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Hedonic Hunger Is Associated with Intake of Certain High-Fat Food Types and BMI in 20- to 40-Year-Old Adults

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ABSTRACT

Background: Hedonic hunger occurs in response to a desire to consume food for pleasure. The μ-opioid system regulates the hedonic impact of food and the opioid receptor mu 1 gene (*OPRM1*) polymorphism has been associated with fat intake.

Objectives: The aim of this study was to determine whether the intake of high-fat food is associated with hedonic hunger and the *OPRM1* polymorphism and whether these variables are related to BMI.

Methods: Participants were 20- to 40-y-old women and men enrolled in Poznań, Poland in 2016–2018. The frequency of consumption of high-fat food was measured using a validated application for mobile devices. Hedonic hunger was assessed with the use of the Power of Food Scale (PFS). PFS1, PFS2, and PFS3 scores were generated for food available, food present, and food tasted, respectively. Genotyping of rs1799971 in the *OPRM1* gene was performed using TaqMan probes. The associations were analyzed using linear regression or logistic regression, as appropriate.

Results: Hedonic hunger scores were not associated with total high-fat food intake. Total PFS was associated with snack intake (β: 0.16, $P = 0.0066$). PFS1 was positively associated with healthy high-fat food intake (β: 0.27, $P = 0.0001$) and PFS2 with sweet high-fat food and fast-food intake (β: 0.27, $P = 0.0030$). *OPRM1* genotype and hedonic hunger interacted on fast-food intake (β: −0.17; $P < 0.0154$). Total PFS and PFS2 increased the chance of having a BMI $\geq$ 25 kg/m^2 (OR: 1.43; 95% CI: 1.03, 2.01; $P = 0.0335$ and OR: 1.89; 95% CI: 1.37, 2.61; $P = 0.0001$, respectively), whereas PFS3 decreased it (OR: 0.61; 95% CI: 0.41, 0.87; $P = 0.0082$).

Conclusions: Hedonic hunger is associated with the intake of selected types of high-fat food, but not with its total intake, in people aged 20–40 y. Associations between hedonic hunger and fast-food intake can be modified by *OPRM1* genotype. Hedonic hunger is associated with BMI. *J Nutr* 2021;151:820–825.

Keywords: hedonic hunger, high-fat products, food choice, gene polymorphism, *OPRM1*

Introduction

A positive energy balance leads to overweight and obesity, which have reached epidemic proportions globally (1). In well-nourished populations, eating is driven not only by energy needs, but also by pleasure, which can be intensified by environmental cues, such as the availability of palatable food (2). The type of hunger which does not occur in response to prolonged food deprivation, but occurs because of a desire or drive to consume food for pleasure, has been called hedonic hunger. Hedonic hunger refers to the subjective state, not to actual food intake (3, 4). Psychological components of food reward include "liking," which is a hedonic reaction to pleasure, and "wanting," which reflects incentive motivation. "Liking" and "wanting" components of rewards are related to distinct neuroanatomical and neurochemical brain reward systems (5, 6). Hedonic eating motives could be driven by "liking," "wanting," or both (3). The hedonic impact of food, especially of sweet and fat food, is regulated by the μ-opioid system (7). Opioid μ-agonists are known to increase food intake, primarily by amplifying the hedonic properties of food (8).

The Power of Food Scale (PFS) has been developed to measure the appetite drive to consume palatable food, in food-abundant environments, at 3 levels of food proximity—namely, food available, food present, and food tasted (2, 9). The PFS does not contain questions related to actual food intake and does not measure overeating (3). Preliminary evidence

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suggests that the PFS reflects the strength of desire for palatable food in the absence of an energy deficit, which may in some circumstances lead to increased eating (4). In addition, the PFS tends to best predict intake of highly palatable food in people with weak inhibitory control (10–12). This also raises a question of whether hedonic hunger is associated with increased body weight. Several studies have been conducted to answer this question, and most of them have found no relation between BMI (in kg/m^2) and hedonic hunger (9, 13, 14). Conversely, Rabiei et al. (15) have recently shown a positive association between hedonic hunger and obesity in women. Their study shows that the relations between hedonic hunger, intake, and body mass have not yet been entirely characterized.

