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EFFECT OF DIFFERENT FEEDING SCHEDULES ON FOOD INTAKE IN CAUDATE LESIONED RATS

D. P. THOMBRE*, N. KRISHNAMURTHY AND G. K. PAL**

Department of Physiology,
Jawaharlal Institute of Postgraduate Medical Education and Research,
Pondicherry - 605 006

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Abstract: Inbred albino rats were exposed to different feeding schedules before and after caudate nucleus lesions. The animals show adaptation to the new patterns of food deprivation even after caudate nucleus lesions. However, the lesioned animals are not able to elevate their food intake or body weight to the prelesion levels. These findings suggest that feeding, satiety and body weight mechanisms are disturbed in the absence of intact caudate nucleus, possibly due to removal of nigro-striatal dopamine influence.

Key words: caudate nucleus feeding schedules operant behaviour food intake

INTRODUCTION

Diminished energy stores and light-dark cycles are amongst the important determinants of food intake in rats (1, 2). If the availability of food is restricted to short periods of a day, the rats elevate food consumption to *ad libitum* level even during limited access to food (3, 4). This adaptation may be dependent on dopaminergic systems influencing feeding satiety mechanism, since food intake is reduced by electrolytic lesions of striatum and globus pallidus (5, 6). Depletion of dopamine in brain also suppressed food intake and operant behaviour in rats (7).

In order to assess the role of nigro-striatal dopaminergic terminals in such adaptation, this study was designed to evaluate feeding response and operant behaviour of caudate (CP) lesioned rats to varying periods of food deprivation.

METHODS

Twentyfour rats (bw 175-225 g) were housed individually in rat cages under natural light-dark cycles and ambient temperature of 25° ± 1°C. Water was available *ad lib.*

Food intake : According to the duration of food availability, rats were assigned to three groups of eight each. Group I rats had access to food (rat chow) throughout 24 h. For the Group II and III, food was available for 18 hr (15.00 to 9.00 hr) and 6 hr (9. 00 to 15.00 hr) respectively.

Food intake was measured accurately upto 0.1 gm. Animals were weighed on alternate days. After the intake was stabilized the animals were subjected to caudate lesions and one week after the lesion intake was monitored for 20 days. In all groups the intake stabilized during the last 10 days of monitoring.

Operant behaviour: Sham (n=6) and experimental group (n=8) with lesion of CP were concurrently trained for bar pressing for food reward in an operant chamber (Takei & Co., Japan). Each training session lasted for 15 min every morning after 22 hr food deprivation. Food pellets (45 mg) were prepared from soaked bengal gram.

Every animal was first trained under continuous reinforcement (CRF) followed by FR_2 and FR_4 schedules (fixed ratio schedule of 2:1 and 4:1). Training

*Corresponding Author : **Present address : Department of Physiology, V.S.S. Medical College, Burla (Orissa)

was continued till the animal reached asymptote under each schedule.

Surgery and Histology: Under nembutal anaesthesia (45 mg/kg, ip), bilateral CP lesions were produced by passing 1 mA anodal current for 15-20 sec through stainless steel electrodes, insulated except at its tip; using co-ordinates of Konig & Klippel (6). No current was allowed to pass in sham group.

The animals were sacrificed at the end of the study and intracardiac 0.9% saline followed by 10% formalin were administered immediately for histological processing to confirm the lesions.

RESULTS

1. *Food intake and body weight:* When restrictions were imposed on access to food, the rats consumed 12.6 ± 0.3 gm and 11.2 ± 0.4 gm in 6 hr and 18 hr respectively. This consumption was not different from that of *ad lib* consumption (11.9 ± 0.7 gm) in all animals before lesion.

