Intake of High-Fat Food Is Selectively Enhanced by Mu Opioid Receptor Stimulation within the Nucleus Accumbens

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ABSTRACT
The present study was designed to further investigate the nature of feeding induced by opioid stimulation of the nucleus accumbens through an examination of the effects of intra-accumbens (ACB) opioids on macronutrient selection. In 3-hr tests of free-feeding (satiated) rats, intra-ACB administration of the mu receptor agonist d-Ala²,N-Me-Phe⁴,Gly-ol⁵-enkephalin (DAMGO; 0, 0.025, 0.25 and 2.5 μg bilaterally) markedly enhanced the intake of fat or carbohydrate when the diets were presented individually (although the effect on fat intake was much greater in magnitude). Intra-ACB injections of DAMGO, however, produced potent preferential stimulatory effects on fat ingestion with no effect on carbohydrate ingestion when both fat and carbohydrate diets were present simultaneously. Moreover, this selective stimulation of fat intake was independent of base-line diet preference and could be blocked by systemic injection of naltrexone (5 mg/kg). We also examined the effect of 24-hr food deprivation on the pattern of macronutrient intake in rats with access to both carbohydrate and fat. In contrast to the DAMGO-induced selective enhancement of fat intake, food deprivation significantly increased the intake of both diets to the same extent; however, in this case, only the stimulated fat intake was blocked by systemic naltrexone. Intra-ACB administration of DAMGO in hungry rats produced an effect similar to that observed in free-feeding rats; preference was strongly shifted to fat intake. Similarly, the opioid antagonist naltrexone (20 μg) infused directly into ACB preferentially decreased fat intake in hungry rats. These findings suggest that endogenous opioids within the ventral striatum may participate in the mechanisms governing preferences for highly palatable foods, especially those rich in fat.

Endogenous opioid peptides have been found to be involved in the modulation of feeding behavior in both humans and rats. Since the initial findings that morphine increased food consumption (Flowers et al., 1929; Martin et al., 1963), the role of endogenous opioids in different aspects of feeding behavior, such as caloric intake, taste reactivity, diet preference and the interaction with other neuropeptides, has been investigated extensively. Generally speaking, peripheral or central injections of opioid agonists, including mu, delta and kappa agonists, increase food intake and opioid antagonists decrease food consumption, at least in the short term, under various testing conditions (Holtzman, 1974; Reid, 1985; Levine and Billington, 1989). In addition to modifying total food consumption, opioid peptides influence macronutrient selection. Several studies with dietary self-selection paradigms showed that acute systemic morphine injections preferentially stimulate fat consumption while suppressing carbohydrate intake and having little modulatory effect on protein intake (Marks-Kaufman and Kanarek, 1980; Marks-Kaufman, 1982; Ottaviani and Riley, 1984). A similar pattern has been observed when rats were treated with morphine chronically (Ottaviani and Riley, 1984; Gosnell and Krahn, 1993). In contrast, the opioid antagonist naloxone selectively decreases fat intake when carbohydrate and protein are also available to rats (Marks-Kaufman and Kanarek, 1981, 1990). However, when base-line diet preference of rats was taken into consideration, Gosnell et al. (1990) found that morphine primarily increased preferred diet consumption rather than the intake of a specific macronutrient. Evans and Vaccarino (1990) also found that systemic morphine enhances intake of preferred food. These findings seem to support the currently favored hypothesis that opioids play a role in modulating the palatable aspects of food, making food more rewarding (Cooper and Kirkham, 1993).

Most of the studies described above have been conducted with the peripheral or intraventricular administration of morphine. Although a number of studies have demonstrated the involvement of the ACB in opioid-induced hyperphagia, little work has been done to investigate its role in macronutrient selection. In light of evidence implicating the accumbens in the processing of reward-related information, the investigation of how this structure modulates macronutrient preference could be particularly valuable for further under-