Our recent studies have focused on finding the determinants of high-fat food intake. We have shown that high fat preference and low fat restraint are associated with a greater intake of high-fat food (16), but this was unrelated to the ability to discriminate fat in food and polymorphism of *CD36*, which is responsible for fatty acid detection (17). We further hypothesized that variation in high-fat food intake may be explained by different responses to hedonic hunger. It should be highlighted, however, that intake of some types of high-fat food is driven by caloric needs, not by hedonic motivation.

Polymorphism of the opioid receptor mu 1 gene (*OPRM1*), which is a part of the aforementioned μ-opioid system, has been associated with fat intake in genome-wide association studies (18). A common variant of the *OPRM1* gene, rs1799971 (A118G), leads to the amino acid asparagine (N) being replaced by aspartic acid (D) at position 40 of the extracellular receptor region. This substitution affects endogenous opioid binding and receptor activity (19). Furthermore, the minor *OPRM1* allele (rs2281617) is positively associated with amygdala volume, but negatively with fat intake (18, 20). Because the amygdala is an important food intake regulator, its volume can affect dietary behavior (21). For these reasons *OPRM1* seems a good positional and functional candidate gene for an association study on high-fat food intake.

Taken together, we hypothesized that higher hedonic hunger is related to more frequent intake of high-fat food, which then contributes to increased body weight. We also assumed that the relation between hedonic hunger and intake of high-fat foods can be modified by *OPRM1* genotype.

Methods

Study design

Participants were enrolled in Poznań, Poland in the period 2016–2018. Because food intake and choice may depend on season, the study was conducted only during spring and fall. The subjects were women and men between 20 and 40 y of age, with BMI <25 and ≥25 kg/m^2. The research protocol was approved by the Local Ethics Committee (no. 966/15). All participants gave their written informed consent. Recruitment was conducted using online advertisements circulated through social media. Participants were in addition encouraged to mention the study to friends and family members, and to ask them to consider participating, which served as a snowball sampling technique. The exclusion criteria included chronic diseases (such as diabetes, metabolic syndrome, cancer, and hypothyroidism); recent dieting or consumption of a calorie-restriction diet; use of medications known to affect taste, body weight, lipid profile, or appetite; moderate or heavy smoking (>1 pack/wk); shift work; and being pregnant or lactating.

Eligible participants came in person to the Institute of Human Nutrition and Dietetics at Poznań University of Life Sciences, where all the procedures were conducted. **Supplemental Figure 1** presents the study flow of participants. During the first visit (day 1) anthropometric measurements and blood collection were performed. The mobile phones with the application for measuring frequency of high-fat food intake were given to the participants. They were also instructed in the use of this application and filled out the questionnaires, with the PFS among others. During the second visit the mobile phones were collected (day 10). Other procedures were also conducted, but those are not related to the present study.

Measurements

Height was measured to the nearest 0.5 cm. A Bod Pod (Cosmed) was used to determine body composition in line with the manufacturer's recommended procedure. Body weight was measured to the nearest 0.1 kg, using the calibrated scale included in the Bod Pod. Subjects wore a bathing suit and had fasted overnight. Height was measured with a stadiometer and recorded to the nearest 0.5 cm. BMI was calculated as kg/m^2.

