INTRODUCTION
In recent years, pollution of aquatic environment by heavy metals has become a national and international problem. This is because of the unprogrammed usage and monitoring processes of wastes in the evergrowing chemical industries resulting in high discharge of metals into the aquatic eco-system. Nature could not bear this most unfortunate formulation made by man and biomass of the continent could not accept such high standard of environmental erosion.

Certain metals play a very important role in life systems. More than 99% of the structure of living things is composed of 12 bulk elements and these in trace amounts are mostly concerned with metabolism. However, most of the remaining metals in the periodic table are highly toxic. A metal can be regarded as essential if the organism can neither grow nor complete its life cycle in the absence of it or it cannot be replaced by any other metal or if it exerts a direct influence of the organism and its metabolism. Similarly a metal can be regarded as toxic if it injures growth or metabolism of an organism when accumulated above to a required or tolerable concentration.

The term heavy metal is a loose one which includes transition metals like arsenic, antimony, bismuth, cadmium, chromium, cobalt, copper, lead, mercury, nickel, zinc etc., having the atomic number 22 to 92 in all the groups from periods III to VII in the periodic table. But it is very difficult to establish a satisfactory grouping to identify these metals. Passow et al., (1961) defined heavy metals as
those having density greater than five and included above 38 metals under this grouping. The common feature of these metals is that they are relatively toxic even at fairly low concentrations and are readily accumulated by the aquatic organisms. The seriousness of their presence in water is compounded by the facts that generally they are water soluble, non-degradable, vigorous oxidizing agents and strongly bonded to many biochemicals, especially polypeptides and proteins. All heavy metals are toxic to aquatic organisms at high concentrations, but some are highly toxic even at lower concentrations. Metals like mercury, copper, cadmium, lead and zinc are very toxic, of which, except copper and zinc the others are non-essential metals (Nammalwar, 1983).

Heavy metals occur naturally at a fairly low concentrations in aquatic environment since the beginning of geologic time, added principally through weathering of rocks and volcanic activity (Klein and Goldberg, 1970). In addition, high concentrations of these metals entering the aquatic system by direct discharges of industrial waste products, agricultural effluents, sewage waters and surface run off and indirectly from aerial fall out. As certain metals are required in life processes, most of the aquatic organisms have the capacity to accumulate them. Some of the non-essential metals also accumulate in this way inside the body of organisms, form complexes with organic substances and cause greatest harm to them.
Recent attention has been called to the problem concerning the pollution of aquatic environment by mercury which is divalent group IIIB metal. It is because of the fact that this metal has proved to be highly toxic to aquatic fauna. Mercury is the only metallic element existing in the liquid state at ordinary temperatures and pressures. It has an atomic number 80 and atomic weight 200.59 It occurs naturally in the form of seven stable isotopes. Both as the metallic (quick silver) and the sulfide (cinnabar) forms mercury is known to different people around the world for thousands of years and has played a prominent role in therapeutics, alchemy and folklore (Berman, 1980). Woodall (1939) described mercury as the "hottest, the coldest, a true healer, a wicked murderer, a precious medicine, and a deadly poison - a friend that can flatter and lie". Alkyl mercurials are of great importance as contaminants in the environment. Methyl mercury compounds are the most toxic of alkyl mercurials. In fact, the elemental mercury and mercury compounds can be converted to methyl mercury by micro-organisms (Jensen and Jernelow, 1969; Jernelow, 1969). Most recent concern with alkyl mercurials stems from repeated outbreaks of methyl mercury poisoning in Japan and elsewhere of which, the most famous incident was that of "Minamata disease" that occurred in Minamata, Japan from 1953 to 1960, where cats and human beings showed spastic movements, partial paralysis, coma and death due to the ingestion of fish and shellfish contaminated by industrial effluents containing methyl mercury (Takeuchi et al., 1977). The mass intoxication epidemics that resulted in Iraq were
due to the consumption of bread made from treated grains with methyl mercury (Baker et al., 1973; Al-Tikriti and Al-Multi, 1976). Epidemics of this type were also recorded in different parts of the globe including India (Takizawa, 1979). Methyl mercury is a highly persistent kind of pollutant that accumulates in food chain and its half life in human beings has been estimated about 70 days.

