Influence of Diet Palatability on the Meal Taking Behavior of Hypothalamic Hyperphagic and Normal Rats

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SCLAFANI, A. AND C. N. BERNER. Influence of diet palatability on the meal taking behavior of hypothalamic hyperphagic and normal rats. PHYSIOL. BEHAV. 16(3) 355-363, 1976. - Female rats made hyperphagic by parasagittal hypothalamic knife cuts overate a palatable milk diet by greatly increasing their meal size, while meal frequency was only slightly increased. Reducing the palatability of the diet by quinine or salt adulteration decreased to normal levels the total intake and meal size, but not meal frequency of hyperphagic rats. The disruption in the diurnal feeding pattern displayed by the hyperphagic rats was relatively unaffected by diet palatability changes. The results are discussed in terms of the short and long term regulatory deficits thought to be responsible for the hypothalamic hyperphagia syndrome.

Numerous studies have demonstrated that the overeating produced by ventromedial hypothalamic damage in the rat is usually accomplished by an increase in meal size rather than in meal frequency [1, 5, 33, 35]. This finding has often been interpreted as evidence for a short term satiety deficit in the hypothalamic hyperphagic animal as originally proposed by Miller et al. [21]. Several investigators, however, have failed to obtain direct evidence that VMH lesions impair short term satiety [20, 23, 30]. Furthermore, if lesioned animals are prevented from eating large meals they continue to overeat by increasing their meal frequency [5, 34].

The enlarged meals of hyperphagic animals, therefore, may not be due to reduced satiety, but rather may be caused by some other factor which influences meal size. For example, it is known that hunger induced by food deprivation increases the meal size of normal rats when they are allowed to refeed [16, 18]. Although hyperphagic rats have usually been considered not to have increased hunger, some recent reports question this conclusion [12, 22]. Changes in appetite produced by alterations in diet palatability also influence meal size. Normal rats take larger meals when diet palatability is increased, and take smaller meals when palatability is decreased or eliminated by intragastric feeding [15, 16, 18, 34]. Previous work in our laboratory, in fact, suggests that hypothalamic hyperphagia is associated with increased appetite rather than increased hunger or decreased satiety [25, 26, 27, 28].

The present experiment, in line with the enhanced appetite hypothesis [27], examined the influence of diet palatability on the meal taking behavior of hyperphagic and normal rats. It is well documented that palatability is an important factor determining the food intake of hyperphagic animals [7, 32], but its effects on meal size and distribution have not been previously studied. If hyperphagic rats eat more than normal because their appetite for good tasting foods is enhanced, then decreasing the palatability, i.e., appetizing effect of the diet, may decrease their meal size as well as their total intake towards normal levels.

EXPERIMENT 1

METHOD

Animals

Eight adult female CFE rats (Carworth, N.Y.) were used. The animals weighed 200–225 g at the start of the experiment and were housed in an isolated room with a 12 hr light—dark cycle.

Surgery

Surgery was performed under Equithesin anesthesia (2.5 cc/kg BW). Bilateral knife cuts between the ventromedial and lateral hypothalamus (VL cuts) were made in 4 rats (VL group) while the remaining 4 animals received sham

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operations. One of the sham operated controls died shortly after surgery. The knife cuts were made in the parasagittal plane, 0.8 mm from the midline using the encephalotomy procedure previously described by Sclafani [25,29]. The cuts extended from the anterior to posterior hypothalamus and were situated just medial to the fornix. Further description and photomicrographs of similar type cuts are provided by Sclafani et al. [29].