ABBREVIATIONS: ACB, nucleus accumbens; DAMGO, d-Ala²,N-Me-Phe⁴,Gly-ol⁵-enkephalin.
standing of mechanisms underlying opioid-induced hyperphagia. Indeed, it is well documented that the ACB is a crucial brain area responsible for the establishment and maintenance of drug-seeking behavior induced by opiates, psychostimulants and alcohol (Wise and Bozarth, 1987; Koob and Bloom, 1988). Recently, evidence emerging from both clinical surveys and animal studies demonstrated parallels between drug or alcohol addiction and food cravings (Mitchell et al., 1985; Morabia et al., 1989; Krahn and Gosnell, 1991; Bell et al., 1994; Gosnell et al., 1995). Accordingly, it is possible that the ACBs may be involved in both drug reward and the subjective reward derived from food. This is consistent with our previous studies showing that in satiated rats, microinjections of mu and delta opioid receptor agonists into the ACB stimulate chow feeding and intake of liquid sucrose, effects both of which are blocked by pretreatment with naltrione (Bakshi and Kelley, 1993a; Kelley et al., 1996; Zhang and Kelley, 1997). Furthermore, the phenomenon of conditioned feeding induced by multiple morphine microinjections into the ACB provides further evidence to suggest the involvement of reward mechanisms (Bakshi and Kelley, 1994; Kelley et al., in press). The current study extended these investigations to examining intra-ACB opioid effects on macronutrient selection. Because our past findings showed that the orexigenic effect of opioids seems to be mediated primarily by mu receptors (Bakshi and Kelley, 1993a; Zhang and Kelley, 1997), the current study was focused on the mu receptor. In this study, the relationship between base-line diet preference and the feeding stimulatory effect of opioids was investigated. We further compared the pattern of diet selection induced by opioid treatments with that induced by food deprivation. We assumed that intake in food-restricted rats is driven primarily by energy needs, with taste or palatability being relatively less important. In consideration of the notion that opioids facilitate feeding in satiated animals by enhancing the orosensory palatability of food, we predicted a difference in the profile of these two feeding paradigms.

**Materials and Methods**

**Animals.** A total of 54 male rats (Harlan-Sprague Dawley, Indianapolis, IN) weighing between 270 and 320 g were used in the present study. Animals were housed two or three to a cage with access to unlimited water and standard normal laboratory chow (Purina Chow) unless they were under specific food deprivation schedule (see details of experimental design). The lights were on at 7:00 A.M. and off at 7:00 P.M.

**Surgery.** Animals were handled for several days after arrival. For surgery, animals were anesthetized with a mixture of ketamine and xylazine (90 mg/kg ketamine and 9 mg/kg xylazine). Bilateral guide cannulae were secured to the skull with stainless steel screws and light curable dental resin (Dental Supply of New England, Boston, MA). Coordinates for the aimed sites were (in mm, with tooth bar 4 mm below interaural zero): +1.5 from the bregma in the anteroposterior plane, 1.5 from the midline in the intermedial plane and −5.5 from the skull in the dorsoventral plane. For this study, placements were not aimed specifically at the core or shell and for the most part were localized in an area between these two subregions. After the surgery, wire stylets were placed in the guide cannulae to prevent occlusion.

**Drugs and microinjection.** DAMGO, the mu receptor agonist, and the general opioid receptor antagonist naltrione were obtained from Research Biochemicals (Natick, MA). Both of these drugs were dissolved in sterile 0.9% saline. The vehicle was always sterile 0.9% saline.

After the stylets were removed, the drugs in a volume of 0.5 μl were infused through 12.5-mm injector cannulae into the ACB by a microdrive pump (Harvard Apparatus, South Natick, MA), connected via polyethylene tubing (PE-10). Thus, injector tips extended 2.5 mm beyond the end of the guide cannula, for a final dorsoventral coordinate of −8.0 mm from skull. The rate of injection was 0.32 μl/min with the total duration of infusion being 1 hr 33 min. One additional minute was allowed for diffusion. Injectors then were removed, and the stylets were replaced.

**Behavioral testing and experiment design.** After 2 to 3 days of recovery, the rats were brought into the testing room and provided daily with the testing diet (high-carbohydrate diet, high-fat diets or both, depending on the experimental design) for 3 hr in a testing cage. As indicated in table 1, both diets contained equal amounts of vitamins, minerals, choline chloride, fiber and protein, when equated on a caloric basis. Diets were obtained from Teklad Diets (Madison, WI). This period lasted for several days until stable food consumption across days was obtained. Except for the daily 3-hr testing period, animals were provided with standard laboratory chow, either ad libitum or with food restriction, depending on the experimental design (see below). Before the testing period began, a saline injection and a sham injection were given on separate days to habituate the rats to the injection procedure. Preweighed jars containing diets were attached to the testing cages. At the end of each testing period, the diet jars were removed and weighed, and the corresponding diet intake in calories was calculated and corrected for spillage. Water was available during the entire testing session.

