SUMMARY AND CONCLUSIONS

The fish, *Channa punctatus* were exposed to 3 mg/l CuSO$_4$ and 16 mg/l ZnSO$_4$ separately for 30 days (Chronic study). Similarly the fishes were exposed to 30 mg/l CuSO$_4$ and 160 mg/l ZnSO$_4$ (Acute study).

No mortality was recorded in control as well as experimental fishes after chronic exposure.

Fishes collected from natural habitat showed persistence of copper and zinc ranging between 0.19 to 0.22 µg/gm wet
weight and 0.28 to 0.30 µg / gm wet weight respectively.

The static model aquatic ecosystem was prepared in laboratory for exposing the fishes to toxicants.

The static model aquatic ecosystem was prepared in large aquaria containing soil, aquatic plant *hydrilla*, water and fishes.

During acute exposure of ecosystem to Copper sulphate and Zinc sulphate, the aquatic plants found to accumulate more heavy metals, followed by soil, water and then fish tissues.

Copper was found to be more persistent in all the components of aquatic ecosystem. Zinc sulphate was found to be degraded faster in aquatic ecosystem as compared to copper sulphate. More persistence of copper and zinc was seen during lethal exposure.

During sublethal exposure the persistence of copper was found to be in the order of Gill > Liver > Kidney > Muscle > Brain after 10 days of exposure however, after 30 days of exposure the, copper persistence was seen in the order Liver >
Muscle > Gill > kidney > Brain. In muscle and brain the persistence of copper was increased gradually after 30 days and there was gradual depletion in copper contents in gill, kidney and liver, which are the detoxifying organs of the fish.

In recovery phase the copper persistence depleted in all tissues except brain.

During lethal exposure of copper sulphate more persistence of copper was evident in muscle and brain of the fish.

In sublethal exposure of zinc sulphate the persistence of zinc in different tissues of the fish was in the order of Liver > Kidney > Gill > Brain > Muscle after 10 days of exposure however, after 30 days the persistence was in the order of Liver > Brain > Muscle > Gill > Kidney. Persistence of zinc went on increasing and no depletion was seen in it. Muscle showed slight increase in zinc persistence after 30 days to that observed after 20 days of exposure.

In recovery studies there was decrease in the persistence of zinc except brain and muscle. When the fishes were exposed to zinc sulphate (lethal dose) maximum accumulation was noted and it was in the order of Liver > Kidney > Gill > Muscle > Brain after 96 hours.
After exposing the fishes to sublethal dose of copper sulphate blood glucose level was found to increase by 69.11% after 30 days of exposure. When fishes were exposed to sublethal dose of zinc sulphate blood sugar level was depleted by 32.70 % after 30 days.

Serum total proteins were depleted in all experimental fishes after 10 days, there after no significant changes were seen upto 30 days.

Serum cholesterol was found to be increased after chronic exposure and acute exposure to copper sulphate where as fishes exposed to zinc sulphate acutely, exhibited lowered serum cholesterol level.

Glycogen, protein and cholesterol content were estimated in the tissues, liver and muscles of the fish, *Channa punctatus*, after exposing them to copper sulphate and zinc sulphate separately.

Liver and muscle protein were found to be depleted significantly in all the experimental fishes lowering down the nutritive value of the fish.
Significant decrease in tissue cholesterol was observed which could be due to utilization of cholesterol by the fish in the intermediary metabolism when the fishes are under chronic stress. Decrease in the tissue cholesterol also indicated retardation in fat metabolism due to impaired hepatic function.

The persistence of copper and zinc in gill resulted into decreased branchial electrolytes and total alterations in ions regulation of the fish.

The present investigations revealed more toxic nature of copper sulphate as compared to zinc sulphate and both metals showed in persistence even after a long duration resulting into harmful effect on the fish reducing its nutritive value.