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Dietary Obesity in Adult Rats: Similarities to Hypothalamic and Human Obesity Syndromes[1]

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SCLAFANI, A. AND D. SPRINGER. *Dietary obesity in adult rats: similarities to hypothalamic and human obesity syndromes.* PHYSIOL. BEHAV. 17(3) 461–471, 1976. – Normal adult female rats fed a variety of supermarket foods in addition to lab chow rapidly gained weight and became obese compared to rats fed only lab chow. Group housing the animals in an enriched environment did not alter the development of dietary obesity, but housing the rats in activity wheels reduced, although did not prevent, the obesity. The dietary obese rats did not normally defend their excessive weights since they were less willing to eat quinine diets, worked less for food, failed to increase their activity when deprived, and regained their weight at a slower rate following a fast than did controls. The similarity between this behavioral pattern and that displayed by hypothalamic obese rats and overweight humans is discussed.

Dietary obesity Hypothalamic obesity Human obesity Diet palatability Food motivation Environment and feeding Activity Body weight set point

THE search for the causes and cures of overweight has involved the study of various animal obesity syndromes including those produced by neural lesions [43], endocrine disorders [2], genetic anomalies [3], and dietary factors [25]. Most investigations, however, have focused on one syndrome, that produced by hypothalamic damage, while the other forms of experimental obesity have received less attention. In particular, little behavioral work has been done with dietary obesity, although several nutritional experiments have demonstrated that rats become obese when fed highly palatable and caloric diets for long periods [14,25,26 28].

In the one behavioral study reported to date, Maller [22] observed that dietary obese rats display the same aversion to a cellulose adulterated diet as do hypothalamic obese animals. This finding, along with earlier observations reported by Kennedy [16] suggest that excessive weight in otherwise normal rats produces the finickiness and the reduced hunger motivation characteristic of the hypothalamic obese animal [44,45]. The present study was designed to further explore this possibility and to examine other behavioral aspects of the dietary obesity syndrome.

In most previous studies, dietary obesity has been produced by giving rats a high fat diet from the time of weaning [22,25,29]. Early overfeeding, however, may produce changes in adipose tissue different from that associated with hypothalamic obesity [13]. Therefore, in order to more appropriately compare the two syndromes, it was deemed necessary to produce dietary obesity in adult animals. Although feeding high fat diets to adult rats has been reported to result in overweight [25,29], two attempts in our laboratory produced only modest weight gains (Sclafani and Kluge, unpublished obeservations). In the present study, therefore, we attempted to produce dietary obesity by feeding adult female rats an assortment of highly palatable supermarket foods in addition to standard laboratory chow and high fat diets.

EXPERIMENT 1

METHOD

Animals

Twenty adult CFE female rats (Carworth, N.Y.) approximately 120 days of age were used. The animals were individually housed in wire mesh cages measuring 16 x 10 x 7 in. (Wahmann #LC-75/SB) kept in an air conditioned colony room under a 12 hr light–dark cycle.

Apparatus

Ten identical operant chambers (BRS/LVE #143-22) enclosed in sound attenuation boxes (BRS/LVE # 132-02) were used to measure bar pressing performance. Depression of the right bar activated programming equipment in an

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adjacent room and delivered a 45 mg Noyes pellet to a food cup adjacent to the bar.

Procedure

The animals were given ad lib access to Purina chow and tap water for several weeks before being divided into 2 groups of 10 rats each matched for body weight (means = 234 vs 239 g). The control group continued to receive the Purina chow diet, while the experimental group was given, in addition to the chow and water, a high fat diet (33% Crisco fat, 67% Purina powder), sweetened condensed milk (Magnolia brand mixed with water, 1:1), and a variety of other palatable supermarket foods including chocolate chip cookies, salami, cheese, banana, marshmallows, milk chocolate, and peanut butter. At least 7 different foods were available at any one time and the menu was changed frequently except that the Purina pellets, high fat diet, and milk were always available. Body weights were recorded daily, but because of the complexity of the diet, food intake measures were not taken.

After 60 days on the diets described above, a quinine finickiness test was conducted. For 3 days a .1% quinine hydrochloride-Purina powder diet was the only food available to the experimental and control groups. Food intakes were recorded daily. The animals were then returned to their original diets for 10 days before they were food deprived to maintain them at 80% of their ad lib body weights. The rats were trained to bar press for food rewards during daily 1 hr sessions in the operant chambers and then tested for 2 days on each of the following fixed ratio schedules: 1; 4; 8; 16; 32; 64; and 128. At the completion of these tests the experimental and control groups were again allowed ad lib access to their respective diets for 20 days. At the end of this period, 5 of the experimental rats were given only Purina chow to eat for the next 25 days while the remaining rats continued to receive the palatable foods. During this time, 5 control rats were switched to a .4% quinine-Purina diet, while the other 5 control animals continued on the Purina chow.

RESULTS

Figure 1 summarizes the findings of this experiment. When switched to the palatable diet, the experimental group gained at a faster rate than did the controls. By the 10th day, the difference in the weight gains of the two groups was significant (means = 34 vs 13 g, $p<0.05$), and by

