On Effect of Chlordane on the Blood Glucose and of Glucose Administration on the Acute Chlordane Toxicity in *Meriones hurrianae* Jerdon, the Indian Desert Gerbil

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Various reports on the effect of insecticides on the central nervous system and liver are available (HART and FOUTS 1963, WITTER and FARRIOR 1964, HATHWAY, 1965 and KIMBROUGH et al., 1971) but their exact biochemical effect on blood of mammals is not completely known. In earlier studies the authors have reported the effect of pyrethrum on the haemoglobin (SAXENA and KAREL, 1974) and an alternation in protein metabolism as indicated by hyperproteinemia (SAXENA and KAREL, 1975). Further an alternation in enzyme activity as reflected by increased serum alkaline phosphatase level with acute doses of chlordane (KAREL and SAXENA, 1975a) and effect on haematology of Indian desert gerbils treated with chronic doses of chlordane is recorded (KAREL and SAXENA, 1974). In the present investigation, it is intended to record the effect of different acute doses of chlordane on the blood glucose and of glucose tolerence in chlordane intoxicated Indian desert gerbils, Meriones hurrianae Jerdon.

MATERIALS AND METHODS

Healthy adult male gerbils weighing 70 to 90 gms, fed wheat bread, grass and wet gram, were used in the present investigation. The animals were acclimatized before subjecting to administration of chlordane. Technical grade chlordane, obtained from Bharat Pulverising Mills Pvt. Ltd. Bombay as gratis, was diluted by petroleum ether to different required concentrations (1.0%, 2.0% and 3.0%). Blood glucose concentration was estimated by the method of Nelson and Somogyi (SOMOGYI, 1945).

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DESIGN OF EXPERIMENT

EXPERIMENT NO.1 :

The experiment was performed to record the glucose level in the blood of gerbils administered with 0.2 cc of 1.0% (25 mg/kg body weight) chlordane. 40 healthy adult male gerbils divided into two groups, experimental and control were used in this experiment. 0.2 cc of 1.0% chlordane was administered by intramuscular injection into the thigh of animals of experimental group. Control group animals received 0.2 cc of petroleum ether only. The animals were autopsied 1,2,3 hours and 1,2,3,7 and 14 days after the treatment. At each autopsy the blood was taken separately from experimental and control animals. 4 animals were sacrificed at each autopsy and the glucose concentration was estimated in both experimental and control gerbils.

EXPERIMENT NO.2 :

The experimental design was similar to experiment No.1. 0.2 cc of 2.0% (50 mg/kg body weight) of chlordane was administered to animals of experimental group while control animals received 0.2 cc of petroleum ether only.

EXPERIMENT NO.3:

The experimental design was similar to experiment No.1. 0.2 cc of 3.0% (75 mg/kg body wt.) chlordane was administered to experimental animals. Control group animals received 0.2 cc of petroleum ether only.

EXPERIMENT NO.4 :

In this experiment it was intended to test glucose tolerance in gerbils administered with 0.2 cc of 1.0% (25 mg/kg body weight) chlordane. 0.1 ml of blood was collected by cutting the tips of tails of animals fasted for 20-24 hours. A 50%

glucose solution was then administered orally by cannula at a dose level of 2 gm/kg of body weight. Thereafter the blood samples were collected at 45 minutes intervals for a period of three hours and blood glucose level was estimated.

Results

It is recorded that chlordane causes hyperglycemia (increased glucose concentration) in the blood of treated animals (Fig.1,2,and 3). A maximum level of blood glucose is attained after an hour of treatment which remains elevated well above the control values the following day and then falls gradually approaching control values within a week after the administration of insecticide. Further, it is observed that the decrease in blood glucose level is more pronounced in gerbils treated with higher concentrations (2.0% and 3.0%) than those treated with low concentration (1.0%) of chlordane (Fig.1, 2 and 3).

