ACUTE EFFECT OF STREPTOZOTOCIN INDUCED DIABETES ON BAR PRESSING FOR FOOD REWARD IN ALBINO RATS

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Summary : The operant behaviour in streptozotocin (45 *mg* or 65 *mg/kg*) treated rats was studied using albino rats of either sex. Animals were trained daily for 15 days in an operant chamber (Takei & Co.) to press the bar for getting the reward in the form of 45 *mg* food pellet following 18 hr of food deprivation. After initial training under continuous reinforcement schedule (CRF), animals were trained under FR_2 and FR_4 schedules, in which after every second (FR_2) or every fourth bar press (FR_4) rat received the pellet.

The results indicate that the bar pressing for food reward is not altered after induction of experimental diabetes with streptozotocin. It is suggested that insulin lack or hyperglycaemia in this condition fails to influence higher centres associated with regulation of motivated behaviour.

Key words : operant behaviour, streptozotocin, hyperglycaemia, food reward

INTRODUCTION

Hyperphagia is a well known manifestation of diabetes mellitus. This hyperphagia is possibly due to glucose lack at cellular level in the absence of insulin. Glucose deprivation of ventromedial hypothalamic cells is known to stimulate the food intake in this condition (1). Motivation for food is also likely to increase in hyperphagia. Satiety level which can be modified by insulin lack is also considered to be an important factor modulating feeding behaviour and motivation (4). To our knowledge, there are no reports of study of operant behaviour in experimental diabetes. Therefore, the present work was planned to determine changes in the bar pressing behaviour in experimental diabetes.

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MATERIAL AND METHODS

Eighteen rats of either sex weighing 150-200 *gm* were used. Diabetes was induced using intraperitoneal injections of streptozotocin (UpJohn Co., Kalamazoo, Michigan, USA) 45 *mg/kg*, b.w and 65 *mg/kg*, b.w. Streptozotocin was dissolved in saline acidified to pH 4.5 with citrate.

Blood glucose was estimated using a glucometer (AMES - Glucometer). The instrument was standardised by using known standards with the method of Hagedorn and Jensen (2). Operant behaviour was observed after 18 hr of fasting in an operant chamber (Takei & Co.) for 15 min at the same time every day. Food reward used was a 45 *mg* pellet of bengal gram.

Animals were initially trained under continuous reinforcement schedule (CRF), where they received one food pellet for every bar press. Then the animals were trained under FR_2 and FR_4 schedules till they attained a constant level (asymptote) of bar pressing in FR_4 schedule. The animal received one food pellet for every second (FR_2) or for every fourth (FR_4) bar press.

After their training the animals were randomly divided into 3 groups. Group II and Groups III rats were made diabetic by ip administration of streptozotocin in the dose of 45 *mg/kg* and 65 *mg/kg*, b.w. respectively. Bar pressing activity under FR_4 schedule was recorded in Group II and Group III rats from the second day of the streptozotocin injection. Group I rat received ip injections of the vehicle and served as control and they also continued bar pressing along with experimental groups.

RESULTS

Fasting blood glucose in Group II and Group III was increased as compared to control Group I. Hyperglycemia seen in Group II and III reveals that serverity of diabetes was more in Group III. Blood glucose (*mg*%) of Group I, Group I and Group III was respectively 59.6 ± 8.5, 210 ± 10 and 254 ± 25. Bar pressing, however, did not change after induction of diabetes. Rate of bar pressing remained the same as that in the control rats. (Control Group I - 140 ± 8, Group II - 145 ± 6, and Group III - 140 ± 8) (Fig. 1),

DISCUSSION

Absence or low levels of insulin interferes with glucose utilisation by ventromedical nucleus (Satiety centre) of hypothalamus. STZ destroys selecetively B cells of islets of Langerhans and reduces insulin levels in the blood in a dose related manner (3). Resul-

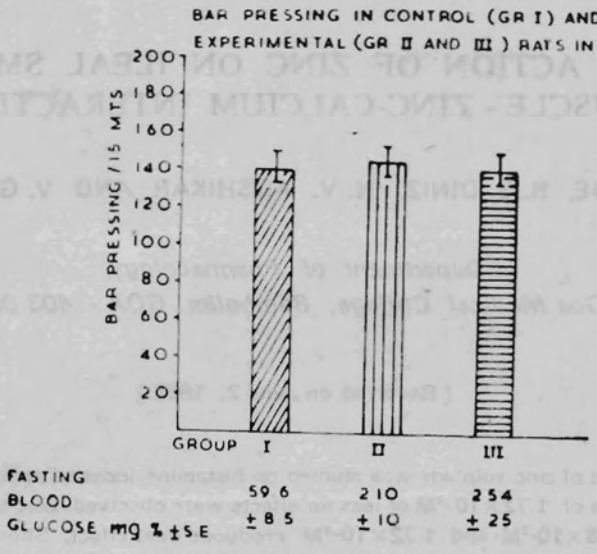

Fig. 1 : Showing bar pressing under FR_4 schedule and blood glucose in control (GR. I) and experimental rats (GR. II & III). Each bar indicates mean value of 6 rats for 10 days. Vertical line indicates S.E.

tant hoperyhagia in this type of diabetes is due to low insulin levels reducing thereby the activity of the satiety centre, even in the face of hyperglycaemia (1). The present study shows that insulin lack fails to alter the bar pressing activity suggesting that bar pressing behaviour attained during euglycaemia or prediabetic state is not affected after induction of diabetes and resultant hyperglycaemia. It is likely that the activity of physiological mechanisms and higher centres controlling operant feeding behaviour is not affected by STZ induced diabetes of moderate or severe degree and also by lack of Insulin. Our results pertain to early stages of diabetes. Whether this behaviour will be affected by diabetes of long duration associated with severe metabolic disturbances is under investigation.

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