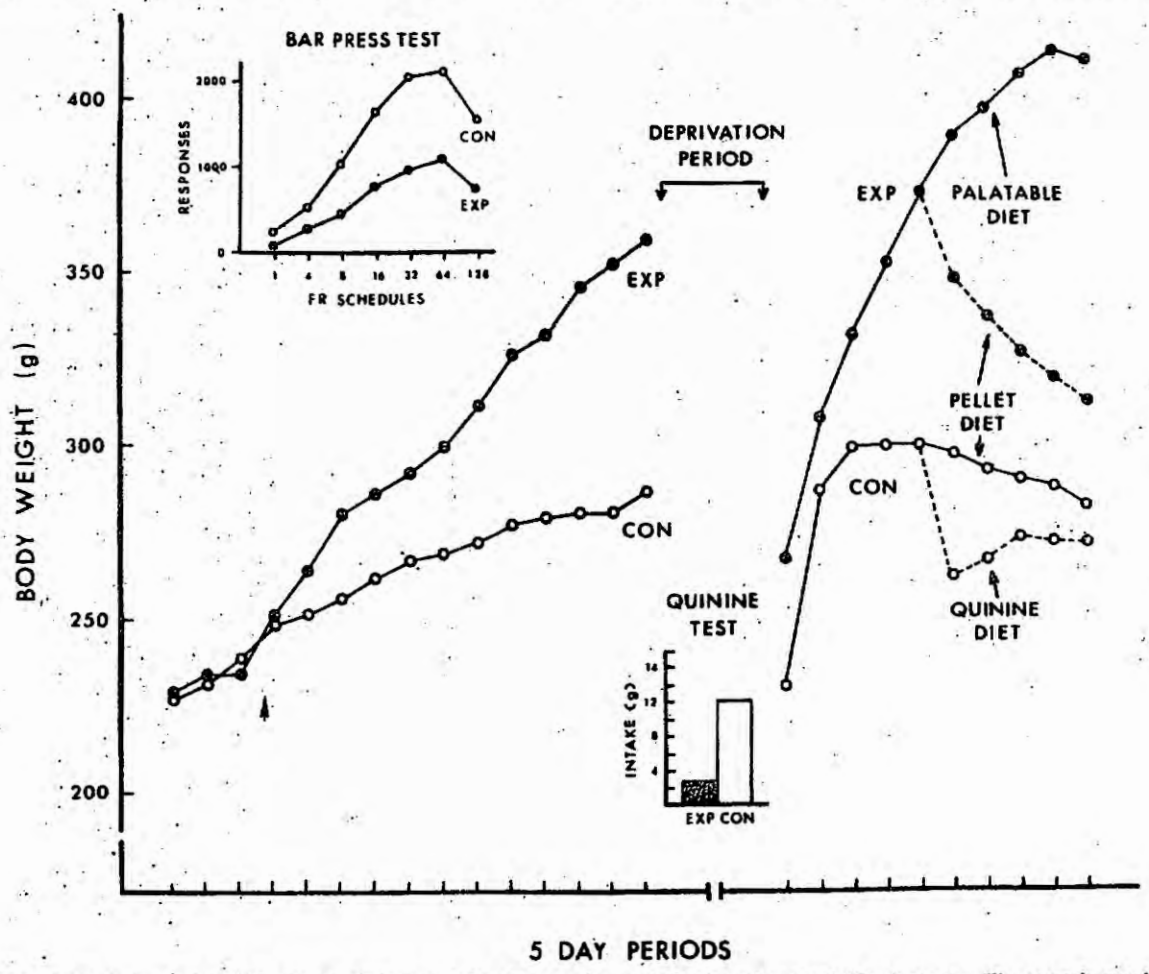

FIG. 1. Body weight as a function of days and diet in experimental (Exp) and control (Con) groups. The experimental group was switched to the palatable diet at the point indicated by the arrow. Graph in upper left shows bar pressing responses of the two groups as a function of the fixed ratio schedule, while graph in lower middle illustrates food intake during the 3 day quinine diet test.

the 60th day, the experimental group had gained 124 g (range = 100–146 g) compared to the control group's gain of 46 g (range = 39–71 g). Although food intake measures were not taken, it was obvious that the experimental group ate little Purina chow and high fat diet, but consumed considerable quantities of the milk diet and other foods.

With only the .1% quinine diet available, the experimental group consumed significantly ($p<0.01$) less food (2.6 vs 12.0 g), and consequently lost more body weight than did the controls (26 vs 9 g, $p<0.01$). By the 10th day after the quinine test the experimental and control groups had regained their pretest body weights. During the subsequent bar pressing test the experimental group bar pressed significantly less ($F(1,18) = 5.21$, $p<0.05$) than did the control group. As indicated in Fig. 1, the difference between the two groups in mean bar pressing rates increased as the fixed ration schedule became more demanding.

At the end of the bar pressing test, when the animals were once again returned to their original diets, both the experimental and control animals rapidly regained and then surpassed their pretest body weights (Fig. 1). The experimental rats given the Purina chow to eat rapidly lost weight (mean =60 g) during the next 25 days, while the 5 animals maintained on the palatable diet continued to gain weight (mean = 30 g). The control rats switched to the .4% quinine diet also lost weight and maintained their body weights below that of the controls on the Purina chow. The control animals maintained on the chow throughout showed a slight weight loss which was due to 2 of the 5 rats in the subgroup who had become ill and lost 30 g in weight. Because of a spreading illness in the colony, the experiment was formally terminated at this time. The weight loss displayed by the experimental-pellet and control-quinine subgroups was not due to illness, however, as these rats rapidly regained their weights when their original diets were returned.

DISCUSSION

Feeding neurologically intact adult rats a variety of highly palatable supermarket foods was found to be a particularly effective way of producing dietary obesity. The 53% weight increase displayed by the experimental group after 60 days on the diet is greater than that observed in previous studies using adult rats and high fat diets ([25,29], Sclafani and Kluge, unpublished observations). The degree of relative obesity obtained in this experiment would have been even greater if the control group was fed a diet less palatable than the Purina chow throughout the study. The rats given the .4% quinine diet, for example, maintained lower body weights than did the Purina fed subgroup and this dietary leaness has been observed in previous experiments [8,40].

The supermarket foods differed from the Purina chow not only in palatability, but in nutritional content as well. In particular, most of the foods were lower in protein and vitamin content, and higher in carbohydrate content than is Purina chow [5]. It is unlikely, though, that the obesity displayed by the experimental rats resulted from a nutritional deficiency. The animals were not forced to eat the supermarket foods since they always had Purina chow available. The fact that they ignored the chow and overate the supermarket foods can only be attributed to the palatability of these foods. Furthermore, supplementing the milk diet, which all rats avidly consumed, with vitamins and minerals to make it a nutritionally adequate diet does not reduce the weight gain displayed by the experimental animals (see Experiment 3). Therefore, although the post-ingestive effects of the diet may be of importance to some aspects of the syndrome, it it the orosensory properties of the diet that appear to be primarily responsible for the development of dietary obesity.

