INFLUENCE OF REINFORCER PALATABILITY ON FR-32 OPERANT PERFORMANCE OF DORSOLATERAL TEGMENTAL RATS

by

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ABSTRACT

Influence of Reinforcer Palatability on FR-32 Operant Performance of Dorsolateral Tegmental Rats

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The effects of dorsolateral tegmental (DLT) lesions on female rats were examined through use of an operant barpress paradigm. The DLT lesioned rats exhibited hyperphagia and obesity. It was found that in DLT rats normal serum glucose levels existed in both day and night phases with hyperinsulinemia present during the day and the night. Interscapular brown adipose tissue (IBAT) upon extraction was found to be less in DLT rats. Upon examination of acquisition of the barpress response no differences were found among the groups (control, DLT, operate controls) at fixed ratio (FR)-32 for chow pellets. Sucrose pellet and amphetamine (injected 30 minutes prior to chow pellet manipulation) both produced suppressed responding in the animals. So, although tegmental rats overeat and become obese, these effects may not be due to increased hunger motivation as indexed by an operant conditioning paradigm.

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TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INTRODUCTION

Electrolytic lesions of the dorsolateral tegmentum (DLT), a brainstem region located just below the central gray, will elicit in rats a variety of altered behavioral responses. DLT rats exhibit obesity, nocturnal hyperphagia and attenuated amphetamine anorexia. More precisely, DLT rats overeat, becoming obese, and continue to eat after treatment with amphetamine, a drug that reduces food intake in normal rats. (Ahlskog, 1974; Ahlskog and Hoebel, 1973; Ahlskog, Hoebel, and Randall, 1975; Wellman and Peters, 1980.) Interscapular brown adipose tissue of DLT rats has been found to display marked atrophy, and incorporation of (14-C)-glucose is significantly reduced suggesting lower activity (Note [1]). Inasmuch as brown adipose tissue serves to burn off excess calories, thereby preventing obesity, defective metabolism in brown fat of DLT rats may contribute to their obesity. Thus, DLT rats may not only eat more than neurologically intact rats, but are also more efficient at storing fat. Tegmental rats also display hyperinsulinemia during the day, and during the hyperphagic night stage (Note [2]). This suggests that the hyperphagia is not a direct result of hyperinsulinemia, but that hyperinsulinemia may in part produce obesity (i.e., high insulin levels cause glucose to enter cells to be deposited as fat).

Consideration of the characteristics of tegmental rats leads to the idea that perhaps these lesions disrupt a brain satiety mechanism such that feeding is no longer inhibited and that drugs such as amphetamine cannot activate this satiety mechanism to induce anorexia. A disrupted

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tegmental satiety mechanism seems indicated in that rats undergoing DLT surgery exhibit increased food consumption following surgery. Messages from the gut that may signal ingestion may not be interpreted by the brain, thereby increasing hunger motivation and willingness to work for food. Similarly, drugs that produce anorexia, such as amphetamine, may do so by activating the tegmentum in such a way that lesions of the tegmentum attenuate amphetamine anorexia.

Lesions of the tegmentum also increase peripheral insulin levels (Note [2]). Insulin injections produce feeding in satiated normal rats, presumably by lowering blood glucose level. Increased insulin level would decrease the amount of blood glucose present in DLT rats and, in turn, produce increased food intake. The increased feeling of hunger produced by insulin is typically indexed by increased food intake, or an increase in activity when seeking food.

In searching further for an answer as to why DLT rats overeat and become obese, another peripheral area needs to be considered, more specifically the disruption of peripheral brown adipose metabolism. Hyperphagic tegmental rats have been found to display a decrease in interscapular brown adipose tissue mass. This tissue, located between the shoulder blades, burns off excess calories. DLT lesions decrease the amount of brown fat available to burn excess calories; an outcome that could explain why DLT rats become obese although overeating is prevented by feeding control and DLT rats an equivalent amount of food (Luttmers, 1978).