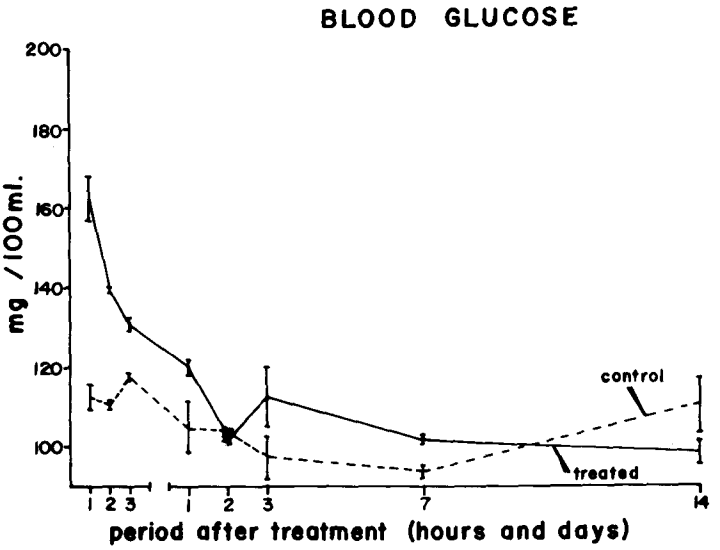

Fig.1 Acute toxic effect on blood glucose level of 0.2 cc of 1.0% chlordane administered by intramuscular injection to male Indian desert gerbil.

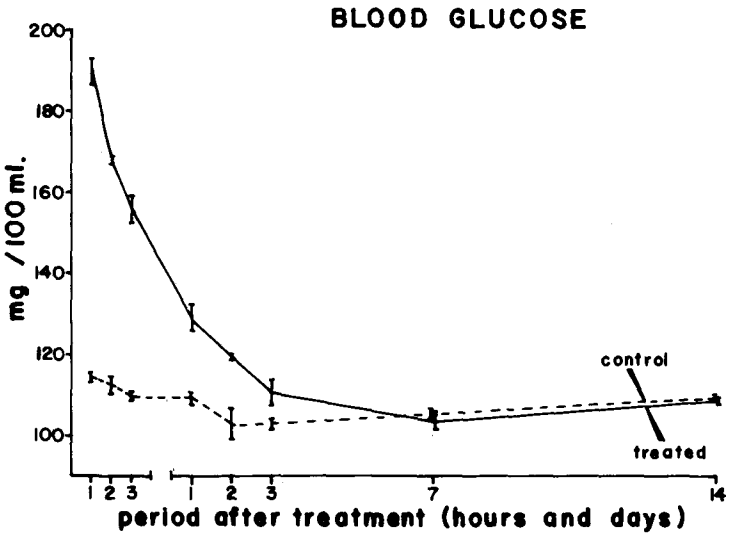

Fig.2 Acute toxic effect on blood glucose level of 0.2 cc of 2.0% chlordane administered by intramuscular injection to male Indian desert gerbil.

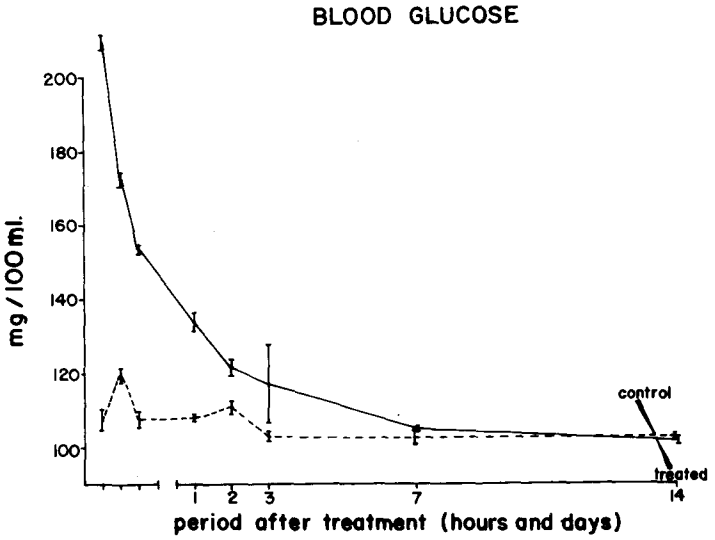

Fig.3 Acute toxic effect on blood glucose level of 0.2 cc of 3.0% chlordane administered by intramuscular injection to male Indian desert gerbil.