While the experimental animals overate and became obese on the palatable diet, they did not defend their excessive body weights when the feeding conditions were less favorable. That is, the dietary obese rats ate less quinine diet and worked less for food rewards when deprived than did the control animals. These findings are consistent with Maller's [22] previous report that dietary obese rats are finicky to cellulose adulteration of their diet. Furthermore, the experimental animals did not maintain their obesity when given only Purina chow but rather reduced their weights to control levels, which confirms earlier findings [29]. On the other hand, the experimental rats rapidly recovered their obese weights when returned to their supermarket diets following the deprivation period. In fact, their rate of weight gain following the deprivation was greater than that originally displayed when the supermarket diet was first made available (see Fig. 1). This suggests that prior obesity influences subsequent weight gain, although other data indicate that age is an important factor determining the rate of weight gain in rats fed supermarket diets (Sclafani and Gorman, unpublished findings.)

EXPERIMENT 2

In the first experiment the dietary obese rats ate less quinine adulterated diet and bar pressed less for food than did controls which suggests that excessive body weight in neurologically intact rats suppresses hunger motivation. Another interpretation of these results is possible, however. The experimental and control groups were not maintained on the same diet prior to the motivational tests and thus the difference between their diet conditions, rather than their body weights, may have been responsible for the reduced appetitive motivation displayed by the dietary obese subjects. That is, the obese rats may have eaten less quinine diet than did the controls because the sensory contrast between the supermarket foods and the quinine diet was much greater than that between the quinine diet and Purina chow; likewise, the controls may have bar pressed more than did the obese rats for the Noyes pellet rewards because this food was more palatable than the Purina chow, but less palatable than the supermarket diet. Therefore, in the present experiment the food motivation of dietary obese and normal rats was examined using paradigms in which the contrast between the pretest and test diets was the same for both groups.

METHOD

Animals

Seven dietary obese female rats weighing between 437 and 517 g (mean = 490 g) and 6 control female rats weighing between 292 and 322 g (mean = 311 g) were used. These animals were originally used in the study reported here as Experiment 3. Between the end of that experiment and the beginning of the present one, the animals were

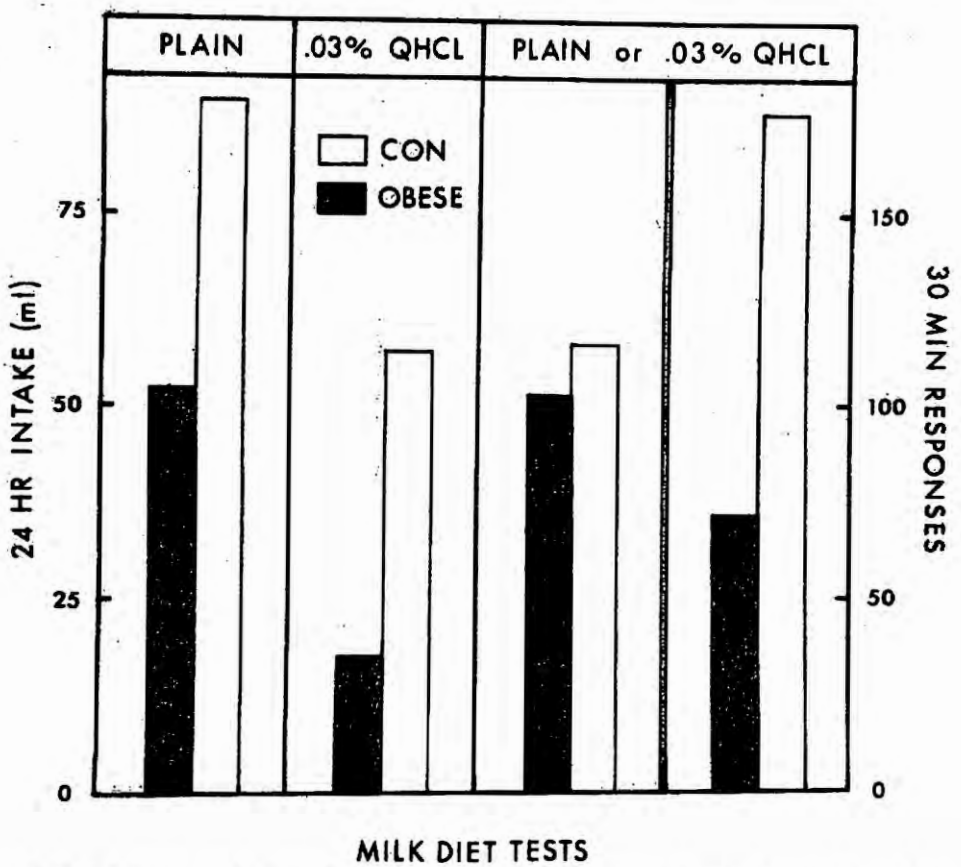

FIG. 2. Milk intake and bar pressing responses (right box) of dietary obese and control female rats. Right half of graph represents tests in which obese rats were given the plain milk diet and the control rats were given the 0.03% quinine milk diet.

housed in standard size cages and fed either the supermarket (obese group) or Purina chow (control group) diet.

A supplementary experiment was conducted with 21 CFE male rats weighing approximately 420 g which were part of a study to be reported elsewhere. The male animals were housed in standard size wire mesh cages and fed a supermarket diet similar to that described in Experiment 3.