The frequency of consumption of high-fat foods was measured with a validated application for mobile devices employing an ecological momentary assessment approach (22). Each participant received a smartphone that contained an app designed for this study and implemented by IT Generator, Poznań, Poland, and was monitored for 7 d. Participants were prompted at 09:00, 13:00, 17:00, and 21:00, with each prompt asking the participant whether they had eaten any food since the previous prompt. If the subject replied "yes," the subject was presented with a list of high-fat foods and instructed to choose all the food items that applied. The collected data were processed as previously described (22). Only valid responses were considered in the study, defined as when an individual gave replies to ≥3 prompts on ≥5 d. Based on that assumption, 91 participants were excluded from the initial sample of 421 individuals. The products were selected from a database of Polish food products (National Food and Nutrition Institute, Warsaw) based on having a fat content of ≥10% (g/g of product), resulting in a list of 37 high-fat food items. Weekly frequencies of consumption of all high-fat products were calculated by dividing the total consumption number of the reported food by the appropriate number of days (i.e., 5, 6, or 7, depending on the participant), and then transforming it into the times per week scale. We also categorized high-fat foods as healthy (avocado, oil, nuts), sweet (doughnuts, pastries, chocolates, cookies, bars), salty (potato chips, French fries, peanut butter), meat (bacon, cheeseburger, hamburger, hot dog, kabanos sausage, kebab, regular sausage, duck/goose meat, fried chicken, pork, salami, pâté), snacks (bars, chips, cookies, chocolate, doughnuts, nuts), and fast-food products (cheeseburger, hamburger, baked or fried sandwich, pizza, kebab, French fries, hot dog). The following products were not included in any of these categories and only contribute to the total weekly frequencies of consumption of all high-fat food: margarine, lard, butter, mayonnaise/salad sauce, fried fish, smoked salmon/eel/mackerel/halibut, egg yolk, cheese, processed cheese, baked or fried sandwiches, pancakes/crepes, potato pancakes, whipped cream, and cream.

Physical activity level was assessed using the short version of the International Physical Activity Questionnaire (23). Hedonic hunger was measured using the PFS (2, 9). This is a 15-item questionnaire presented on a 5-point Likert scale ranging from 1 (do not agree at all) to 5 (strongly agree). All items were then scored, with a higher score indicating a greater responsiveness to the food environment. Three scores were generated using the PFS: factor 1 (food available), factor 2 (food present), and factor 3 (food tasted). An aggregated factor was calculated as a mean of these 3 scores. We classified here high and low hedonic hunger categories, based on the median total PFS value.

Blood was collected into tubes containing EDTA, and the DNA was isolated from fresh blood using a NucleoSpin Blood kit (Macherey-Nagel). Genotyping of A/G rs1799971 in the *OPRM1* gene was performed using TaqMan probes (single tube assays, Thermo Scientific) on a LightCycler 480 instrument (Roche Diagnostics). PCR was run using a 10-μL reaction mix containing a Probe Master kit (Roche Diagnostics). Gene and genotype frequencies were calculated.

TABLE 1 Characteristics of the study participants[1]

Parameters	All ($n = 421$)	BMI < 25 kg/m^2 ($n = 208$)	BMI ≥ 25 kg/m^2 ($n = 213$)	P value
Baseline characteristics				
Age, y	27.7 ± 5.5	27.0 ± 5.4	28.0 ± 5.2	0.0607
Men/women, *n*/*n*	207/214	107/101	100/113	0.6252
BMI, kg/m^2	26.0 ± 5.3	21.9 ± 1.9	30.0 ± 4.5	<0.0001
Body mass, kg	78.6 ± 18.1	65.9 ± 9.5	90.8 ± 16.1	<0.0001
Hedonic hunger measures				
Total PFS	3.02 ± 0.71	2.91 ± 0.66	3.13 ± 0.73	0.0017
PFS factor 1[2]	2.76 ± 0.84	2.62 ± 0.79	2.88 ± 0.88	0.0030
PFS factor 2[2]	3.09 ± 0.96	2.87 ± 0.91	3.30 ± 0.96	<0.0001
PFS factor 3[2]	3.29 ± 0.80	3.28 ± 0.79	3.30 ± 0.80	0.8102

[1]Values are means ± SDs unless otherwise indicated. PFS, Power of Food Scale.
[2]PFS1, PFS2, and PFS3 are measures of hedonic hunger in the cases of food available, food present, and food tasted, respectively.