There are numerous sources of mercurial contamination of the aquatic environment. They include pulp and paper industry, organic chemicals, petrochemicals, inorganic chemicals, pesticides, fertilizers, basic steel works, motor vehicles, explosives, plastic and other allied industries. In addition, mercury contamination is also confronted in mercury mining, felt making, mirror making and chemical synthesis of mercury fulminate primers (Patering and Tepper, 1976). After discharge into the aquatic environment by various sources, the inorganic forms of mercury get converted into organic compounds. The transformation of inorganic and organic mercury in biological or biologically controlled systems play an important role in the ecological distribution of mercury. The studies of Jensen and Jernelow (1969) and Jernelow (1969) have thrown light on the formation of methyl mercurials from inorganic forms of mercury on microbial or enzymatic actions. On the other hand, the biotransformation of organo-mercurials to inorganic mercury has been demonstrated in animal tissues by Miller et al., (1960, 1961) and Gage (1964). The importance of these biotransformations is that we can no longer compartmentalise the toxicity of inorganic mercury or
organic mercury, because mercury in any form taken by animals can be converted into either methyl mercury or inorganic mercury. In general, within the organism, mercuric ions act as potent enzyme inhibitors, protein precipitants and corrosives. Mercury has a great affinity for sulphydryl groups and also combines with phosphoryl, carboxyl, amide and amine groups (Berman, 1980).

As far as protection of aquatic organisms is concerned there is a great need to learn about the effects of mercury on these organisms, especially on fish. Fish as an important source of food represent a transfer route of metals to higher trophic levels (Johnels et al., 1967; Lofroth, 1969; Berlin, 1969; Miettinen, 1970). Survey of literature shows a number of reports on the toxicity with reference to acute and chronic exposure to different mercurials. Rodgers et al., (1951) found that 6 mg of Hg/litre as pyridyl mercuric acetate was toxic to rainbow trout, brook trout and brown trout. Clemena and Sheed (1959) investigated the toxicity of pyridyl mercuric acetate, phenyl mercuric acetate, EMP and ethylmercuric-p-toluene sulphonamide to channel catfish and found the 24-96 hours median tolerance limit (TLM) in the range of 0.5 to 4 mg/l. Lindahl and Hell (1970) reported inhibition of respiration of isolated gill filaments, inhibition of oxygen uptake and glycolysis in liver, pronounced decrease of blood oxygen (82%), injuries to gill filaments, decreased circulation in secondary gill lamellae following exposure of roach, Leuciscers rutilus, to phenyl mercuric hydroxide. Ultra-structural studies by Olson et al., (1973) showed a decrease in height of epithelial ridges, vacuolated epithelial cells and degenerated chloride cells in the gills of rainbow trout exposed to methyl mercuric chloride at
0.275 \mu g/l for 8 weeks or to mercuric chloride at 50 \mu g Hg/l for 40 days or to 250 \mu g/l for one week. Further, mercurials are known to interfere in biochemical reactions of the organisms. According to Webb (1966) mercurials are mostly bound to plasma proteins and erythrocytes, form complexes with thiols like cysteine, attack - SH groups and alter enzyme activities where some enzymes are stimulated by low concentrations and inhibited by high concentrations of the metal. Mercurials uncouple oxidative phosphorylation (Wadkins and Lehninger, 1958; Chiga and Plant, 1959), inhibit electron transport chain (Kahn and Jagendorf 1961; Griffiths and Chaplain, 1962) and Porphyrin bio-synthesis (Lescelles, 1956).