Procedure

At the start of the experiment, the dietary obese and control female rats were taken off their respective diets and given a sweetened condensed milk diet (described in Experiment 3) as their only food. Baseline food intake and body weights were recorded daily for one week before the milk diet was adulterated with quinine hydrochloride to .03% and given to all animals for 3 days. The dietary obese rats were then returned to the plain milk diet, while the control rats continued on the quinine milk for 3 additional days. The animals were next food deprived to 85% of the body weights they displayed on the plain milk diet (obese rats) or the quinine milk diet (control rats). Using the operant chambers described in Experiment 1, the rats were trained to barpress for food rewards. Depression of the right bar activated a dipper mechanism which provided the controls with .1 ml of the quinine milk, and the dietary obese rats with .1 ml of the plain milk diet. The rats were given daily 30 min. tests on a continuous reinforcement schedule for 8 days. Statistical analysis was based on the *mean response scores* for the last three days.

The male rats were given either unlimited (n = 11) or limited (n = 10) amounts of the supermarket diet in addition to Purina chow. In the latter case, the amount of supermarket food was restricted such that the rats gained weight at the same rate that they previously displayed when fed only Purina chow (approximately 2.2 g/day). The restricted group was not food deprived since Purina chow and tap water were always available in sufficient amounts. After 20 days all rats were given a .1% quinine-Purina chow mash as their only food for 24 hours and food intakes were recorded to the nearest 0.1 g.

RESULTS AND DISCUSSION

The data from the female rats are depicted in Fig. 2. The control rats consumed significantly ($p<0.01$) more of the plain milk diet than did the dietary obese rats. This finding is not surprising and presumably, if maintained on this diet long enough, the food intake and body weight of the two groups would have converged. Quinine adulteration reduced the milk intake of both groups but the dietary obese rats displayed a significantly ($p<0.01$) greater reduction in intake (−70%) than did the controls (−35%). When the obese rats were returned to the milk diet, and the controls continued on the quinine milk, the intake of the two groups became equal. Thus, the feeding suppressive effect of quinine adulteration in the control rats apparently equalled the suppressive effect of excessive body weight in the obese rats. Despite the similarity in ad lib food intakes, however, when deprived and required to barpress for food

the dietary obese rats worked less ($p<0.01$) for their plain milk diet than did the controls for their quinine diet.

The male rats given unlimited amounts of supermarket food gained more ($p<0.05$) weight during the 20 day period than did the rats given limited amounts (73 vs 42 g). The former group, therefore, was obese compared to the latter group just prior to the quinine test (BW = 495 vs 462 g, $p<0.05$). When given the quinine adultered diet the obese male rats consumed significantly ($p<0.05$) less than did the normal weight males (3.3 vs 7.1 g). Comparison of the data obtained with the male and female subjects reveals that differences in the pre-quinine test food intake cannot account for the increased quinine aversion displayed by the dietary obese rats. That is, while the dietary obese males were eating more food than the non-obese males just prior to the quinine test, the nonobese females were eating more than the obese females.

These findings confirm the results of Experiment 1 that dietary obese rats are less willing to eat a bitter diet or work for food when deprived than are normal weight subjects. In the present experiment, though, the results cannot be attributed to a sensory contrast effect since the change, or lack of it, in diet palatability from pretest to test conditions was identical for the obese and control groups. Thus, it is the excessive body weight of the dietary obese rats which is responsible for their quinine finickiness and reduced barpressing performance. The fact that the increased aversion to the quinine diet was associated with reduced barpressing behavior suggests that the finickiness was the result of an obesity-produced motivational suppression rather than from an alteration in taste sensitivity *per se*. This interpretation is supported by the finding of Maller [22] that dietary obese rats display normal responsivity to sapid solutions.

EXPERIMENT 3

Experiment 1 revealed that rats fed palatable supermarket foods overeat and become obese compared to animals maintained on standard laboratory chow. As is most feeding studies, the animals were housed in cages which allowed for little physical activity and minimal sensory and social stimulation. In fact, other than grooming, sleeping, sniffing, and walking around the cage, there was little for the rats to do but eat. It is well known that lack of exercise is associated with overweight in animals and humans [4, 6, 10] and social and sensory isolation also appears to lead to overeating and obesity [23, 24, 26, 42]. Thus, the dietary obesity observed in Experiment 1 may, at least in part, be an artifact of the relatively sterile laboratory housing conditions used. This possibility was examined in Experiment 3 by comparing the body weight gains displayed by rats maintained in housing conditions which differed in the degree of physical activity and social stimulation available. This experiment also further examined the responses of dietary obese and control rats to food deprivation.

METHOD

Animals

Forty-eight female CFE rats purchased from Carworth, N.Y. were used. The rats were maintained in an air-conditioned colony room under a 12 hr light–dark cycle.

Housing Conditions

The animals were maintained in 1 of 3 housing conditions: standard size wire mesh cages (7 x 10 x 7 in., Wahmann, LC-75/SA); small wire mesh cages attached to activity wheels (Wahmann, LC-34); or in large complex environments (18 x 19 x 27 in.). The complex environments consisted of 3 large wire mesh cages (Wahmann, LC-27) stacked on top of each other (see Fig. 3). The doors of the cages were replaced with a single clear Plexiglas door containing wire mesh ramps which connected the 3 levels. The upper two levels of the environment had perforated metal floors, while the bottom level contained a metal pan filled with wooden shavings. Wooden, metal, and at times, plastic objects were located in the upper two levels.

Procedure

Initially, all rats were individually housed in the standard size cages and given Purina chow and tap water ad lib. Baseline body weights were recorded for several days before the animals were transferred to the 3 housing conditions. Sixteen rats were individually housed in the activity wheels (A = active condition), 16 rats were housed in groups of 8 in the two complex environments (E = enriched condition), and 16 rats remained individually housed in the standard cages (I = isolated condition). All cages were kept in the same colony room. Ten days later, half of the animals in each of the 3 housing conditions were given an assortment of supermarket foods in addition to the Purina chow (S = supermarket diet; groups = E-S, A-S, and I-S), while the other half of the animals continued to receive only the Purina chow (P = Purina chow diet; groups = E-P, A-P, and I-P). The supermarket foods included marshmallows, milk chocolate, a cheese flavored snack, cream-filled chocolate cookies, salami, banana, and sweetened condensed milk. The condensed milk was mixed with two parts water and supplemented with vitamins and minerals to make a nutritionally adequate diet [19]. Fresh food was given to all rats each day. Body weights were recorded every 5 days, except where noted otherwise, and wheel revolutions were recorded daily.