Statistical analysis

The data were presented as means ± SDs. Normality of the data was tested using the Shapiro–Wilk test. Correlations were evaluated using the Pearson correlation coefficient. Student's t test was used to compare crude means for people with BMI <25 kg/m^2 and people with BMI ≥25 kg/m^2. To study the associations between high-fat food intake, hedonic hunger, and *OPRM1* genotype, we used a multivariate linear regression model, which was adjusted for physical activity (low, moderate, high), BMI (<25 compared with ≥25), and sex. Based on the research framework, we simultaneously included in the multivariate models all the independent variables which could be associated with the tested outcomes. In analyzing the interaction effects, we used a linear regression model and in this case hedonic hunger was considered as categorical (low or high). The cutoff was established at the median total PFS value of 3.07. The interaction model was adjusted for sex, physical activity, and BMI. The OR of being overweight or obese was calculated using logistic regression and the models were adjusted for gender. Two types of linear and logistic regression models were considered. In model 1, the total PFS was included, whereas in model 2 we used subscales of the PFS. $P < 0.05$ was considered statistically significant. Moreover, associations between different tested factors were considered as separate hypotheses, to eliminate the need for a severe multiple comparison correction. Data were analyzed using Statistica software (StatSoft).

Results

Hedonic hunger scores and the frequency of eating high-fat food were normally distributed. Table 1 shows the group characteristics. *OPRM1* genotype and gene frequencies were as follows: AA, 0.42; AG, 0.46; GG, 0.12; and A, 0.65 and G, 0.35. Hedonic hunger measures differed between people with BMI < 25 kg/m^2 and those with BMI ≥ 25 kg/m^2 (Table 1). Moreover, hedonic hunger positively correlated with the frequency of consumption of high-fat food, especially in normal-weight people (**Supplemental Table 1**). For example, all 3 hedonic hunger measures were correlated with snack intakes.

We further investigated the associations between the intake of high-fat food, hedonic hunger, and *OPRM1* genotype using linear regression models. No associations were revealed between the total PFS score and the total intake of high-fat foods (Table 2), but the total PFS score was associated with the frequency of eating healthy and snack high-fat food. We next considered the 3 components of hedonic hunger separately: PFS1 was positively associated with healthy high-fat food intake and PFS2 with sweet food and fast-food intake. PFS2 was also negatively associated with healthy high-fat food intake. There were no associations found between the *OPRM1* genotype and the intake of high-fat food (Table 2). However, we found an interaction effect between *OPRM1* genotype and hedonic hunger on fast-food intake (β: −0.17; 95% CI: −0.32, −0.02; $P = 0.0246$). Among people with low hedonic hunger, the *OPRM1* genotype was not associated with fast-food intake. In contrast, in those with high hedonic hunger, the minor allele was associated with lower fast-food intake (β: −0.17; $P < 0.0154$) (**Figure 1**). No other interactions were detected.

Finally, we explored whether hedonic hunger, *OPRM1* genotype, and high intake of high-fat food were associated with being overweight or obese. The total PFS score, as well as PFS2, increased the chance of having a BMI ≥25 kg/m^2, whereas PFS3 decreased this. Neither *OPRM1* genotype nor intake of high-fat food contributed to being overweight or obese (**Table 3**).

Discussion

Our exploratory study has shown, to our knowledge for the first time, that components of hedonic hunger are related to the intake of selected types of high-fat food and that the aggregated hedonic hunger score and PFS2 (related to food present) are associated with increased BMI, whereas PFS3 (related to food tasted) is inversely associated with being overweight or obese.