Diets
The animals were initially maintained on Purina Chow. To measure meal patterns a liquid diet was used which consisted of 300 ml of sweetened condensed milk (Borden's Magnolia brand) mixed with 600 ml of tap water. Added to each 900 ml of diet were 0.3 ml of vitamin mixture (Polyvisol, Mead Johnson), 24.12 mg ferrous gluconate, 2.4 mg cupric sulfate, 3 mg manganese acetate, and a few drops of Formalin to retard spoilage. This diet is a more dilute form of that described by Kissileff [14]. Quinine flavored diets were prepared by adding the appropriate amount of quinine hydrochloride (QHCl) to 1000 ml of diet. For example, the 0.03% QHCl diet contained 0.3 g. of QHCl in 1000 ml of milk diet. Between the 2 meal pattern measurement periods the rats were maintained on a high fat diet consisting of 33% Crisco fat and 67% Purina powder. Tap water was available to drink at all times.

Apparatus
The animals were housed in 8 stainless steel wire mesh cages (24 x 17 x 18 cm). A 2.5 cm square opening in the front wall of the cage allowed the animal access to the milk diet contained in a stainless steel drinking tube positioned 0.8 cm outside the cage. Licking of the tube was detected by an electronic contact relay drinkometer [35]. A preliminary study demonstrated that the small current supplied by the drinkometer did not alter the animal's taste discrimination or preference of the milk diets (see [19]). Each lick of milk diet was recorded by print out counters and an Esterline event recorder located in an adjacent room. The print out counters were programmed to count the number of licks in each bout of feeding and to print out after a 10 min period of no licking occurred.

Procedure
The rats were maintained on the plain milk diet for several weeks before surgery and for 1 week following it. Then in successive 5 day periods they were given the following diets: 0.03% quinine–milk, plain milk, 0.06% quinine-milk, 0.12% quinine–milk, and plain milk. After an additional 14 days on the milk diets the rats were given the high fat diet for 60 days. They were then returned to the plain milk diet for 17 days followed by 3 days each on 0.03% quinine–milk, 0.01% quinine–milk, and plain milk again. Throughout the experiment daily milk intake and body weight were recorded, and daily mean milk size and number were calculated. A meal was defined as a bout of feeding containing at least 5 licks and pauses no greater than 10 min. The minimal criterion of 5 licks was used to exclude the occasional contacts the rat made with the drinking tube while exploring the cage. The 10 min criterion was arbitrarily chosen after a review of the meal pattern literature (see Experiment 2).

RESULTS
The results of the first part of this experiment are summarized in Fig. 1. Preoperatively the VL and control groups were well matched on total daily intake, mean daily meal size, and mean daily meal number. Following surgery the 4 VL rats were hyperphagic and almost tripled their daily milk intake, while the 3 control rats showed a slight depression in their milk intake. The increased milk intake of the VL rats was associated with a large increase in their meal size, which was significantly greater than that of controls (p<0.01), but little change in meal frequency.

Adulteration of the diet with 0.03% quinine produced considerable reductions in the VL rats' total intake and meal size, while the control rats displayed little or no change. The mean total intake and meal size of the VL rats were still greater than that of the controls, but the differences were not significant. Both the VL and control groups increased their meal frequency when given the 0.03% quinine diet although the increase was reliable (p<0.05) only for the VL group. With the plain diet returned the VL rats again consumed more (p<0.01) milk per day and per meal than did the controls. The 0.06% quinine diet suppressed the total intakes and meal sizes of both the VL and control groups with the VL group showing the greatest reductions. The VL rats ate slightly but not significantly more of this diet per day and per meal than did the controls. Doubling the quinine concentration to 0.12% further reduced the intake and meal size, and the intakes of the 2 groups were very similar. Finally, when the plain diet was returned again both groups increased their food intake and meal size. The differences between the VL and control rats were less here than in the previous plain diet conditions partly because of the overeating of the
controls who were recovering from the weight lost during the 10 days on the quinine diet. In contrast to the effects on total intake and meal size, changes in diet palatability produced little or no effects on meal frequency.

The above findings are further illustrated in Fig. 2 which presents representative meal pattern records from an individual VL rat. These records clearly demonstrate the increase in meal size and total intake following surgery and the decrease in meal size and total intake when the quinine diets are presented. The records also indicate that the knife cuts disrupted the normal nocturnal feeding pattern of the rat by increasing its food intake during the day.