On Day 65 of the experiment, the wheel housed rats were returned to the standard laboratory cages (thus becoming the AI-P and AI-S groups) while the rats in the standard cages were transferred to the activity wheels (thus becoming the IA-P and IA-S groups). Starting on Day 95 all animals were food deprived for 4 days and were then returned to ad lib access to their respective diets for the following 20 days. During the deprivation and post-deprivation periods, body weights were recorded daily.

RESULTS

The effect of diet and housing conditions on body weight are summarized in Table 1 and Fig. 4. Table 1 also includes the final group sizes since several animals became sick during the experiment and were discarded.

Analysis of variance revealed that both diet and housing conditions significantly influenced ($p<0.01$) the body weight gains displayed by the rats on Day 65. Irrespective of housing conditions, the animals fed the supermarket diet gained more weight than did the Purina chow fed rats, and irrespective of diet, the rats in the activity wheels gained less weight than did the rats in the isolated or enriched environments. The latter two housing conditions did not

FIG. 3. Complex environment used to house E-P (illustrated here) and I-P groups.

differentially affect body weight with the E-S and I-S groups both gaining about 98 g or 228% more than the E-P and I-P groups. The A-S group, on the other hand, gained only 62 g or 153% more than did the A-P group during the 65 days on the supermarket diet.

The suppressive effect of wheel running on body weight was not the same for the supermarket diet-fed and Purina chow-fed rats. Both groups initially lost weight during the first 10 days in the activity wheels, but whereas the A-P rats then gained at the same rate as did the E-P and I-P groups during the next 65 days, the A-S rats gained at a slower rate than did the E-S and I-S groups. (Between Days 50 and 65, the rate of weight gains for the three supermarket diet groups did not differ significantly). Thus, by Day 65, the body weights of the A-S group was about 60 g less than that of the I-S and E-S groups, while the body weight of the A-P group was only about 20 g less than that of the I-P and E-P groups (Table 1). As indicated above,

TABLE I

MEAN BODY WEIGHTS (+ RANGES) OF GROUPS FED SUPERMARKET OR PURINA CHOW DIETS AND HOUSED IN ENRICHED, ISOLATED, OR ACTIVE ENVIRONMENTS

	N	Day −10	Day 0	Day 65	Day 95
E-P	8	239 (225-248)	251 (241-258)	296 (278-313)	302 (282-322)
E-S	6	241 (228-257)	249 (235-265)	390 (324-441)	427 (352-467)
I-P (IA-P)	6	236 (220-263)	251 (238-269)	294 (278-320)	287 (278-294)
I-S (IA-S)	8	238 (224-263)	248 (231-272)	389 (328-505)	382 (316-524)
A-P (AI-P	7	237 (223-247)	233 (222-252)	274 (258-292)	296 (281-322)
A-S (AI-S)	8	230 (216-247)	228 (213-253)	331 (278-370)	412 (340-485)

Note: Day −10 = last day before transfer to different housing conditions.
Day 0 = last day before supermarket diet presented to E-S, I-S, and A-S groups.
Day 65 = last day before I-P and I-S groups transferred to active, and A-P and A-S groups transferred to isolated conditions.
Day 95 = last day before 4 day fast.

