CHAPTER - II

AIM OF THE INVESTIGATION
Dental and skeletal manifestations resulting from a deficiency or an excess of fluoride are indicative of a profound effect of fluoride on the health and well being of an organism. The effects of fluoride are not only confined to skeletal and dental tissues and the deleterious effects of fluoride on soft tissues are receiving greater attention now. These effects are very large and varied and the mechanism of fluoride action remains still elusive. However, studies have been concentrated to provide an insight into the role of fluoride by studies in experimental animals treated with either low or toxic amounts of fluoride, acute or chronic and also in human beings living in endemic areas of low or high fluoride.

Considerable data on fluoride toxicity in laboratory animals are now available. Unfortunately, the doses and routes of fluoride administered have been at the Scientists' discretion, resulting in reporting varied toxic effects of fluoride.

The maintenance of stable levels of glucose in blood is one of the most finely regulated of all homeostatic mechanisms in which the liver, kidney and other tissues, several hormones and factors play a part. One response to an acute toxic dose of fluoride in rabbits and rats is marked hyperglycemia. Earlier work on the mechanism have led to suggest the mediation of catechol-
amines, glucocorticoids and central nervous system in the mediation of fluoride-induced hyperglycemia. Hyperglycemia may result from increased glucose production by glycogenolysis in the liver and/or by increased gluconeogenesis in the kidney and liver and/or decreased peripheral utilization of glucose, emphasizing the role of the kidney and liver in glucose homeostasis. However, not much attention has been paid to the role of kidney and liver to this marked hyperglycemic response observed in animals treated with acute toxic dose of fluoride.

The present investigation is primarily aimed at in rats: (1) The relative toxic effects in relation to the hyperglycemic response to different doses of fluoride and to the same dose of fluoride when administered by two different routes i.e. oral and intraperitoneal. (2) The role of liver and kidney in the fluoride-induced hyperglycemia.