Mercurials also affect the reproductive processes of the animals. Kihlstrom et al., (1971) found decreased egg production and frequency of hatching in zebra fish, Brachydanioreropo, in water containing 0.8 \mu g Hg/l as phenyl mercuric acetate. The fertilizing capacity of spermatozoa of steelhead trout, Salmo gairdneri, was found to decrease following exposure to 1 ppm of methyl mercuric chloride for 30 minutes (Mc Intyre, 1973). Mercury as low as 10 ppm has shown to produce teratogenic effect in pre-eyed Sockeyl Salmon eggs (Servizi and Gordon, 1974). Damage to developing eggs, caudal fin abnormalities, hemorrhage and loss of circulating blood were reported in rice fish, Oryzias latipes, when exposed to low concentrations of mercuric chloride (Heisinger and Green, 1975). Exposure to mercuric chloride in early or late blastula stage caused marked defects in development of eggs of Heteroiclitus fundulus (Weiss and Weiss, 1976).
The forgoing account clearly reflects the magnitude and multiplicity of deleterious effects of mercury on aquatic animals, especially on fish. However, information on the comparative account of the effects of lethal and sublethal concentrations of mercury on energy yielding metabolic pathways, particularly the metabolic pathway concerned to lipids, of the freshwater fishes, which serve as staple food for human beings, is very limited. There is still a great need of information of this nature to arrive at definite conclusions. For proper management of our renewable resources in the face of growing technology and industrial production, research on this line is immediately warranted. It is to contribute to such type of knowledge, in the present investigation an attempt is made to study and compare the lethal and sublethal effects of mercury on a few aspects of fat metabolism in the freshwater fish *Cyprinus carpio*.

Lipids are heterogenous group of complex macromolecules, having high caloric value, present in the bio-systems. Essentially, they are esters of fatty acids or substances capable of forming esters which consist of fats, oils, phospholipids, triglycerides, glycerol, cholesterol, neutral lipids etc. They constitute not only the architecture of the cell but also form a co-basis for the structure of some enzymes like Mg$^{2+}$ATP ase. Lipids offer full complementary structure to steroid hormones, and also contribute for energy synthesis as an alternative to carbohydrates (Goldfine, 1968; Harper, 1977; Lehninger, 1978; Guyton, 1981). Especially in recent years evidence has been
coming forth to indicate that lipids form the chief fuel during prolonged and sustained activity of animal. Thus birds, insects and bats have been shown to utilize fats as chief fuel during sustained muscular activity (Weisfogh, 1952; George and Jyoti 1955, 1958). The same situation is reported to occur in fishes too, such as the Salmon (Hoar, 1976).

Meischer-Rush (1883) was the first to study the changes in the quality of lipids in the body of atlantic salmon, *Salmon salar*, during its migration. Panton (1898) suggested that the lipids which accumulate within and outside the muscle fibres in the atlantic salmon during active feeding are later used up either for the development of gonads or as energy source during spawning migration. Greene (1913) suggested that the very large quantity of fat stored in the muscle cells of king salmon, *Oncorhynchus tshawytscha*, prior to the upstream migration is to be regarded as stored fuel. Similar instances of the fat storages prior to migration and utilisation during migration have also been reported in a number of fishes (Pattor et al., 1970; Robinson and Mead, 1970). This indicated the participation of lipids in the energetic aspects of the fish during stress conditions and the same can be expected in fishes exposed to toxicants. However, very little information is available on the shifts of fat reserves in freshwater fishes exposed to heavy metals. Further, studies on total fatty acids of fishes exposed to toxicants, particularly heavy metals, are scanty.
Utilisation of lipid reserves involves several biochemical changes, and the energy yielding processes of fatty acid oxidation is known to precede by the release of fatty acids from triglycerides and and then they are transported to the site of utilisation. The physiological role of lipase in mobilisation of lipids consists essentially in promoting the hydrolysis of fat deposits (Bilinski, 1969) and the synthesis of neutral fats by its reverse action (George and Talesere, 1962). Earlier studies on lipase activity were directed towards correlating its activity to the general activity of the fish. In general, the lipase activity is higher in more active fish than in those forms which are sluggish (Chesley, 1934). In many species of fish the fat deposits consists largely triglycerides, long-chain fatty acids having carbon chain lengths generally ranging from 12 to 24 carbons, and fish muscle is known to contain a lipase capable of catalyzing the hydrolysis of short-chain triglycerides rather than the long-chain ones (George, 1962). George et al., (1958) generalised that the occurrence of such high levels of lipase activity necessarily reflects correspondingly high levels of fat utilisation. But studies on fat metabolism including the lipase activity in freshwater fishes exposed to heavy metals have not been carried out so far.