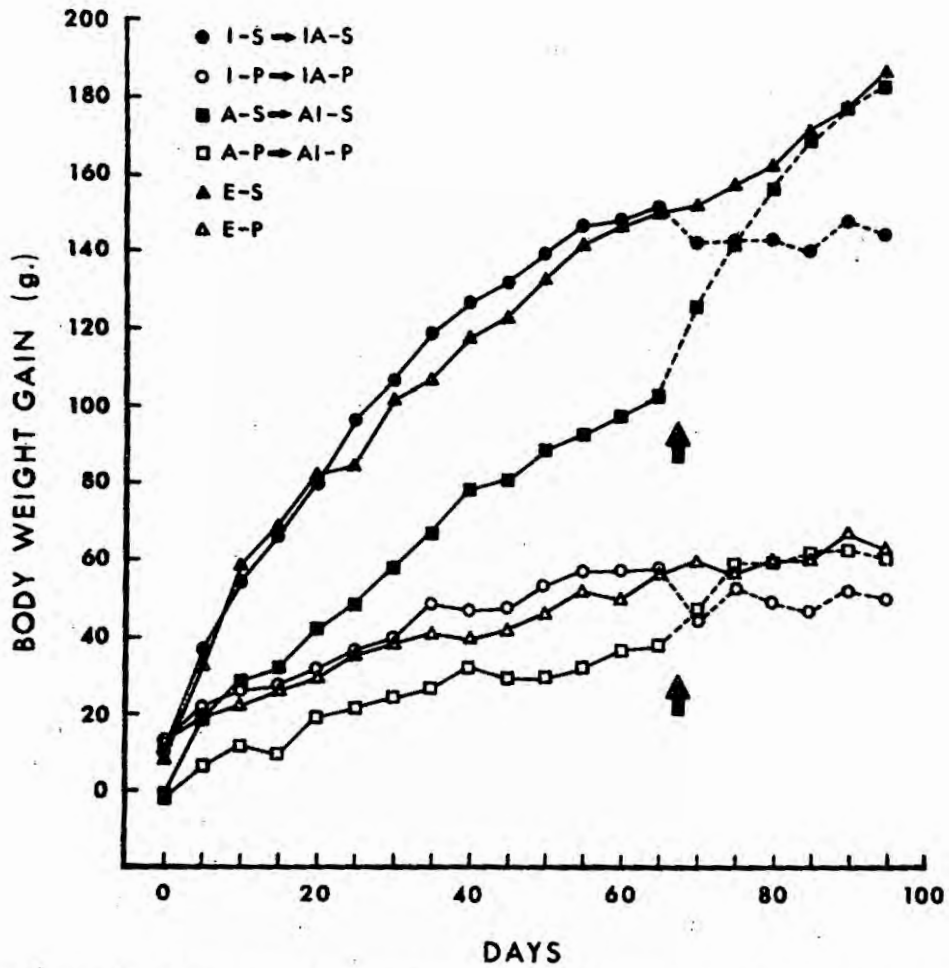

FIG. 4. Mean body weight gains of groups fed supermarket (S) or Purina chow (P) diets and housed in enriched (E), isolated (I), or activity (A) cages. Weight gains based on body weights of rats just prior to their transfer to the different housing conditions (Day-10). On Day 0, E-S, I-S, and A-S groups were given supermarket diet. On Day 65, indicated by arrow, I-S and I-P groups were placed in the activity wheels becoming groups IA-S and IA-P, while A-S and A-P groups were returned to isolated cages becoming AI-S and AI-P groups.

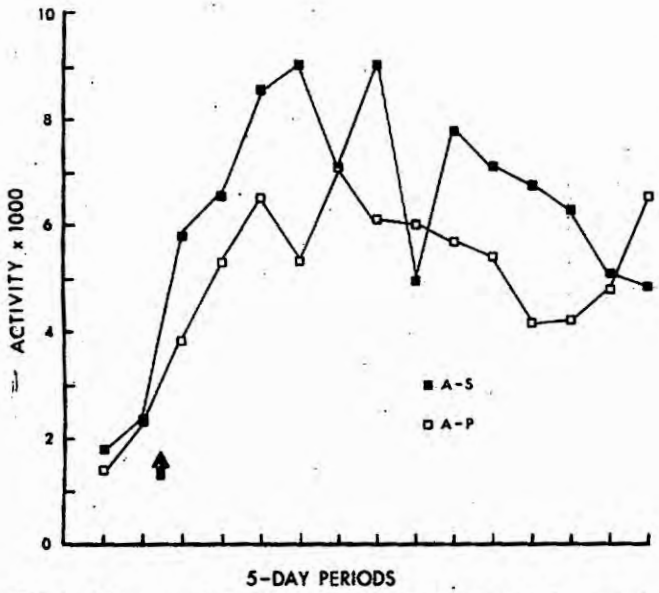

FIG. 5. Mean running activity (revolutions/day) of A-S and A-P groups during 5 day periods. At point indicated by arrow (Day 0) A-S group was given supermarket diet while A-P group remained on Purina chow diet.

though, the A-S rats gained more weight than did the A-P rats. The A-S rats also tended to be more active in the wheels than the A-P rats although this difference failed to be significant (Fig. 5).

On Day 65 of the experiment when the rats in the activity wheels were transferred to the stationary cages, they increased their rate of weight gain such that their body weights rapidly reached the levels maintained by the enriched environment subjects (Fig. 4). The increase in weight during Days 65 to 95 was significantly ($p<0.01$) greater for the AI-S group than for the AI-P group (80 vs 22 g). On the other hand, the rats in the stationary cages initially lost weight when transferred to the activity wheels on Day 65 and maintained their weights at this reduced level during the 30 day period. Furthermore, the weight loss displayed by the IA-S and IA-P groups were similar (Table 1, Fig. 4). The wheel running activity of the two groups during Days 65 to 95 also did not significantly differ, although the IA-S rats tended to be less active than the IA-P rats (992 vs 1442 revolutions/day).

Just prior to the 4 day fast, the supermarket diet groups weighed from 95 to 125 g more than the Purina chow groups (Day 95, Table 1). During the fast, the weight losses of the E-S (53 g) and AI-S (59 g) groups were similar to those of the E-P (47 g) and AI-P (62 g) groups, while the IA-P rats lost significantly ($p<0.01$) more weight than did the IA-S rats (73 vs 55 g). When food was returned following the fast, however, the Purina chow fed rats regained their weights at a significantly ($p<0.01$) faster rate than did the rats fed the supermarket foods. During the first 10 postfast days, for example, the IA-P, E-P, and AI-P groups gained 72, 53, and 65 g respectively which represents 99 to 113% of their weight loss while the IA-S, E-S, and AI-S groups gained 41, 41, and 37 g respectively, or 63 to 77% of their weight loss. By the 20th day after the fast, all of the supermarket diet fed groups had regained their pre-fast body weight.

The daily activity scores of the IA-P and IA-S groups during the 4 day prefast and fast periods are presented in Fig. 6. Prior to deprivation, the IA-S rats were slightly, but not significantly, less active than the IA-S animals. However, when food deprived the IA-P group significantly ($p<0.05$) increased its running activity while the IA-S group displayed no change. Thus, the activity of IA-S rats during the deprivation period was significantly ($p<0.05$) less than that of the IA-P rats. (The decline in activity of the IA-P group on day 4 of deprivation resulted from the death of the 2 most active animals. No other animals died during the fast.) The increased activity of the IA-P rats during the fast accounts for their greater weight loss relative to the IA-S group.

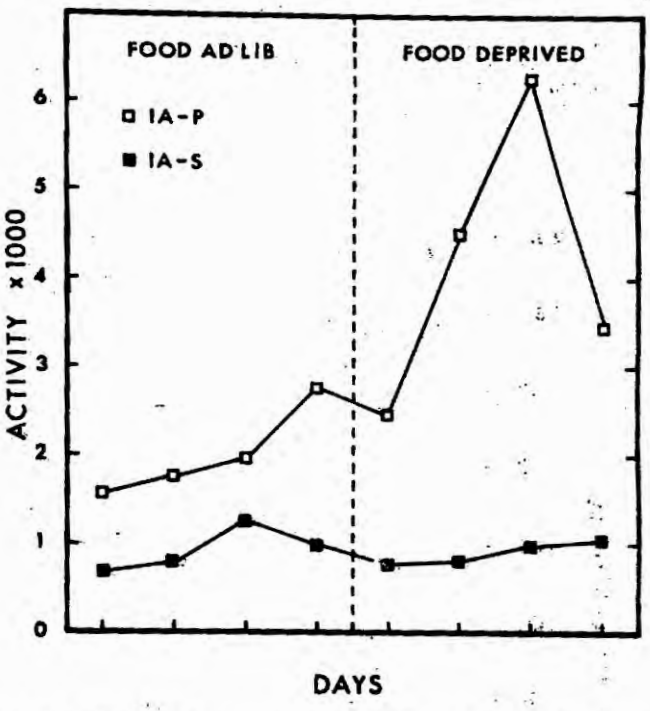

FIG. 6. Mean activity (revolutions/day) of IA-S and IA-P groups just prior to and during 4 day food deprivation period.