Were a satiety mechanism to be disrupted or peripheral insulin levels increased, tegmental rats should display increased hunger motivation, or an increased willingness to work for food. Behaviorally, there are several ways to assess changes of hunger motivation (Miller, 1957). These include:

techniques such as runways, electroshock, and barpressing for food. In runway tasks, in which rats are allowed to run to a goal box containing food, the running speed down the runway is a dependent measure of the magnitude of hunger motivation. Rats deprived of food for longer periods display faster running speeds. Electroshock paradigms assess hunger motivation by measuring the level of shock tolerated, the hungrier the animal is likely to be, whereas the more sated an animal the more likely a low level of shock will stop or suppress feeding. The most sensitive method and that of choice is barpressing for food. In a Fixed Ratio (FR) schedule of reinforcement, an animal must emit a predetermined number of responses to acquire a piece of food. Level of food motivation is primarily related to the number and speed of barpresses that an animal makes to acquire food. Hungrier animals press faster and acquire more pellets than satiated animals in a FR paradigm during a thirty minute test. Moreover, it has been demonstrated that once an animal begins to press it does so at a constant rate until the food is obtained. Following ingestion of the pellet the animal pauses for a period of time and then begins to respond, at a constant rate, until the next food pellet is obtained (Felton and Lyon, 1966). Presumably pause times (i.e., how long the animal waits to initiate a string of presses) is related to its hunger motivation such that hungrier rats should have shorter pause times.

The purpose of the present study is to examine the hunger motivation of the dorsolateral tegmental rat using an operant conditioning paradigm. Because of the nature of the tegmental rat, several considerations were made. Firstly, commercially available chow food pellets (45 mg Noyes pellets) are not a palatable food. In as much as DLT rats have been shown to overeat only very palatable diets (i.e., cookies, junk food, high-fat

diets, Peters, Wellman and Gunion, 1979), and display normal food intake and weight gain when fed a diet comparable to a Noyes pellet, one would not expect enhanced barpressing in DLT rats for chow pellets. Thus, in the present study, the acquisition of barpress behavior of control and tegmental rats for chow Noyes pellet and the changes in responding when a very palatable sucrose pellet served as reward was examined. It was predicted that tegmental rats would display normal press rates and pause times when such presses produced a chow pellet in an FR-32 paradigm, but faster press rates and shorter pause times when a sucrose pellet was the reward. Finally, the influence of amphetamine on barpress responding for chow pellets was examined in control and tegmental rats. Inasmuch as DLT rats display attenuated amphetamine anorexia in the free-feeding situation (Wellman and Peters, 1980; Ahlskog, 1974), it was predicted that amphetamine treatment (2.0 mg/kg, intraperitoneal) would suppress barpress rates and enhance pause times in control rats, but less markedly so in tegmental rats.

METHOD

Animals

The animals were 26 female Long-Evans hooded rats (obtained from Blue Spruce Farms, Inc.) 90 days old and weighing between 202-268 grams at the outset of the experiment. Each rat was housed individually in plastic rodent cages supplied with raised-grid floors in a temperature-controlled (23°C) room under a normal day/night illumination schedule (lights on at 0800 hours). Unless specified otherwise, the rats were given free access to tap water and food.

Diet

Upon arrival in the laboratory, the animals were fed a standard pellet diet (Purina Rat & Mouse Diet). A high-fat diet consisted of 2 parts ground Purina lab chow and 1 part melted vegetable fat (Crisco). The high-fat diet was prepared fresh every third day and was offered to the rats in glass jars.

Surgery

All rats were treated with 1.0 mg/ml/kg atropine sulfate (ip) and anesthetized with Halothane using a vapor system adapted for a Kopf stereotaxic incisor plate. Because of the rapid induction and recovery characteristics of Halothane, the rats were not deprived of either food or water prior to surgery.

Each surgical electrode was fashioned from 30 gauge nichrome stainless steel wire insulated except for a 0.5 mm conical tip. Bilateral tegmental lesions were produced in 19 rats by passing 1.25 mA cathodal current

for 20 seconds between each electrode and a rectal anode. With the upper incisor bar set at 2.4 mm below the interaural line, each electrode was positioned 2.3 mm anterior to the interaural line, 1.5 mm lateral from the midline and 3.0 mm below the interaural line. The remaining 7 rats served as sham-operated controls that underwent anesthesia and a scalp incision.