In experiments 1 and 2, non-food-deprived rats were treated with intra-ACB microinjections of DAMGO in doses of 0 (saline), 0.025, 0.25 and 2.5 μg/dose in a counterbalanced order. There was at least 1 day spaced between injections. The testing session began immediately after the microinjections. For the first experiment, there were two groups of rats in which the effect of DAMGO on the consumption of high-carbohydrate diet and high-fat diet were evaluated separately (n = 10 for carbohydrate group, n = 14 for fat group). The diet was weighed every hour over the 3-hr testing period. For experiment 2 (n = 14), non-food-deprived rats had simultaneous access to both diets in the testing cage, and the position of diet jars was exchanged daily. After the rats were adapted to the testing diets, base-line intakes over 3 hr were measured for 3 consecutive days; thus, rats were divided into carbohydrate-prefering (CP) and fat-prefering (FP) groups on the basis of the ratio of the average of carbohydrate intake to fat intake. For CP group, the ratio was >1; otherwise, rats were assigned to the FP group. One day after the determination of

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Composition of diets used in experiments</th>
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<tbody>
<tr>
<td></td>
<td>Carbohydrate/protein</td>
</tr>
<tr>
<td>Corn starch</td>
<td>917.9</td>
</tr>
<tr>
<td>Dextrin</td>
<td>455.8</td>
</tr>
<tr>
<td>Sucrose</td>
<td>152.9</td>
</tr>
<tr>
<td>Cacao</td>
<td>429.2</td>
</tr>
<tr>
<td>DL-Methionine</td>
<td>6.5</td>
</tr>
<tr>
<td>Vegetable shortening</td>
<td>578.0</td>
</tr>
<tr>
<td>Safflower oil</td>
<td>101.8</td>
</tr>
<tr>
<td>AIN-76A vitamin mix</td>
<td>19.9</td>
</tr>
<tr>
<td>AIN-76 mineral mix</td>
<td>70.0</td>
</tr>
<tr>
<td>Choline chloride</td>
<td>5.0</td>
</tr>
<tr>
<td>Cellulose (Alphacel)</td>
<td>99.8</td>
</tr>
<tr>
<td>Weight (g)</td>
<td>7800</td>
</tr>
<tr>
<td>Total energy (kcal)</td>
<td>1310.1</td>
</tr>
</tbody>
</table>

All components are expressed as weight (g).

a Assuming a protein content of 99%.

b The vitamin and mineral mixes contain 97% and 12% sucrose, respectively.

c Based on energy values of 4, 9 and 4 kcal/g for carbohydrate, fat and protein, respectively.
Experiment 2: Effect of Intra-ACB Mu Opioid Agonist Infusion on Diet Preference in Nondeprived Rats

In this test with the two macronutrient-specific diets simultaneously available, free-feeding rats given an intra-ACB infusion of DAMGO displayed a strong preference for fat compared with the carbohydrate diet (fig. 2). Significant effects of dose condition [F(3,39) = 22.029, P < .01], diet [F(1,13) = 9.851, P < .01] and dose × diet interaction [F(3,39) = 14.141, P < .01] were found. As shown in figure 2 (top), the 0.25- and 2.5-µg doses of DAMGO significantly increased total caloric intake (the sum of fat intake and carbohydrate intake) (P < .01) and fat intake (P < .01), whereas carbohydrate consumption remained unchanged. Furthermore, the enhancement induced by 2.5 µg DAMGO was completely blocked by naltrexone treatment (P < .01).

Fig. 1. The effect of intra-ACB injection of vehicle and different doses of DAMGO on fat and carbohydrate intake when these two diets were presented to rats separately for 3 hr. Bars represent mean ± S.E.M. **P < .01, *P < .05, compared with vehicle control. Carb, carbohydrate; kcal, kilocalories.
The correlation between the intake of each diet and baseline diet intake was shown in Table 2, which suggested increased fat intake elicited by the 2.5-μg dose was positively correlated with base-line fat intake (P < .01) and negatively correlated with base-line carbohydrate intake (P < .05). To investigate the relationship between drug effect and baseline preference in detail, the data were reanalyzed after dividing the rats into CP and FP groups based on the criteria mentioned earlier. As demonstrated in Figure 2 (middle and bottom), the 0.25- and 2.5-μg doses significantly facilitated fat feeding in both CP and FP groups. In contrast, no effect was observed on carbohydrate intake in both groups. Moreover, the effect on fat intake induced by the 2.5-μg dose was blocked by naltrexone pretreatment (P < .01). These results indicated that DAMGO exerted a pronounced preferential influence on fat intake, regardless of preexisting diet preference.