Table 1 presents the mean body weights of the VL and control groups on the last day of each treatment condition. Preoperatively the 2 groups did not differ significantly, but by the 7th postoperative day on the plain diet the VL rats weighed significantly more (p<0.05) than did the controls. The body weights of the VL rats remained significantly greater than that of the control group, although, as expected on the basis of the food intake data, their weights were more affected by the changes in diet palatability than were those of the control.

After 60 days on the high fat diet 3 of the VL rats were in static stage and their body weights (X = 663 g) were not increasing relative to the control group (X = 357 g). The fourth VL rat became ill and died shortly after the 60th day. When returned to the plain milk diet, however, the VL rats entered a second dynamic stage and gained more weight (X = 80 g) during the first 17 days on this diet than did the controls (X = 28 g). As indicated in Table 1, the VL rats remained heavier than the controls when the milk was adulterated with quinine, although they lost more (p<0.01) weight than did the controls on the quinine diets.

Figure 3 summarizes the meal pattern data obtained from the 3 obese VL and 3 control rats. Compared to controls, the VL rats consumed almost twice as much plain diet per day, and 3 times as much per meal, but took slightly less meals per day. When offered the 0.03% quinine diet, the VL group consumed less (p<0.05) diet (range = 13 to 18 ml/day) than did the controls (range = 27 to 54 ml/day), while the mean meal sizes of the 2 groups were similar. The daily intakes and meal sizes of both groups increased when given the 0.01% quinine diet, with 2 of the VL rats eating less, and 1 eating more of this diet per day relative to the intakes of the 3 control rats. Daily intakes were further increased with the return of the plain diet and the VL rats again ate more food per day and per meal than did the controls. Meal frequency was relatively unaffected by these diet changes and the VL rats ate consistently fewer meals compared to controls.

**EXPERIMENT 2**

The second experiment was conducted to replicate and expand the results of Experiment 1. Similar procedures were used except for differences in diets and order of diet presentation. In particular, the animals were maintained on a quinine milk diet preoperatively in an attempt to prevent the knife cut rats from overeating following surgery.

The present experiment also examined the effects of diet palatability on the diurnal distribution of feeding. Previous work has demonstrated that the diurnal feeding pattern is disrupted in VMH lesioned hyperphagic animals and they eat as much during the day as during the night [1, 2, 5, 10] (see also Fig. 2). Since reducing diet palatability was found in Experiment 1 to decrease meal size in hyperphagic rats to normal levels it is possible that is also restores a normal diurnal feeding pattern.

Finally, the data of Experiment 2 were analyzed using different meal criteria since the criteria used to define a meal may influence the nature of the results [13]. A meal was initially and arbitrarily defined, as in Experiment 1, as a bout of feeding containing pauses no greater than 10 min. The data were then reanalyzed using 20 min and 40 min
TABLE 1
MEAN BODY WEIGHTS OF VL AND CONTROL GROUPS ON THE LAST DAY OF EACH MILK DIET CONDITION

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Condition</th>
<th>N</th>
<th>Preop</th>
<th>Postop</th>
<th>0.03%</th>
<th>0.06%</th>
<th>0.12%</th>
<th>Plain</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Plain</td>
<td>4</td>
<td>289</td>
<td>367*</td>
<td>359*</td>
<td>416*</td>
<td>386*</td>
<td>358*</td>
</tr>
<tr>
<td></td>
<td>QHC1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>411*</td>
</tr>
<tr>
<td>CON</td>
<td>3</td>
<td>293</td>
<td>295</td>
<td>291</td>
<td>298</td>
<td>284</td>
<td>273</td>
<td>307</td>
</tr>
</tbody>
</table>