Procedure

Table I (p.13) displays the experimental procedures of this study. The rats were offered the high-fat diet for 8 days prior to surgery and 17 days following surgery. The Lee Index of Obesity was recorded for each rat on the day of surgery:

$$\text{Lee Index} = \left[\frac{\text{Body Weight (grams)}^{.33}}{\text{Ano-Nasal Distance (mm)}}\right] \times 10^4$$

Body weights were recorded daily for each rat throughout the experiment whereas food intakes (corrected for spillage) were recorded to the nearest gram for each rat every third day during Days 1-12 following surgery. Day and night food intakes were recorded during days 13 and 14 at the beginning and end of the light period. On days 15 and 17, the rats were deprived of food for 5 hours, anesthetized with halothane and 1.0 ml of whole blood collected via cardiac puncture (23 gauge needle). A day sample (0200-0400 hours) and a night sample (1400-1600 hours) were obtained for each rat on one of these days with order balanced across groups. Each sample was allowed to coagulate at room temperature for 10 minutes, spun down for 5 minutes, and the serum decanted and frozen at -30°C until serum determinations of glucose and insulin (I^{125}; rat insulin standard, Immuno Nuclear) could be carried out.

All the rats were then shifted to a pellet diet for 30 days. In this time period, body weight losses were recorded for all rats. All rats were

Table I. Experimental Protocol

PRE SURGERY - Access to a high-fat diet for eight days

SURGERY - Sham or dorsolateral tegmental

METABOLISM - BW gain (surgery - day 15 following surgery)
Cumulative food intake (72 hour)
Day/night - Food intake
Blood glucose and insulin

POST METABOLISM - Diet down to 80% normal body weight

ACQUISITION - FR 1, 2, 4, 8, 16 and 32 - All pellet
FR-32 Sucrose
Pellet
Sucrose
Pellet
Amphetamine

SACRIFICE - IBAT Weight
Perfuse left ventricle and store brain in 10% formalin for 48 hours
Slice and photograph brain sections

then deprived of food until their weight reached a level 80% of normal body weight, corrected for normal growth. Groups were formed on the basis of body weight gain. Tegmental rats that did not display weight gains that exceeded the average weight gain of the control animals during the 15 day period following surgery were classified as operate-control rats; a procedure that resulted in nine rats forming an operate-control group. Whereas 10 rats were classified as hyperphagic DLT rats. Groups of six rats were then selected to continue the study. The animals were then trained to barpress for chow pellets. A continuous schedule of reinforcement was used in shaping the animals to press for thirty minutes per day. Initial shaping occured for two days with the operant chambers manually operated until the barpress response was learned. Upon mastering the operant task, the rats were maintained on a FR-1 schedule for eight days. Following shaping, the ratio requirement was increased in the following manner: FR-2 (2 days), FR-4 (2 days), FR-8 (2 days), FR-16 (6 days) and FR-32 (8 days). On the 4th and 6th days of FR-32, the normal chow pellet was replaced with a sucrose pellet to examine palatability effects. On day 8 of FR-32, the animals were injected with amphetamine (2.0 mg/kg) 30 minutes prior to barpressing. During the drug manipulation, chow pellets were used as the reward. Following the last session of barpressing all rats were deeply anesthetized with sodium pentobarbital (40 mg/ml/kg,ip) and the dorsal coat overlying the interscapular region was shaved. All visible IBAT was dissected away from muscle and white adipose and weighed to the nearest mg. Lesioned rats were then perfused intracardially with .9% saline followed by buffered 10% formalin. After 48-hours fixation in 10% formalin, the locus and extent of tegmental damage was determined by

examination of photographically enlarged 80μ sections through the coronal plane described by K̈onig and Klippel (1963).

Equipment

The experiment involved the use of three LVE operant chambers programmed by a microcomputer (Apple II, 48K) and an interface (Med Associates Inc). The computer and interface were programmed to record responses, administer reinforcements and to assess time between a reward and the next response. The program recorded any errors which might have occurred during the trials.