The most important of these components was PFS2, which was positively associated with the intake of sweet high-fat food and fast food. In addition, the total PFS score was associated with intake of 2 groups of high-fat food: healthy and snack. Generally, there has been no agreement on the relation between hedonic hunger and food intake. Verhoeven et al. (24) showed, for example, that snacking is more related to habit than to hedonic hunger. Hedonic hunger was associated with the frequency of unhealthy snack intake, but only when habit strength was not controlled for. On the other hand, Schüz et al. (25) observed that higher PFS scores were associated with more everyday snacking. However, habits were not considered in that study, which may explain this discrepancy. Forman et al. (26) revealed that higher PFS scores were predictive of greater cravings for and consumption of chocolate. Furthermore, a study of Nansel et al. (27) found that hedonic hunger was positively associated with the intake of sweet or salty snacks and fast food. No firm conclusion has yet been made on hedonic hunger and food intake (4). It has been suggested that factors other than hedonic hunger may be needed to trigger consumption, or that self-control may be a protective factor which suppresses the impact of hedonic hunger. A recent study

TABLE 2 Results of linear regression models examining whether intake of high-fat food is associated with hedonic hunger and *OPRM1* genotype in participants aged 20–40 y[1]

Independent variables	Outcome: intake of different types of high-fat foods													
	All		Healthy		Sweet		Salty		Meat		Snack		Fast food	
	β (95% CI)	*P*	β (95% CI)	*P*	β (95% CI)	*P*	β (95% CI)	*P*	β (95% CI)	*P*	β (95% CI)	*P*	β (95% CI)	*P*
Model 1														
Total PFS	0.09 (−0.02, 0.21)	0.1189	0.12 (0.01,0.23)*	0.0389	0.08 (−0.03, 0.19)	0.1893	0.10 (−0.01, 0.22)	0.0738	0.06 (−0.04, 0.17)	0.2610	0.16 (0.04,0.27)*	0.0066	0.06 (−0.05, 0.17)	0.3106
OPRM1	−0.08 (−0.20, 0.03)	0.1541	0.01 (−0.10, 0.11)	0.8826	−0.07 (−0.18, 0.04)	0.2280	−0.05 (−0.16, 0.06)	0.3956	−0.02 (−0.13, 0.09)	0.7557	−0.07 (−0.18, 0.04)	0.1985	0.07 (−0.04, 0.18)	0.1953
R^2	0.02		0.07		0.04		0.03		0.09		0.06		0.06	
R^2_{adj}	0.01		0.05		0.02		0.01		0.07		0.04		0.04	
Model 2														
PFS1	0.05 (−0.10, 0.19)	0.5270	0.27 (0.13,0.41)*	0.0001	−0.08 (−0.23, 0.05)	0.2245	−0.01 (−0.15, 0.14)	0.9174	−0.01 (−0.15, 0.13)	0.8984	0.07 (−0.08, 0.21)	0.3606	−0.04 (−0.18, 0.10)	0.5678
PFS2	0.11 (−0.03, 0.26)	0.1217	−0.25 (−0.39, −0.12)*	0.0003	0.21 (0.07,0.35)*	0.0030	0.12 (−0.02, 0.26)	0.1078	0.13 (−0.01, 0.27)	0.0640	0.10 (−0.04, 0.24)	0.1574	0.17 (0.03,0.31)*	0.0159
PFS3	−0.04 (−0.18, 0.10)	0.5505	0.10 (−0.03, 0.23)	0.1450	−0.02 (−0.15, 0.12)	0.8175	0.02 (−0.11, 0.17)	0.7216	−0.03 (−0.17, 0.10)	0.6113	0.03 (−0.11, 0.17)	0.6992	−0.04 (−0.18, 0.09)	0.4933
OPRM1	−0.07 (−0.19, 0.00)	0.2088	0.01 (−0.10, 0.12)	0.8672	−0.07 (−0.18, 0.04)	0.2371	−0.05 (−0.16, 0.07)	0.4000	−0.01 (−0.12, 0.10)	0.8330	−0.07 (−0.18, 0.04)	0.2273	0.08 (−0.03, 0.19)	0.1658
R^2	0.03		0.14		0.07		0.03		0.10		0.06		0.08	
R^2_{adj}	0.01		0.11		0.04		0.01		0.07		0.03		0.05	