Studies on mammals indicated increase utilisation of free fatty acids during fasting and vigorous exercise (Pruett, 1970; Seven, 1971). Free fatty acids have been studied in fishes too, but most of these studies are pertained to their metabolic utilisation in relation
to starvation (Mazeaud, 1973; Larrson and Lewander, 1973; Ince and Thorpe, 1976; Gorendave, 1976). However, studies on the role of free fatty acids in the energetics of the fish exposed to heavy metal stress have not been undertaken so far.

Glycerol and other similar substances may exert considerable protective influence on cellular enzymes by preventing changes in their tertiary and quaternary structure and thus maintain the important cellular enzymes in an active state during stress conditions (Devries, 1970; Hochachka and Somero, 1973). In such case glycerol can be expected to play a considerable role in the animals exposed to toxicants. However, studies on the role of glycerol in freshwater fishes exposed to heavy metals have not so far been undertaken. The glycerol moiety of fat hydrolysis seems to be converted to glucose and then to glycogen which is said to contribute to high levels of glycogen in the red muscle of the fishes (Bokdawala and George, 1967). Glycerol being highly soluble in water, it readily diffuses into the blood stream from the site of its formation through fat hydrolysis and then transported to liver, where it is phosphorylated to \( \alpha \)-glycerophosphate in the presence of an enzyme glycerokinase. Fried et al., (1969) reported that the teleost liver has relatively low \( \alpha \)-glycerophosphate dehydrogenase activity, imply greater dependence on the part of fishes on \( \alpha \)-glycerophosphate production through glycerokinase reaction. A high rate of glycerol incorporation into glycogen has been observed in the muscles
of lamprey during its spawning migration and said to play an important role in replenishing glycogen in this tissue (Savina and Wojtczak, 1977). These studies indicating the importance of the role of glycerol and call for studies on this substance in fishes exposed to toxicants.

The foregoing data on a few parameters of lipid metabolism indicates the possible involvement of this metabolism in animals under stress conditions. However, studies on lipid metabolism in freshwater fishes exposed to heavy metals are scanty. Rama and Ajaykumar (1980) reported significant changes in lipid metabolism of albino rats, *Ratus ratus*, exposed to copper. Hence an attempt is made in this investigation to study the shifts in a few aspects of lipid metabolism, involving the levels of total lipids, total fatty acids, free fatty acids and glycerol and the activity of lipase in different organs of the fish, *C. carpio*, on exposure to lethal and sublethal concentrations of mercury.

There are evidences for physiological categorisation of organs in fish and the gill, kidney and intestine with direct roles in osmo and ionoregulatory processes being grouped as osmoregulatory organs and the others like brain, liver, muscle and so on without such direct roles in the osmo- and ionoregulatory processes being grouped as non-osmoregulatory organs (Schmidt-Nielson, 1974, Bashamohideen and Parvatheswararao, 1976, Radhakrishnaiah, 1984). Perhaps, the physiological and biochemical responses to pollutants may differ between these two categories of organs, and there is evidence that
in fishes osmoregulatory failure is one of the important causes for death at acute exposures to heavy metals (Renfro et al., 1974). Further, there are a few reports which are relevant in this context that the osmoregulatory enzymes have a lipid base in their structure (Jackim et al., 1970; Kuhnert and Kunhert, 1976; Bouquegnean, 1977; Sheppard and Simkiss, 1978; Watson and Beamish, 1980; Bala Venkatasubbaiah et al., 1983), and hence differential changes in lipid metabolism, if any, can be expected in between the osmoregulatory and non-osmoregulatory organs of fish. So, the present study has been carried out in the gill, kidney, intestine, brain, liver and muscle of the fish representing both the osmoregulatory and non-osmoregulatory categories.