RESULTS

The acquisition of the operant response by tegmental rats is depicted in Figure 1 (p.17). The same changes occurred in all 3 groups across the different FR schedules. As the number of responses necessary to get a reward went up, the number of operant responses emitted by each group increased (F (17,255) = 44.6, p <.0001). There was no significant group*day interaction effect; that is, each group displayed similar changes in response rate across the days (F (34,255) = .65, p >.94). The operate controls had the highest number of responses with 2064 on the FR-32 schedule, controls were second with 1848 and DLTs had 1714. These differences among the three groups were not, however, statistically significant. Because these values depicted in Figure 1 represent presses collapsed across days, a day by day analysis of FR-32 response rate is considered in Table IIA (p.18). Analyses of the mean number of barpresses emitted by control, operate control, and DLT rats for a pellet reward on a FR-32 schedule on five successive days finds no significant between-group differences when collapsed across the days (F (2,15) = .32, p >.72). However, a significant effect of day is apparent, as the rats pressed more over the days, represented by an increase in controls from 1702-2010 responses, operate from 1763-2274 responses and DLTs from 1644-1892 responses (F (5,75) = 5.6, p <.0002). There was no significant interaction though between group and day, indicating that each group displayed roughly similar increases in response rate.

Pause time (or the latency from reward to first response following reward) is related to response number (i.e., the pause time should decline as the animal increases the number of responses emitted in thirty