In experiment conducted to find out the glucose tolerance in chlordane treated gerbils, an elevation in blood glucose level confirming a decrease in glucose tolerance is recorded (Fig.4).

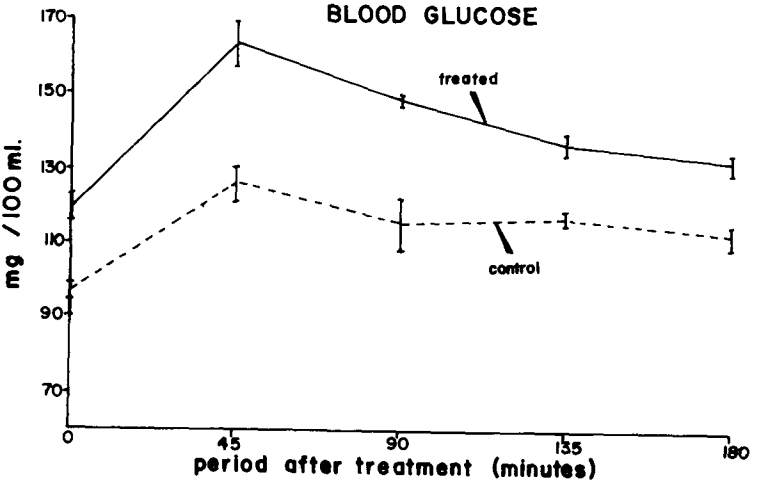

Fig.4 Effect of glucose administration on the blood glucose level of Indian desert gerbil following the administration of 0.2 cc of 1.0% chlordane.

Discussion

The hyperglycemic condition in the blood of chlordane intoxicated gerbils is attributed to increased glucose production by the liver. The effect of chlordane involves some sort of activation of sympathetic nervous system. The sympathetic nervous system then releases adrenaline from adrenal medulla which stimulates the adenohypophysis to secrete increased amounts of adrenocorticotropic hormone (ACTH). ACTH, then activates the adrenal cortex to produce more glucocorticoid hormones. The glucocorticoids then stimulate the hepatic glucose production. The hepatic glucose is, then, released from the liver into the blood and thus

increase the blood glucose level. This suggestion is confirmed by the fact that in glucocorticoid administered animals there is a rise in glucose concentration (CLEGG and CLEGG, 1969). The glucose produce due to glucocorticoids comes from some endogeneous source as CLEGG and CLEGG (1969) have reported the same effect in fasting animals. According to KAREL and SAXENA (1975b) the glucose appearing after the increased production of glucocorticoids probably arises from the break down of proteins (gluconeogenesis).

In the presence of glucagon and in the absence of insulin, the liver secretes glucose in the blood and vice versa. Therefore, it is presumed that the administration of insecticide somehow stimulates glucagon secretion and inhibits the synthesis of insulin in chlordane intoxicated gerbils. The insecticide may cause selective destruction of cells of islets of langerhans and thereby result in hyperglycemia.

Further, an elevation in blood glucose, as a result of glucose administration in the chlordane intoxicated gerbils indicates a decrease in glucose tolerance. This observation suggests that chlordane causes an impairment in the uptake and utilization of glucose in the body of intoxicated gerbils. The present observation finds support from BHATIA et al.,(1972) and KAREL and SAXENA (1975b).

The observations in the present investigation find support from the work of STOHLMAN and LILLIE (1948) ROSEN etlal, (1959), WIBER et al., (1964) and MURPHY PORTOR, (1966) and KAREL and SAXENA (1975b) who reported hyperglycemia in rats administered with different insecticides and irritant chemicals.

The recovery in blood glucose may be attributed to metabolism of chlordane in the body of intoxicated animals. When the concentration of chlordane in the body, as a result of metabolism and excretion out of the body, decreases naturally its effects on the blood glucose level diminishes resulting in the values approaching the control ones

which in due course of time becomes normal to a considerable extent.

Summary

The effect of 25, 50 and 75 mg/kg body wt. chlordane on the blood glucose level and of glucose administration in chlordane intoxicated Indian desert gerbils are investigated. Chlordane produces hyperglycemia and lowers the glucose tolerance indicating an impairment in the uptake and utilization of glucose in intoxicated gerbils. The possible reasons for these effects are discussed.

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