Experiment 1 B

<table>
<thead>
<tr>
<th></th>
<th>Plain</th>
<th>0.03%</th>
<th>0.01%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>QHC1</td>
<td>QHC1</td>
</tr>
</tbody>
</table>

| VL       | 3     | 743†  | 683†  |
| CON      | 3     | 385   | 373   |

Experiment 2

<table>
<thead>
<tr>
<th></th>
<th>0.03%</th>
<th>0.03%</th>
<th>0.03% or</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>QHC1</td>
<td>QHC1</td>
<td>0.06%</td>
</tr>
<tr>
<td>Preop</td>
<td>Postop</td>
<td>QHC1</td>
<td></td>
</tr>
</tbody>
</table>

| VL       | 4     | 274   | 301†   | 301*    | 420†   | 408†   |
| CON      | 5     | 254   | 254    | 262     | 294    | 284    |

*Significantly different from controls, p < 0.05.
†Significantly different from controls, p < 0.01.

pauses to define a meal which are criteria widely used by other investigators [1, 2, 15].

METHOD

Animals
Ten female CFE rats (Carworth, N.Y.) weighing 240–280 g were used. Five of the rats (VL group) received bilateral parasagittal knife cuts as in Experiment 1, while the remaining 5 rats received sham operations. One sham operated control animal shortly after surgery.

Apparatus
The housing and recording apparatus described in Experiment 1 and 2 additional living cages and associated recording equipment were used.

Procedure
The animals were habituated to the plain milk diet and housing conditions for 10 days. They were then maintained on a 0.03% quinine-milk diet for 9 days before and for 7 days following surgery. Four of the 5 VL rats who overate the 0.03% diet were then switched to a 0.06% quinine-milk diet, while the remaining VL and 4 control animals continued on the 0.03% quinine-milk diet for 6 days. All animals were then given 6 days of the plain milk diet. The diet was next adulterated with Tabasco sauce for 4 days, but this adulterant tended to curdle the milk and the data from this test are not presented. The rats were given another 6 days on plain milk diet followed by 6 days of a 4% sodium chloride diet. The intakes of plain diet were similar during the two 6 day periods and data are presented only for the second plain diet period.

The data were initially collected using a 10 min meal criterion as in Experiment 1. The data were then reanalyzed using the event recorder records with 20 min and 40 min meal criteria. The exact time the colony lights were turned on and off was indicated on the event recorder records and using this information and the individual print out tapes from each animal the diurnal distribution of food intake and meals were determined.

RESULTS

Figure 4 summarizes the meal pattern findings based on the 10 min criterion. The VL and control groups were well matched preoperatively on the 3 parameters of total intake, mean meal size, and meal number. Following surgery the VL rats consumed more of the 0.03% quinine-milk diet per day (p<0.01), and per meal (p<0.05) than did the controls. The VL rats also took slightly more meals compared to controls, but this difference failed to be significant. When the VL group was switched to the 0.06% diet they ate slightly more food per day and slightly less per meal compared to the controls on the 0.03% diet and these differences, as well as the difference in meal number, were not significant. With the plain diet returned the total daily intake and mean meal size of the VL rats were considerably greater (p<0.01) than that of the controls, while the differences in meal frequency were not reliable. Finally, the introduction of the 4% NaCl milk diet reduced the intakes
and meal sizes of both groups. The salt diet intake of the
VL group was slightly, but significantly (p<0.05) greater
than that of the controls, while the meal sizes of the 2
groups did not significantly differ. The meal frequencies
of the VL and control groups also did not reliably differ, but
both groups, taken together, ate more (p<0.05) meals per
day on the NaCl diet than on the plain milk diet. Although
water intake was not recorded, it was obvious that all rats
greatly increased their water consumption on the salt diet.