DISCUSSION

These findings, in addition to confirming the results of Experiment 1, indicate that dietary obesity is not an artifact of restricted laboratory living conditions. That is, the rats housed in the enriched environment gained as much weight on the supermarket diet as did those isolated in the small cages. It is possible of course that a larger and more complex environment than the one used here would have limited the development of obesity. Nevertheless, the similarity between the enriched and isolated groups suggests that environmental complexity is not a major factor in the regulation of body weight in rats.

In contrast to the enriched housing condition, access to activity wheels significantly reduced, but did not prevent, the development of dietary obesity. Wheel running also lowered the body weight of the Purina chow fed rats which is consistent with many previous studies showing that active animals maintain lower body weights than do sedentary ones [6,24]. It is not clear, however, whether the rats living in the enriched environments should be considered inactive.

The animals were certainly more active than the isolated rats and were often observed running, climbing, jumping, and playing in the large cages. The failure of the enriched group to display reduced body weight gains, like the active group, may be because their total activity was less than that of the wheel housed animals. It is possible, though, that there are qualitative differences between the activity in a large stationary environment and running in rotating wheels which are of significance to body weight regulation.

The mechanism by which wheel running lowers body weight is not certain. Increased energy expenditure per se is not an adequate explanation since the rats presumably could maintain their weights by increasing their food intake. Previous findings indicate, in fact, that animals decrease their food consumption when running in wheels or treadmills [6, 24, 31]. This suggests that running activity is associated with the maintainence of a reduced target body weight [6] and the present findings are consistent with this interpretation. As in previous studies [6], the Purina chow fed rats showed an initial drop in body weight when transferred to the activity wheels but then began to gain at a rate which paralleled that displayed by the E-P and I-P groups. The A–S rats also eventually gained weight at the same rate as did the E-S and I-S groups but only after 50 days on the supermarket diet. Thus, a comparison of the weight gains displayed by the two active groups (A-S and A-P) relative to the nonactive groups (E-P, I-P, E-S, and I-S) indicates that wheel running produces a greater reduction in the target weight of obese rats than normal rats.

This suppression in body weight produced by wheel running was not a permanent effect as the active group rapidly increased their weight to the higher target levels of the enriched animals when removed from the activity wheels. This effect was particularly evident in the AI-S rats who gained nearly 4 times as much weight as did the AI-P group in the month following their removal from the wheels. The reversibility of the target weight is further indicated by the weight loss of the IA-P and IA-S groups when given access to the running wheels on Day 65. The IA-S rats, however, did not reduce their weights to the level previously displayed by the A-S group. Rather, they showed the same decrease in weight as that displayed by the IA-P animals. However, the IA-S rats maintained their body weights at this reduced level during days 65 to 95, while the other supermarket fed rats continued to gain weight. Thus, access to the activity wheel produced a greater decrease in potential weight gain for the IA-S group than for the IA-P group which replicates the effect initially observed with the A-S and A-P groups.

The supermarket diet, while increasing body weight, did not significantly alter the ad lib activity levels of the A-S and IA-S groups relative to the Purina fed groups. This is an interesting finding since most other conditions which increase body weight, including refeeding after a fast, VMH damage, and hormonal changes are associated with decreased activity levels [17]. Under food deprived conditions however, a significant difference emerged between the wheel running behavior of the IA-S and IA-P groups. That is, while the IA-P rats showed the typical deprivation-induced increase in running activity, the IA-S rats failed to increase their running during the 4 day fast. The IA-S rats were significantly heavier than the IA-P rats just prior to the deprivation test which suggests that excessive body weight prevents the deprivation-induced increase in activity. This interpretation is supported by our recent findings that dietary obese rats do eventually increase their running activity during a fast as their weight falls to control levels [37]. The failure of the IA-S rats to display the normal deprivation-induced activity increase is consistent with the suggestion made in Experiments 1 and 2 that dietary obese rats have reduced hunger motivation.

One final bit of evidence that dietary obese rats are not as responsive to food deprivation as are non-obese animals is the finding that the supermarket diet fed groups regained their prefast weights at a slower rate than did the Purina fed groups. This finding contrasts with the rapid recovery of predeprivation weights by the experimental animals in Experiment 1. However, in the first experiment the dietary obese rats had lost more weight than did the controls. In fact, their weights fell below the ad lib level of the Purina fed controls so that the experimental rats were no longer overweight at the end of the deprivation period. On the other hand, in the present experiment, the experimental rats lost either the same amount or less weight than did the controls during the fast and were still overweight compared to the ad lib weights of the Purina chow fed rats.

GENERAL DISCUSSION

"The laboratory rat lives in an artificially constant environment, and is fed artifically constant foods; but it is easy enough to render a rat obese if it is taken home, treated as a family pet, and offered unlimited amounts of food which it likes" ([10], p. 2).

