasting effects. It could be due to the rapid proteins synthetic ability at the organ level and metabolic reorganization at cellular level. But the same fish could not resist the sublethal concentration of lead for a longer period due to the suppression of its biochemical activities by lead ions. The loss of biochemical integrity of the cell due to the interaction of lead ions might be the reason for its incapability to adapt to the lead toxicity.