In Table 2 the effects of using 10 min, 20 min, and 40
min criteria on meal size and number are compared. As
expected, increasing the criterion increased meal size and
decreased meal number for both groups under all diet
conditions. In regard to between group comparisons,
increasing the meal criterion generally increased the dif-
f erences between the VL and control groups. For example,
using the 10 min criterion the VL rats ate slightly and
insignificantly smaller meals than did the controls under the
0.03%--0.06% quinine diet condition, but using the 20 and
40 min criteria the meal sizes of the hyperphagic rats were
significantly smaller than that of controls. Also, using the
10 min criterion the VL rats did not eat significantly more
meals per day compared to controls, but with the 20 min
and 40 min criteria the differences reached significance. On
the other hand, increasing the meal criterion did not
increase the difference between the meal size of the 2
groups on the 4% NaCl diet, and while the VL rats ate
larger meals than did controls on the 0.03% quinine diet
following surgery the difference failed to be reliable using
the 20 min criterion.

The effects of surgery and diet changes on the diurnal
distribution of food intake are illustrated in Fig. 5 which
presents the percentage of the total daily intake and of the
total daily meal number consumed during the day. Because
of equipment failures the percentage day intake, but not
meal data excludes one VL and one control rat. Prior to
surgery both the VL and control groups displayed the
typical nocturnal feeding pattern of rats, eating less than
20% of their total intake and meals during the light period.
Following surgery the VL rats increased their day food
intake and meal number and were now eating as much
during the day as during the night. Increasing the quinine
concentration of the diet to 0.06% while it decreased the
VL group's total intake, did not alter the equal day--night
distribution of feeding. Likewise, introduction of the plain
diet increased total food consumption of the VL group but
FIG. 5. Pre- and postoperative diurnal distribution of feeding expressed as percentage of total daily food intake and meal number consumed during the day by VL and control groups.

did not alter its distribution. The 4% NaCl diet, however, decreased not only total intake of hyperphagic rats, but also decreased (p<0.05) their percentage day intake. The percentage of meals taken during the day by the VL group also decreased slightly, but this effect was not reliable.

In contrast to the VL group, the control rats showed only a slight increase in daytime feeding following surgery and they continued to consume less than 20% of their total intake and less than 30% of their meals during the day. Furthermore, the distribution of feeding in the control group was unaffected by diet changes. Under all postoperative diet conditions the percentage day intake and percentage meal number of the VL group were significantly (p<0.05) greater than that of the controls. Also, as indicated in Table 3, the absolute number of meals the VL rats consumed during the day, but not during the night, was greater than that of the controls.

Table 1 summarizes the body weights of the 2 groups on the last day of each treatment condition. Preoperatively the VL and control groups did not significantly differ, although the VL rats weighed 20 g more than did the controls. Postoperatively the VL rats gained significantly (p<0.01) more weight than did the controls, and their weights remained significantly above control levels as diet palatability was altered.

DISCUSSION

The results of this study demonstrate the importance of diet palatability in determining total food intake and mean meal size, but not meal number or day-night distribution of feeding in hyperphagic and normal animals. The effects of the knife cuts and diet palatability on these meal pattern parameters will be discussed in order.

Total Food Intake

The total daily food intake of the hyperphagic rats was, as expected on the basis of previous findings [7, 25, 32], more influenced by diet palatability than was the intake of the controls. That is, while both groups decreased their food consumption when given the quinine or salt diets, and increased it when given the plain milk diet, the hyperphagic rats showed the greatest changes. The dynamic hyperphagic animals, however, ate as much or more of the adulterated diets as did the controls which is consistent with earlier findings [32]. In fact, it was necessary in Experiment 2 to give 4 of the 5 VL rats a more bitter diet than controls in order to equate the intakes of the 2 groups. (This finding is not inconsistent with previous reports of hypothalamic finickiness, for in the present experiment quinine was added to a highly palatable sweet liquid diet in contrast to the dry powder diets used in earlier experiments.) The
obese hyperphagic rats of Experiment 1, on the other hand, ate less of the quinine-milk diet than did the control rats and this confirms the view that obesity, rather than medial hypothalamic damage, is the major cause of the animal's finickiness to unpalatable foods [8, 11, 27, 28, 32].

