SUMMARY

1. The 24 h median lethal concentration of aluminium sulphate to fish was 44.90 ppm and the same for 96 h was 26.42 ppm, indicating that the metal is toxic to fish.

2. Addition of median lethal concentration of aluminium sulphate reduced the pH of the test water from 7.5±1 to 4.9±1. In this pH range polymerisation of monomeric aluminium hydroxide species to higher molecular species may occur, which is highly toxic to fish.

3. Deposition of toxic aluminium polymers on the gill surface might have caused structural alterations such as edema, lamellar fusion of gills and profuse secretion of mucus causing severe clogging of the interlamellar spaces. The above changes might have resulted in impaired gaseous exchange in gills leading to mortality of fish during acute treatment.

4. Na⁺ and Cl⁻ ions were reduced in the plasma of fish exposed to acute and sublethal concentrations of aluminium. This may be due to the deposition of aluminium polymers on the gills leading to reduction in ionic exchange for transport, or due to the inhibition of active ion influx, or stimulation of passive efflux through paracellular channels or due to the inhibition of transporting enzyme in gills.
Elevation in plasma potassium in the present study may be due to rupture of cell membranes causing release of cellular potassium into the plasma, or reduction in the extracellular space or decrease in the plasma volume.

Blood pH and HCO₃⁻ levels of fish treated with acute aluminium toxicity decreased significantly. However, in sublethal treatment the above changes were not significant. Surplus H⁺ ion produced during aluminium hydrolysis, or aluminium induced hypoxia due to impaired gaseous exchange provoking lactic acid, or PCO₂ accumulation or inhibition of acid-base relevant ion exchange might be the probable causes of blood pH decline observed in the present study. Decline of HCO₃⁻ level in the plasma in the present study may be due to inhibition of carbonic anhydrase enzyme.

Fish exposed to both acute and sublethal studies exhibited reduced levels of hemoglobin, hematocrit and RBC count than that of the control. This may be due to disturbance in the fluid volume balance or inhibition of erythropoiesis, or erythroclasia, or interference of hemometabolism by aluminium ions. Leucocytosis observed in the acute and sublethal treatments is probably due to stimulated immunological mechanism against aluminium stress. Swelling of red blood cells may be the reason for the significant changes in MCHC and MCV values in sublethal study.

The decrease in the plasma protein content in the present study during acute and sublethal treatments may be due to rapid utilisation of protein, or increased proteolysis or reduction in the protein synthesis by the fish under aluminium...
stress. During sublethal treatment hypoxia may be responsible for the elevation of plasma protein content which may act as an osmoeffect particle consequent to ion disturbance caused by aluminium toxicity.

9. Plasma glucose was elevated in both acute and sublethal treatments. This may be due to enhanced plasma cortisol level in the experimental fish resulting in glycogenolysis or gluconeogenesis to provide additional energy during the times of metabolic stress.

10. Gill and plasma Na⁺ K⁺ ATPase activity of fish was inhibited by aluminium toxicity both in acute and sublethal studies. This may be due to direct inhibition of enzyme by aluminium ions or replacement of normal co-factor Mg²⁺ ions of the enzyme or 'chloride cell' damage. The increase in the plasma Na⁺ K⁺ ATPase in the sublethal treatment after 21st day may be due to 'hormesis' (Yang and Randall, 1996) which may be a compensation process to inhibitory challenges produced by aluminium.

11. Both acid and alkaline phosphatases activities increased in the experimental fish which may be due to lysosomal membrane damage causing leakage of cellular enzyme into plasma. However, a decrease in the plasma ALP activity was recorded in the sublethal treatment. This may be attributed to direct enzyme inhibition by aluminium metal which is reported to have a high affinity interaction with calmodulin, the normal activator of the enzyme (Ganrot, 1986).