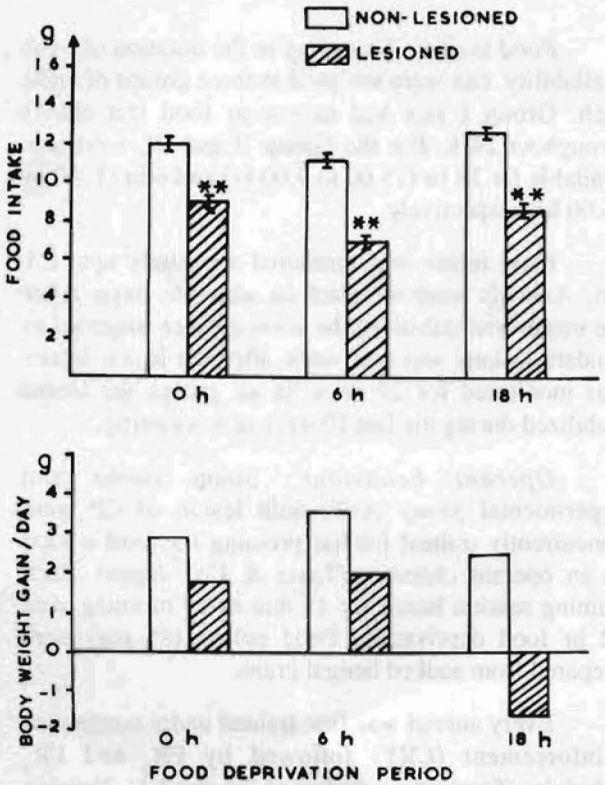

Fig. 1: Showing food intake (above) and change in body weight (below) before and after caudate lesions. 0 hr deprivation period corresponds to *ad lib* feeding. **P < 0.001.

After lesion of CP, food intakes in 6 hr in Group III (8.4 ± 0.2 g) and in 18 hr in Group II rats (7.5 ± 0.1 g) were not different statistically from the *ad lib* intake in Group I rats (8.60 ± 0.6 g). However, these lesioned animals maintained significantly low food intake throughout the period of study and showed either decreased rate of body weight gain (Group I and II) or actual reduction in body weight (Group III) (Fig.1).

2. *Operant behaviour:* The lesioned animals showed higher rates of bar pressing under CRF and FR_2 schedules, when compared with sham animals. However, the rates of bar pressings were not significantly different in these two groups under FR_4 schedule (Table I).

TABLE I : Bar pressing rates for 15 min sessions in non-lesioned and lesioned rats.

Fixed ratio schedules	*Non-lesioned rats (n=6)*	*Lesioned rats (n=8)*
CRF	78.3 ± 1.86	86.83 ± 4.16**
FR_2	164.8 ± 4.79	185.66 ± 14.93*
Fr_4	244.3 ± 23.74	273.00 ± 24.84

**P < 0.001; *P < 0.05

DISCUSSION

Consistently hypophagia is induced by caudate lesions. Destruction of dopaminergic terminals (7, 9, 10) also leads to similar results.

Feeding schedules imposed in this experiment, not only limit the duration of food availability but also lead to homeostatic disturbances in body energy levels. Both the control and lesioned animals restore their food intake to *ad lib* levels during various period of deprivation, but this capacity is limited in experimental group to the new lower level of food consumption. It is likely that damage to CP or nigro-striatal dopaminergic system alters the degree of satiety or sensation of hunger (11).

Injection of dopamine antagonists into caudate nucleus in rats also suppress food intake (12). In this series, the lesions did not lead to motor deficits as seen by the normal operant behaviour.

Ability to elevate food intake in the face of energy depletion in lesioned animals may be possible with the mobilisation of other dopaminergic system, activated due to stress (13).

In lesioned rats, loss of body weight was conspicuous when food was available for 6 hr only during day time. Control rats also showed reduction in gain of body weights when they were subjected to diurnal schedule. Such findings in non-lesioned rats are reported by other workers (1). It is possible that this day time normal food intake was not enough to meet the energy output for the nocturnal activity of rats.

The lesions of CP failed to suppress the operant behaviour in our study. Other workers (7, 13) have demonstrated that widespread destruction of dopaminergic systems lead to inhibition of this function. Such activities for food reward are probably regulated by neural systems other than the nigro-striatal.

This study suggests that feeding, satiety and body weight mechanisms under conditions of availability or deprivation of food are disturbed, after lesions of CP possibly due to loss of nigro-striatal dopaminergic influence.

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