**Meal Size**

The knife cut rats overate primarily by increasing their meal size which is in agreement with most previous results obtained with VMH lesioned animals [1, 5, 33, 35]. However, meal size, like total intake was dependent on the palatability of the diet in both hyperphagic and control animals. Adulteration of the diet with quinine or salt reduced meal size and this effect was not a secondary adaptation to reduce total intake since the very first meals on the adulterated diets were smaller than those taken of the plain diet. These and other recent findings [15, 16, 17, 18] demonstrate that meal size is determined not only by the nutritional state of the animal and the caloric content of the diet, but also by the taste of the food.

The most important finding was that the hyperphagic rats did not consume larger than normal meals when given adulterated diets which limited their total intakes to control levels. In fact, their meal size with the 20 min and 40 min criteria was less than controls when the intakes of the 2 groups were equated under the 0.03%–0.06% quinine diet condition. It appears unlikely, therefore, that the hyperphagic rats suffered from a primary short term satiety deficit which impaired their ability to limit meal size. It may be argued, on the other hand, that the hyperphagic rats had a short term satiety deficit but were more sensitive than controls to the meal size reducing effects of the diet adulteration. However, this interpretation is not consistent with the finding that in order to equate the meal size and total intake of the hyperphagic and control groups it was necessary to give the hyperphagic rats a more concentrated quinine diet. Further evidence against a short term satiety deficit is the finding that hyperphagic animals continue to overeat by increasing their meal frequency when enlarged meals are prevented by surgically reducing stomach capacity [5] or reducing food availability [13]. Even when enlarged meals are not prevented hyperphagic rats may sometimes overeat by increasing their meal frequency rather than their meal size [2]. Finally, several studies have consistently failed to obtain direct evidence for short term satiety deficits in hypothalamic hyperphagic rats [20, 23, 30].

**Meal Frequency**

In addition to increasing their meal size, the hyperphagic rats showed a slight increase in their meal frequency which was significant, relative to controls, when 20 min and 40 min meal criteria were used (Table 2). This increased meal number of the hyperphagic rats resulted from their increased meal taking behavior during the day (Fig. 5) and they did not eat more meals than controls during the night (Table 3). Similar findings have been reported by Becker and Kissileff [2] although conflicting results have been obtained by other investigators [1, 5, 33, 35]. However, while the hyperphagic rats ate more meals than controls when in the dynamic stage, they ate less when tested in the obese stage (Fig. 3).

In contrast to meal size, meal frequency in both the VL and control groups was relatively unaffected by changes in diet palatability. The introduction of the 0.03% quinine diet in Experiment 1 produced a small increase in the number of meals, but subsequent presentations of quinine diets did not increase meal frequency. Meal frequency was also somewhat increased when the rats were given the 4% NaCl diet in Experiment 2. In this case, however, the increased meal number may have resulted from the post-ingestive effects of the salt diet. That is, the diet was hypertonic and necessitated that the rats increase their water intake. Thus, the animals may have taken more and longer pauses in their feeding because they were drinking more water with their meals. Consistent with this view is the finding that meal number was not increased by the salt diet when 20 min and 40 min criteria were used to define a meal. The relative insensitivity of meal frequency compared to meal size to diet palatability changes is compatible with other findings demonstrating that size is a more responsive parameter than frequency to a variety of environmental changes [16, 18].

**Diurnal Distribution of Feeding**

The present results revealed that VL knife cuts, like VMH electrolytic lesions [1, 2, 5, 10], disrupt the normal diurnal feeding pattern of the rat by increasing daytime food consumption to nighttime levels. Our findings further demonstrate that this diurnal disruption is independent of diet palatability and total food intake. That is, the hyperphagic rats not only ate more food during the day when their total daily intakes were greater than controls, but also when their intakes were limited to control levels by quinine adulteration. Unlike the quinine diet the NaCl diet reduced the percentage day intake of the hyperphagic rats, although it remained elevated compared to controls. This result may be related to the post-ingestive effects of the salt diet rather than from its unpalatable taste. Kakolewski et al. [10] recently reported that VMH lesions, while they disrupt the nocturnal feeding pattern, do not alter the