Experiment 3: Effect of Food Deprivation Alone and Food Deprivation in Combination with Intra-ACB Mu Agonist or Antagonist on Diet Selection

Food-deprivation alone. The mean value of intake across 3 consecutive days before the first test day was calculated and served as base-line intake in the satiated condition. The cumulative 3-hr diet intake was analyzed with two-way analysis of variance: feeding condition (satiation vs. hunger) × diet (fat vs. carbohydrate). In contrast to the results in experiment 2, only the main effect of feeding condition was found significant [F(1,15) = 82.819, P < .01], with no interactions or effects of diet content. This profile indicates that 24-hr food deprivation produced a significant increase in caloric intake compared with the satiated feeding condition but, more importantly, that the magnitude of this increase was similar across both diet conditions as shown in Figure 3 (top). Similarly to experiment 2, however, only enhanced fat intake was blocked by intraperitoneal injection of naltrexone (P < .01).

In contrast to the selective effect of DAMGO on fat preference demonstrated in experiment 2, food deprivation produced no preferential alterations in macronutrient consumption in relation to base-line preference. Food deprivation-induced diet consumption was significantly correlated with corresponding base-line diet preference (Table 3). As shown in Figure 3 (middle and bottom), food deprivation increased consumption of both diets to the same degree in both FP (P < .01 for both diets) and CP groups (P < .01 for carbohydrate; P < .05 for fat).

Diet selection induced by intra-ACB DAMGO or naltrexone in hungry rats. As for the effects of DAMGO on diet choice after food deprivation, two-way analysis of variance showed significant main effects for dose [F(1,15) = 13.536, P < .01], and diet [F(1,15) = 9.367 P < .01]. There also was a significant dose × diet interaction [F(1,15) = 7.552, P < .05]. Further analysis of simple comparisons showed that the selective stimulatory effect of DAMGO on fat consumption (P < .01) was responsible for the interaction (Figure 4).

An intra-ACB infusion of naltrexone suppressed food intake, shown in Figure 4. Analysis of variance revealed a significant main effect of drug treatment [F(1,15) = 54.219, P < .01] and a significant drug × diet interaction [F(1,15) = 13.593, P < .01]. Follow-up simple comparison between drug and saline treatment at each level of diet demonstrated that naltrexone treatment dramatically inhibited fat consumption (P < .01) while producing less effect on carbohydrate intake (P < .05).

TABLE 2
Correlation coefficients (Pearson’s r) for the relationship between base-line diet intake and the effect of DAMGO on diet intake

<table>
<thead>
<tr>
<th>Effect of DAMGO on the intake of each diet (difference from control intake)</th>
<th>Fat</th>
<th>Carbohydrate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Base-line intake (μg)</td>
<td>0.025</td>
<td>0.25</td>
</tr>
<tr>
<td>Fat</td>
<td>0.137</td>
<td>0.231</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>-0.096</td>
<td>-0.426</td>
</tr>
</tbody>
</table>

<sup>a</sup> P < .01 and <sup>b</sup> P < .05 indicate significant correlation.
Discussion

The main purpose of this study was to investigate the role of ACB mu opioid receptors in macronutrient preference and to obtain further information concerning the mechanisms underlying opioid-induced feeding. Our data demonstrate that within this brain area, stimulation of mu receptors exerts a powerful preferential facilitory effect on fat consumption. Furthermore, this effect is independent of preexisting diet preference and is blocked by cotreatment with naltrexone. Finally, the difference in the profiles between DAMGO- and food deprivation-induced diet selection suggests that endogenous opioids, at least within the ACB, may play a key role in the factors that govern food intake other than energy deficits, particularly palatability.