FIGURE 1
ACQUISITON OF BARPRESS RESPONSE

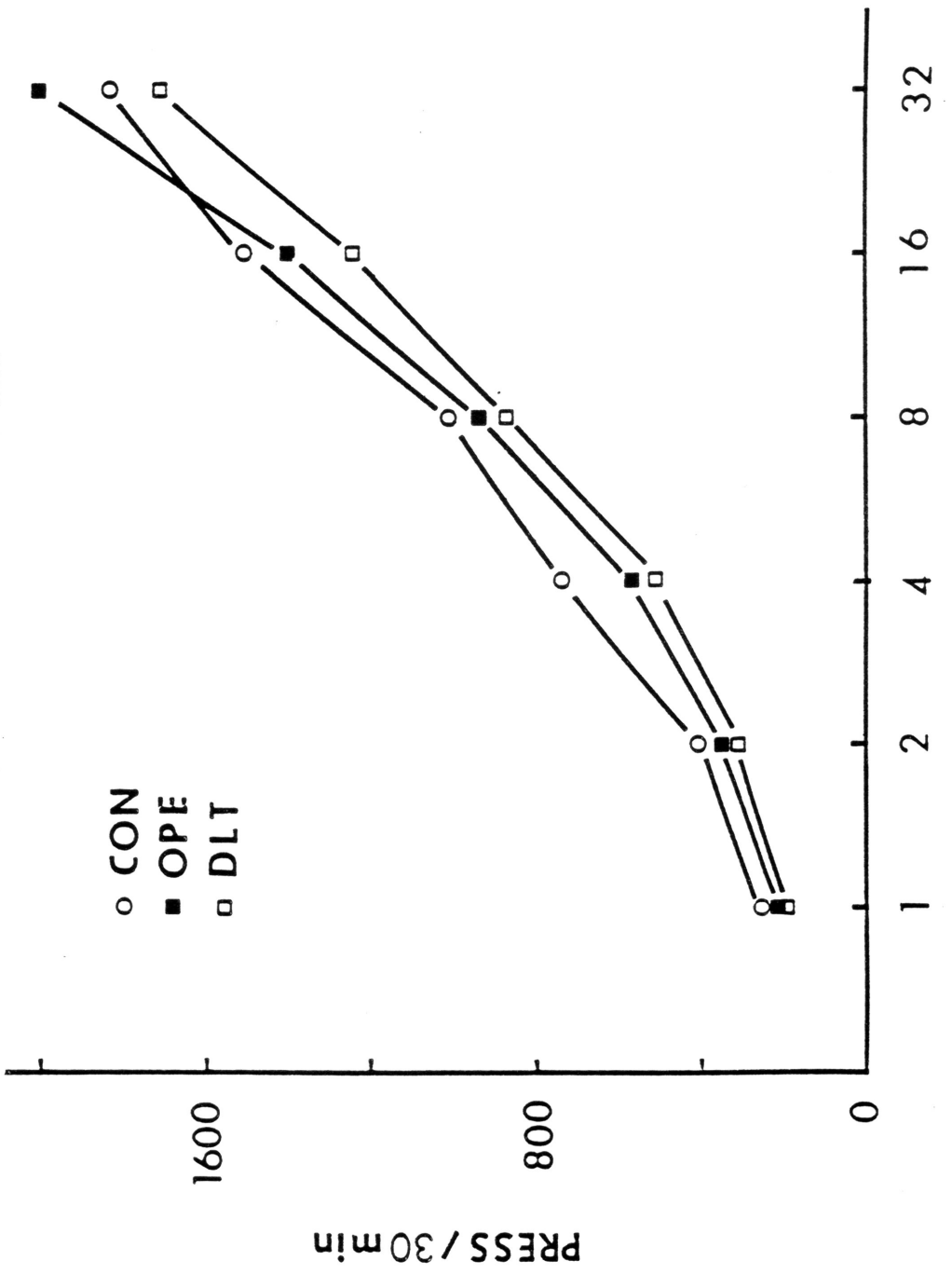


Table IIA Mean number barpresses emitted by Control, Operate control and DLT rats for a pellet reward on a FR-32 schedule on 5 successive days.

Group \ Day	N	1	2	3	4	5
Control	6	1702	1723	1901	1804	2010
Operate	6	1763	2056	2172	2248	2274
DLT	6	1644	1550	1760	1839	1892

Table IIB Mean group pause times (latency from reward to first response following reward) for Control, Operate control and DLT rats for a pellet reward on FR-32 schedule on 5 successive days.

Group \ Day	N	1	2	3	4	5
Control	6	4.1	5.9	3.8	3.9	3.1
Operate	6	3.2	3.5	3.6	3.4	2.9
DLT	6	2.5	4.0	2.9	2.3	2.4

minutes because to do so he must press faster). Analysis of pause time data for the rats on an FR-32 schedule is presented in Table IIB (p.). The controls depicted in Table IIA (p.18) as pressing more each day have in Table IIB correspondingly significantly shorter pause times across the days (Day 1-4.1 sec; Day 5-3.1 sec). The same is true for operates (Day 1-3.2 sec; Day 5-2.9 sec) and DLTs (Day 1-2.5 sec; Day 5-2.4 sec) (F (4,60) = 2.8, p <.04). Again, there were no significant differences between the groups (F (2,15) = .4, p >.68) or an interaction between group and day (F (8,60) = .60, p >.80).

During the sucrose manipulation (depicted in Table IIIA, p.20), the control and DLT rats emitted fewer responses for sucrose pellets (1056, 1663 respectively) than did the operate controls who had an average of 2206 presses. The percent change, as seen in Table IIIA for the controls, was -10%, for operates +7% and for DLTs -3%. Of these three, only the controls showed a significant decrease when given sucrose (t (15) = 2.65, p <.02). In accordance with a reduction in number of bar presses emitted during sucrose manipulation, the pause time for all three groups increased as shown in Table IIIB (p.20). Pause times for controls went from 4.1 sec to 5.1 sec while operates increased from 3.3 sec to 3.8 sec and DLTs increased from 2.8 sec to 3.9 sec. This lengthening of pause times illustrates a significant effect of day for the groups (F (1,15) = 5.0, p <.04). There was no significant group*day interaction (F (2,55) = .22, p <.80). Therefore, each group displayed statistically similar changes when shifted from a pellet diet to a sucrose diet.

In Table IV (p.21) no differential anorexia in the groups is represented under the amphetamine manipulation. Amphetamine, at a concentration of 2.0 mg/kg, produced significant reductions in responding for a

Table IIIA Mean number of barpresses emitted by Control, Operate control and DLT rats on a FR-32 Schedule for a pellet reward (average of days 1-5) and a sucrose reward (average of days 1 and 2).

Group	N	Pellet	Sucrose	% change	p<
Control	6	1849	1056	-10%	.02
Operate	6	2005	2206	+ 7%	NS
DLT	6	1713	1663	- 3%	NS

Table IIIB Mean group pause times for Control, Operate control and DLT rats on a FR-32 schedule for a pellet reward (average of days 1-5) and a sucrose reward (average of days 1 and 2).

Group	N	Pellet	Sucrose	
Control	6	4.1	5.1	
Operate	6	3.3	3.8	p<.05
DLT	6	2.8	3.9	

Table IV Mean group barpresses for a pellet reward on a FR-32 Schedule for Control, Operate control and DLT rats under no-drug and drug (2.0 mg/kg amphetamine, ip) conditions.