The present results demonstrate that it is not even necessary to bring the rat home since obesity can easily be produced in the laboratory by giving normal adult rats ad lib access to an assortment of highly palatable foods. Rats are not equally susceptible, however, to the weight promoting effects of the tasty diet. As indicated in Fig. 7, which combines the data of Experiments 1 and 3, there was considerable variability in the weight gains displayed by the supermarket diet fed animals, although all did outgain the animals fed Purina chow only. It should also be noted that the present study does not set the upper limit of dietary obesity, and even greater weight gains may be possible with more palatable foods and longer periods on the diet. The supermarket foods used here obviously do not represent the ultimate in human cuisine but were chosen with cost, convenience, and spoilage factors in mind.

The findings of this study question the view that rats eat for calories and are precise regulators of their body weight [1, 15, 41]. This position has been based, in part, on the results of many previous studies in which diet palatability was altered by adding adulterants to laboratory chow. This technique, however, allows for only limited improvements in palatability. Furthermore, variety in the diet is thought to be an especially important factor determining food intake. LeMagnen [20], for example, has reported that rats eat more at a given meal when presented with 4 differently flavored foods than when presented with a single food and he concluded, therefore, that satiety is sensory-specific. Consistent with this view, videotape analysis of the feeding behavior of rats offered the supermarket diet revealed that they sample more than one food item during most of their meals. Furthermore, their number of meals appeared normal and thus, their overeating was accomplished by increases in meal size (Sclafani, unpublished observations).

In addition to demonstrating that dietary obesity can be reliably produced in adult rats and is not an artifact of

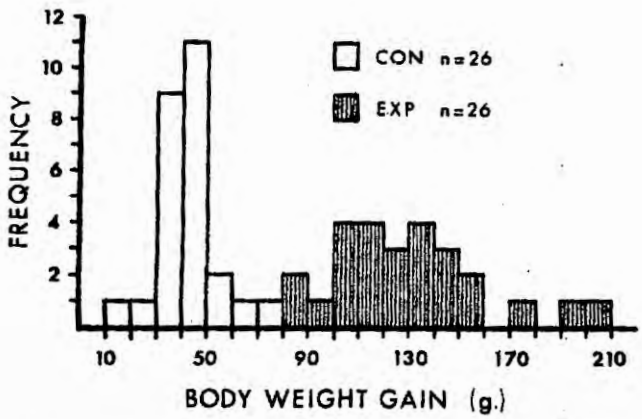

FIG. 7. Frequency histogram of weight gains displayed by experimental and control rats from Experiments 1 and 3 after 60 days on the supermarket or Purina chow diets. Subjects from Experiment 3 include the original rats in groups E-S, I-S, E-P, and I-P.

restricted living conditions, the present study further documents the similarity, first noted by Maller [22], between the dietary obesity and hypothalamic obesity syndromes. Like hypothalamic obese animals [44,45], the dietary obese rats were finicky to quinine adulterated diets, worked less for food, failed to increase their activity when deprived, and regained their weights at a reduced rate following a short fast. The commonality between the two syndromes is further evidenced by recent findings which indicate that obesity, rather than neural damage *per se*, is responsible for the quinine finickiness, suppressed hunger drive, and reduced appetitive and locomotor responses to deprivation displayed by the hypothalamic obese animal [8, 9, 21, 30, 38, 39, 40]. Also, the obesity-produced finickiness in both dietary obese [22] and hypothalamic obese [27,40] rats appears to result from motivational rather than taste sensitivity changes.

Obesity, however, is not a sufficient condition to produce these deficits in body weight defense. That is, other syndromes in the rat, such as genetic obesity [7,12] and ovarian obesity ([11], Gale and Sclafani, unpublished observation) are not associated with finickiness and reduced hunger drive. Sclafani [38] has suggested, therefore, that the critical factor is not the obesity alone, but the relationship between the animal's body weight and its set point weight. According to this view, if the obesity is accompanied by a set point elevation, then body weight defense is normal, but if obesity occurs despite an unchanged set point, then body weight defense is impaired. In the case of the hypothalamic syndrome, it is proposed that the set point is not altered but rather the appetite inhibitory effect of excessive body weight is thought to be reduced. This allows the animal to overeat even on marginally palatable laboratory diets and as a result its body weight exceeds the set point (see [38, 39, 40] for further explanation and supporting evidence). A somewhat similar interpretation can be applied to the dietary obesity syndrome. In this case, appetite is increased not by hypothalamic damage, but by the presence of the highly palatable foods. Therefore, although the etiologies of the dietary obesity and hypothalamic obesity syndromes differ, the net effect -- the supra-set point body weights -- may be the same which could account for the similarity between the two syndromes.

Behavioral similarities have also been reported to exist between hypothalamic obese rats and overweight humans. For example, obese people have been found to be finicky eaters, less willing to work for food, and less responsive to food deprivation than are normal weight individuals [27, 33, 34]. The present results indicate that these symptoms are also characteristic of dietary obese rats. In view of the fact that human adiposity is only rarely associated with hypothalamic damage [32], but is associated with the availability of palatable foods and sedentary activity levels, dietary obesity may be a more appropriate model for human obesity than is the hypothalamic syndrome.

Factors other than diet, of course, are also involved in determining body weight, and not all humans exposed to favorable dietary conditions become obese. Strain, sex, age, and early experience factors all have been demonstrated to be of importance in the development of dietary obesity ([25, 29, 34, 35, 37], Sclafani and Gorman, unpublished observations). Even in the present study, which utilized controlled laboratory conditions and a single strain of rats, there was considerable variability in the weight gains displayed by the supermarket diet fed animals. Furthermore, as mentioned in the introduction, several forms of obesity exist in animals and humans which involve specific physiological, hormonal, or genetic disorders [23], and these obesities may be differentially influenced by diet palatability [7, 11, 12, 38].

In summary, while it is clear that obesity has multiple etiologies, the present study demonstrates that in the normal rat, free access to palatable foods is a sufficient condition to promote excessive weight gain and this dietary obesity is associated with reduced body weight defense. The study of rats fed bland and monotonous laboratory diets, therefore, may give a misleading picture of their weight regulatory abilities, and furthermore, may be of less relevance to the understanding of human feeding behavior than is the study of rats given a varied and tasty diet.

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