### TABLE 3

**DIURNAL DISTRIBUTION OF MEALS**

<table>
<thead>
<tr>
<th></th>
<th>0.03% QHC1 Preop</th>
<th>0.03% QHC1 Postop</th>
<th>0.03% or 0.06% QHC1 Postop</th>
<th>Plain</th>
<th>4% NAC1 Postop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day</td>
<td>2.8</td>
<td>2.6</td>
<td>11.8*</td>
<td>4.4</td>
<td>10.6*</td>
</tr>
<tr>
<td>Night</td>
<td>15.8</td>
<td>16.1</td>
<td>13.3</td>
<td>13.1</td>
<td>14.3</td>
</tr>
<tr>
<td>Total</td>
<td>18.6</td>
<td>18.7</td>
<td>25.1</td>
<td>17.5</td>
<td>24.9</td>
</tr>
</tbody>
</table>

*Significantly different from controls, p<0.01.
nocturnal drinking pattern of rats. Since the hyperphagic rats, as well as the controls, had to increase their water intake in order to handle the hypertonic salt diet, it is possible that they consumed less salt diet per day, and more per night, relative to the other diets, because they were still nocturnal drinkers.

The causes and consequences of the abnormal day-night feeding pattern displayed by hypothalamic hyperphagic rats are not entirely clear. The present findings that the disruption persists when total intake is reduced by quinine adulteration negates the possibility that the hyperphagic rat increases its daytime intake only because its nighttime intake is already maximum and cannot be further increased.

It is also unlikely that the diurnal disruption represents a general impairment in the hyperphagic rat's diurnal or circadian activity pattern, as suggested by Richter [24], since its diurnal drinking and running rhythms remain intact [4, 10]. Finally, the possibility that separate neural disorders are responsible for the effects of VMH damage on total food intake and diurnal distribution of feeding must be considered, since lesions of the dorsomedial hypothalamus which produce hypophagia rather than hyperphagia also produce equal day-night distributions of food intake [3].

The present study along with previous meal pattern experiments demonstrate that neither increased meal size nor increased frequency is invariably associated with VMH damage [1, 2, 5, 33, 34, 35]. Thus, it is unlikely that such damage produces overeating and obesity because it impairs the short term control of meal size or meal frequency. Hypothalamic hyperphagia is always accompanied, on the other hand, by a decrease in the satiety ratio, defined as meal size divided by postintermeal interval [2]. Based on this fact Becker and Kissileff [2] have proposed that VMH damage may destroy the neurons responsible for maintaining satiety between meals. However, any type of hyperphagia, whether caused by previous food deprivation, cold exposure, lactation, or genetic or hormonal disorders, is by necessity associated with a decrease in the satiety ratio. The reduced satiety ratio of VMH damaged animals, therefore, cannot by itself be taken as evidence for a disorder in the neural mechanism responsible for between meal satiety.

Alternative explanations of the hypothalamic hyperphagia syndrome have emphasized the long term regulation of food intake and body weight [9,11]. Sclafani [27,28], for example, has proposed that hypothalamic obesity results from a reduction in the lipostatic inhibition of appetite. The present findings are compatible with this view in that both the increased total daily intake and increased meal size of the hyperphagic rat were dependent upon the availability of a palatable diet. Furthermore, the variability in the meal pattern findings reported to date is not inconsistent with the lipostatic interpretation of the hyperphagia syndrome. The VMH damaged animal may overeat to reach a new upper body weight by altering its meal pattern in a variety of ways and the postulated lipostatic disruption need not dictate a particular meal pattern change. The exact feeding pattern the animal adopts may vary from one experiment to the next because of differences in the environmental conditions known to influence meal taking behavior in rats [6, 15, 16, 17, 18].

REFERENCES