Group	N	No-drug	Amphetamine	% change
Control	6	1848	1246	-33%
Operate	6	2064	391	-81%
DLT	6	1713	441	-74%

chow reward in all 3 groups (F (1,15) = 25.5, p <.0001). There were no between-group differences in press rate for all three groups when expressed as percent change (i.e., number presses amphetamine/number presses non-drug; F (2,15) = 2.0, p >.17). The DLT group paradoxically showed the greatest suppression (-74% change) of responding for pellet reward, but the variability was so great that the difference was not statistically significant. In summary, no between-group difference was found, but a significant drug effect was obtained with 2.0 mg/kg injection of amphetamine, decreasing responding for food in all three groups.

Rats with DLT lesions did overeat and become obese when given access to a palatable high-fat diet (F (2,15) = 9.3, p <.001) as can be seen in Table V (p.23). Bodyweight change during a 15 day period following surgery for DLT rats was 59.0 grams, control was 29.8 grams with operates at 29.7 grams. The cumulative food intake measurements illustrate the overeating by DLT rats. The DLT rats had significantly higher cumulative 72 hour food intake (61.4g) than control (44.8g) or operates (46.5g) (F (2,15) = 8.8, p <.002). In 12 hour food intake the DLTs had higher night intakes (11.3g) compared to controls (9.5g) or operates (7.6g) (F (2,15) = 5.9, p <.01). The day intake for 12 hour periods showed no statistical difference in the three groups (F (2,15) = 1.1, p >3.5). There was no statistical difference in serum glucose levels during day (F (2,15) = .34, p >.71) or night (F (2,15) = .67, p >.53). Although differences were present across the groups, they are not significant. Serum insulin levels were higher (71 μU/ml day, 56 μU/ml night) both day and night for DLT rats. The insulin level of DLT rats was significantly higher during the day (F (2,15) = 7.9, p <.0046) and the night (F (2,15) = 6.6, p <.009). The intrascapular brown adipose tissue (IBAT) was found to weigh less in DLT rats (159 mg)

Group	N	Cumulative BW changes (g)	Cumulative 72-hr food intakes(g)	12-hr food intakes(g) day	12-hr food intakes(g) night	Glucose (mg%) day	Glucose (mg%) night	Insulin (μu/ml) day	Insulin (μu/ml) night	IBAT(mg)
Control	6	29.8	44.8	5.5	9.5	176	208	23	29	221
Operate	6	29.7	46.5	7.3	7.6	172	167	20	28	215
DLT	6	59.0	61.4	6.8	11.3	172	155	71	56	159

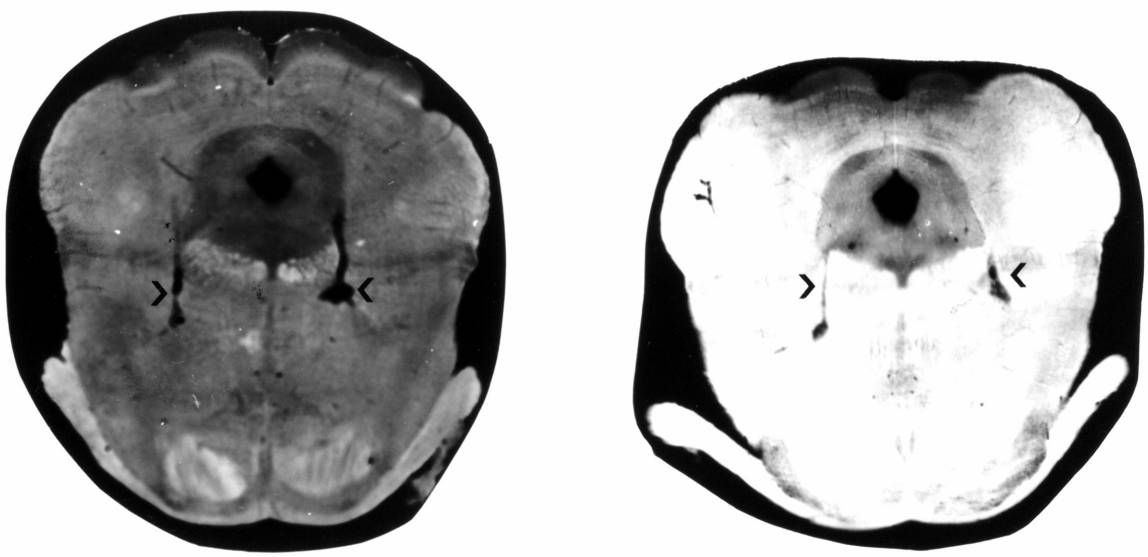
Table V Mean group cumulative changes in body weight (surgery-Day 15 post surgery), cumulative 72-hour food intakes, day and night food intakes, glucose and insulin levels during the day and night and wet weight IBAT at sacrifice, for control, operate-control and DLT rats.