There is some debate with regard to the nature of opioid-induced feeding in terms of whether such feeding is nutrient specific or rather specific to individual base-line preferences. A selective increase in fat intake was reported by several studies with rats undergoing acute (Marks-Kaufman and Kanarek, 1980; Marks-Kaufman, 1982; Romsos et al., 1987) or chronic treatment with morphine under different feeding conditions (Ottaviani and Riley, 1984; Marks-Kaufman and Kanarek, 1990; Gosnell and Krahn, 1993). Consistent with the present results, naltrexone or naloxone selectively decreased fat intake in a self-selection paradigm (Marks-Kaufman and Kanarek, 1980; Marks-Kaufman, 1982). On the other hand, some researchers have argued that opioid-induced feeding is preference specific rather than macronutrient specific, finding that the effects of morphine on nutrient intake were modified when base-line preference was taken into consideration. Three studies provide evidence to support the preference-related hypothesis. The most direct evidence is provided by the study of Gosnell et al. (1990), who found that systemic injection of morphine preferentially increased fat intake in fat-preferring rats and carbohydrate intake in carbohydrate-preferring rats. They concluded that morphine caused a selective increase in the intake of preferred foods. Several differences may account for the discrepancies between that study and the present data. First, in the study of Gosnell et al., morphine was administered systemically, therefore stimulating many regions and subtypes of opiate receptors. Second, rats were allowed 24-hr access to the different diets compared with only 3-hr access in the present experiments. Consequently, the restricted base-line tests may be more strongly influenced by taste preference or palatability. Furthermore, it is noteworthy that in the study of Gosnell et al., although no significant effect was found on carbohydrate intake in fat-preferring rats, morphine still produced a significant stimulatory effect on fat intake, even in the carbohydrate-preferring rats. This finding suggests that morphine tends to produce a relatively greater effect on fat intake, although the size of the effect may be dependent on base-line preference. An additional study reported that systemic administration of morphine facilitated intake of carbohydrate in carbohydrate-deprived rats and protein in protein-deprived rats (Evans and Vaccarino, 1990). Furthermore, Glass et al. demonstrated that the anorexic effect of naloxone on
neuropeptide Y-induced feeding of carbohydrates and fats was dependent on diet preference. In these cases, however, 
the base-line preference of the given nutrient was deter-
mined by either nutrient deficit or neuropeptide Y-induced 
stimulation, both of which introduce a variety of factors that could mask the pure effects of opioids on nutrient selection.

The data from the present experiments help to clarify this 
debate. They suggest, at least for the ACB, that mu receptor 
stimulation preferentially enhances intake of foods that have inherently high palatability, such as fat. This effect is mostly 
clearly observed when animals have a choice of macronu-
trients or foods. When given a choice between carbohydrate and 
food, ACB opioid stimulation enhanced only fat intake. Simi-
larly, when rats are presented with both sweetened 
water and unsweetened water, only intake of the sweetened solu-
tion is enhanced (Zhang and Kelley, 1997), and when given a 
choice between chow and sugar, only sugar intake is en-
hanced (Evans and Vaccarino, 1996). However, because all 
foods that rats eat naturally have some inherent palatability, 
such as rat chow or carbohydrate, DAMGO also is able to 
stimulate intake when such foods are present alone. Further-
more, intra-ACB administration of DAMGO not only in-
creases sucrose drinking (Zhang and Kelley, 1997) but also enhances the intake of salt solution (unpublished observa-
tion). It has also been reported that morphine suppresses 
aversive reactions to a bitter solution such as pure quinine 
(Doyl et al., 1993). Taken together, these experiments sug-
gest that ACB opioids are involved in modulating the palat-
able response to food across a broad range of macronutri-
ents and tastes. This idea may appear somewhat at odds with our 
present finding that the opioid effect was relatively fat spe-
cific. However, it is highly likely that the extent of enhanced 
palatability is correlated with the content of the fat and sugar 
because in general, highly preferred foods contain com-
binations of fat and sugar (or fat and salt) (Drewkowski et al., 
1992b). In support of this notion, there is evidence of a 
correlation between taste palatability and activation of en-
genous opioid systems. For instance, consumption of 
highly palatable food rich in fat and sugar causes an increase in the release of beta-endorphin, (Brennan et al., 1994; Welch et 
al., 1996). Additionally, an interesting interaction has been 
found between drug self-administration and food or taste 
preference. For example, fat-preferring rats consume more 
ethan ethanol than carbohydrate-prefering rats (Forsander, 1988; 
Krahn and Gosnell, 1991). In an earlier study, it was found 
that the rats with high preference for fat ultimately became 
morphone drinkers, whereas those with low preference for fat 
did not consume the drug (Marks-Kaufman and Lipeles, 
1982). Rats that have a high preference for sweet taste show a 
greater tendency to self-administer morphine (Gosnell et al., 1995). These lines of evidence suggest the existence of a 
close relationship between the opioid reward system and 
normal circuits processing taste reactivity and palatability. 
More specifically, we hypothesize that opioid systems with 
the ACB are the major region in which this processing occurs; 
stimulation of opioid receptors in this region results not only in 
hyperphagia but also in reinforcing effects as demon-
strated in the place preference paradigm or intracerebral self-administration (Van der Kooy et al., 1982; Goeders et al., 
1984; Mucha and Iversen, 1986; Zhang and Kelley, 1997).