when compared to control rats (221 mg) and operate rats (215 mg) (F (2,15) = 4.4, p <.03).

Figure 2 displays representative photomicrographs of the tegmental lesions of this study. Hyperphagic tegmental rats (n = 6) sustained bilateral, tear-shaped areas of destruction within the dorsolateral aspects of the tegmentum (see left panel Figure 2, p.25). The lesions extended dorsally to the ventrolateral aspects of the central gray, ventrally to the decussation of the superior cerebellar peduncle, and laterally to a vertical plane at the ventrolateral edge of the central gray. The lesions extended in an anterior posterior direction along the length of the midline interpeduncular nucleus. In contrast, lesions that failed to alter body weight or the other dependent measures were dorsal and posterior (n = 3) or misplaced laterally with respect to the midline (n = 3) (see right panel, Figure 2, p. 25).

Figure 2 Left panel. Bilateral lesions (indicated by >) within the dorsolateral tegmentum that induced overeating and obesity, hyperinsulinemia and reduced IBAT weight.

Right panel. Lesion dorsal and posterior to that of the left panel that failed to alter the dependent measures of this study. Magnification is X15.



DISCUSSION

The proper measurement of feeding and of hunger motivation requires an assessment of the stimuli that produce/inhibit feeding. The dorsolateral tegmental rat is unique among the obesity syndromes in that this rat displays diurnal hyperplagia and obesity only when fed palatable diets. An important consideration for the understanding of this syndrome is whether these rats are in fact hungrier than normal. That is, do these lesions disrupt a central satiety mechanism and induce peripheral hyperinsulinemia and consequent feeding? The present study suggests that these factors do not contribute to the overeating observed in DLT rats. In an operant paradigm that is sensitive to hunger motivation, tegmental rats display normal motivation both when fed a normal chow diet or when fed a palatable sucrose diet. It is unlikely that the paradigm itself is insensitive in that we observed consistent changes in response rate and pause times when the animals were treated with amphetamine.

Consideration of the present data suggests that the tegmental syndrome is best conceptualized as a metabolic disorder. Luttmers (1978) observed that DLT rats fed normal amounts of chow get fatter than controls. In the present study it was observed that tegmental rats show hyperinsulinemia and reduced IBAT weight. Both of these factors might account for the pair-feeding data of Luttmers. Insulin causes glucose to enter cells and to be deposited as fat. High insulin levels would therefore enhance lipid formation even were DLT rats to eat normal amounts of food. Brown adipose tissue serves to burn off excess calories. Two animals may eat the same amounts of food and have similar exercise patterns. If brown adipose tissue (BAT) metabolism is different, however, that animal with

low BAT metabolism will gradually become obese. DLT rats have lower BAT weights at sacrifice, a finding consistent with their lower BAT metabolism and enhanced weight gain. Both insulin and BAT factors may contribute to the obesifying action of tegmental lesions, but not to the overeating observed in these rats. Although DLT rats overeat only at night, their insulin levels are high both day and night; thus the former are unlikely to be caused by the latter.

The failure of DLT rats to display increased press rates for a sucrose reward was unexpected. It should be noted, however, that control rats displayed significant reductions in press rate for sucrose, that is, neophobia. Had time permitted, the sucrose manipulation might have been extended for 5-6 more days to allow the animals to overcome their well-know avoidance of novel foods.

In summary, the present study demonstrates that hyperphagic tegmental rats overeat to obesity, display normal operant FR-32 responding for chow pellets, display hyperinsulinemia during the day and night and display reduced IBAT weight. The absence of enhanced FR-32 barpressing is consistent with a metabolic interpretation of this obesity syndromes.

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