It is of interest to note the difference in the profiles of 
DAMGO-induced and food deprivation-induced diet intakes. 
In nondeprived rats with diets presented concurrently, 
DAMGO augmented only fat consumption without an effect on 
carbohydrate intake. However, 24-hr food deprivation en-
hanced feeding of both diets to the same degree. When base-
line intake of the two diets was taken into consideration, the effect of DAMGO on fat intake was positively correlated with 
base-line fat intake, whereas no correlation was found be-
tween DAMGO-induced carbohydrate intake and base-line 
carbohydrate preference. In contrast to the DAMGO-induced 
selective change in diet preference, food deprivation pro-
duced no effect on diet preference; indeed, the degree of 
intake of each diet after deprivation corresponded to the 
initial base-line preference in the nondeprived state. Support 
for this idea is provided by the finding that naltrexone infu-
sion was able to decrease both fat and carbohydrate intake 
when infused into the ACB in hungry rats. Granted, the effect of intra-ACB naltrexone was less potent on carbohy-
drate than on fat, and in the initial part of that experiment, 
systemic naltrexone did not affect deprivation-induced carbohy-
drate intake. This discrepancy may be accounted for by 
the higher concentration of antagonist in the intracerebral injection. Because naltrexone is not entirely selective, it may 
also be possible that blockade of delta receptors accounts for 
the decrease in carbohydrate intake. A number of studies, 
including our own, have shown that infusion of delta agonists 
into ACB or ventral tegmental area enhances food intake 
(Majeed et al., 1986; Bakshi and Kelley, 1993a; Noel and 

These data also raise the question concerning the relation-
ship between hunger and palatability. Although many foods 
have inherent high palatability and are consumed for their 
pleasurable effects, rather than to relieve an energy deficit, 
hunger clearly increases palatability of all foods. Thus, it is 
important to note that opioids likely play a role in palatabil-
ity both in the nondeprived and food-deprived states. This 
hypothesis is consistent with clinical studies in control and 
obese patients, which show that opioid antagonists decrease 
the intake of foods, especially those rich in sugar and fat, 
even though hunger and satiety ratings are not affected 
(Yeomans et al., 1990; Drewkowski et al., 1992a; Yeomans 
and Gray, 1996). In these reports, the finding that naloxone preferentially decreases intake of sweet and/or high-fat food

Fig. 5. Photomicrographs of cannula placements within ACB of repre-
sentative animals from the different experiments. A, Experiment 1, fat 
group. B, Experiment 1, carbohydrate group. C, Experiment 2, choice 
test, satiated rats. D, Experiment 3.
by reducing the “pleasantness” rating of these food items lends support to the hypothesis that opioids play an important role in palatability.

Foods containing fat and sugar have high caloric density, and thus the preferential effect of opioids on consumption of these foods might have an adaptive significance from the standpoint of evolution. Throughout evolution, famine or food scarcity, rather than obesity, was likely to be a problem. To survive in these conditions, organisms would have to develop strategies to prepare for impending food shortages due to seasonal or climatic variations or, at times, starvation. Because calorie-dense food (especially fats, which are beneficial for long-term energy storage) has a highly rewarding taste, this would ensure that animals obtain and store energy whenever such food might be available. This notion is consistent with our findings that in hungry rats, DAMGO and naltrexone preferentially increased and suppressed the intake of fat, respectively. In modern Western societies, however, in which the food supply is abundant and overweight rampant, overactivation of the endogenous opioid peptides may contribute partly to human obesity, binge eating and uncontrollable craving for sweet and high-fat foods. The present data suggest that in particular, activation of mu opioid receptors within the ventral striatum may participate in the selection and consumption of highly palatable foods.

References