[1] $n = 330$. The regression models were adjusted for physical activity (as a categorical variable), group (BMI <25 and ≥25 kg/m^2), and gender. *OPRM1*, opioid receptor mu 1 gene; PFS, Power of Food Scale; R^2_{adj}, adjusted R squared.
*Statistically significant results, $P < 0.05$.

showed that higher PFS scores but combined with lower self-control capacities were correlated with higher intakes of high-fat salty snack foods and high-sugar foods, higher overeating frequency, and higher snacking frequency (11). Our study and several recent studies have shown that whether hedonic hunger actually stimulates consumption may also depend on the type of food; also, an association between hedonic hunger and intake primarily of foods with the least nutritional value has been suggested (27). Intake of high-fat products may be driven by different factors (17) and, as shown here, hedonic hunger is associated with the intake of only specific types of high-fat food, i.e., sweet high-fat food and fast food, which is in line with the assumptions of the PFS. It should again be emphasized that the consumption of most food items in everyday life is driven by homeostatic, and not hedonic, hunger. Indeed, we did not find any association between hedonic hunger and meat or total intake of high-fat food. An interesting situation occurred with healthy high-fat food: PFS1 (related to food available) was positively associated with its intake, whereas PFS2 was negatively associated with its intake. This may suggest that the proximity of this type of food plays a role in its actual intake, possibly because of a conflict between the goals of healthy eating and eating energy-dense food.

Relations between hedonic hunger and food intake may also be modified by genotype. This aspect has been examined by only 1 study that we know of (28). In that study, and also in ours, *OPRM1* was examined as a functional candidate. This gene encodes a protein which is involved in the endogenous opioid system, which regulates pain, reward, and addictive behaviors. In the present study, we found no relation between *OPRM1* polymorphism (rs1799971) alone and high-fat food intake, but for the first time we described an interaction between the variants of *OPRM1* and hedonic hunger: namely, in people with high hedonic hunger only, the minor allele (G) was associated with lower fast-food intake. The direction of the association between *OPRM1* polymorphism and fatty food intake was as we expected on the basis of previous studies. Davis et al. (28) reported that the GG group had a higher preference for sweet and fatty foods than the other 2 genotype groups. Similarly, the minor allele of rs2281617 had a "protective" effect, because it was associated with 4% lower fat intake, ~2-kg lower body mass, and higher amygdala volume (18). Both rs1799971 and rs2281617 affect μ-opioid receptor density and binding potential (18, 19). For this reason, similar effects of these 2 polymorphisms on phenotype can be expected. Moreover, the results of human studies are in line with those of animal studies. It has long been known that μ-opioid agonists suppress intake, especially of palatable food (8). All these studies showed that the minor allele of *OPRM1* rs1799971 leads to a decrease in fat intake.

Our study also showed that hedonic hunger measures were associated with BMI. PFS2 increased the chance of being overweight or obese, whereas PFS3 decreased it. The odds of being overweight or obese approximately doubled for each unit of PFS2. The majority of past studies did not find any relation between BMI and hedonic hunger (4), and the reports that are available are inconclusive on this point (29). Recently, Rabiei et al. (15) in a case–control study ($n = 140$) found an association between hedonic hunger and obesity in women. A positive association was also found in a Portuguese population (29). PSF2 was positively associated with belonging to the clinical sample, which included people with BMI ≥ 30 (OR: 1.8; 95% CI: 1.2, 2.8; $P = 0.008$). Also, PFS2 was associated with being obese in a population sample (OR: 2.1; 95% CI: 1.6, 2.7;

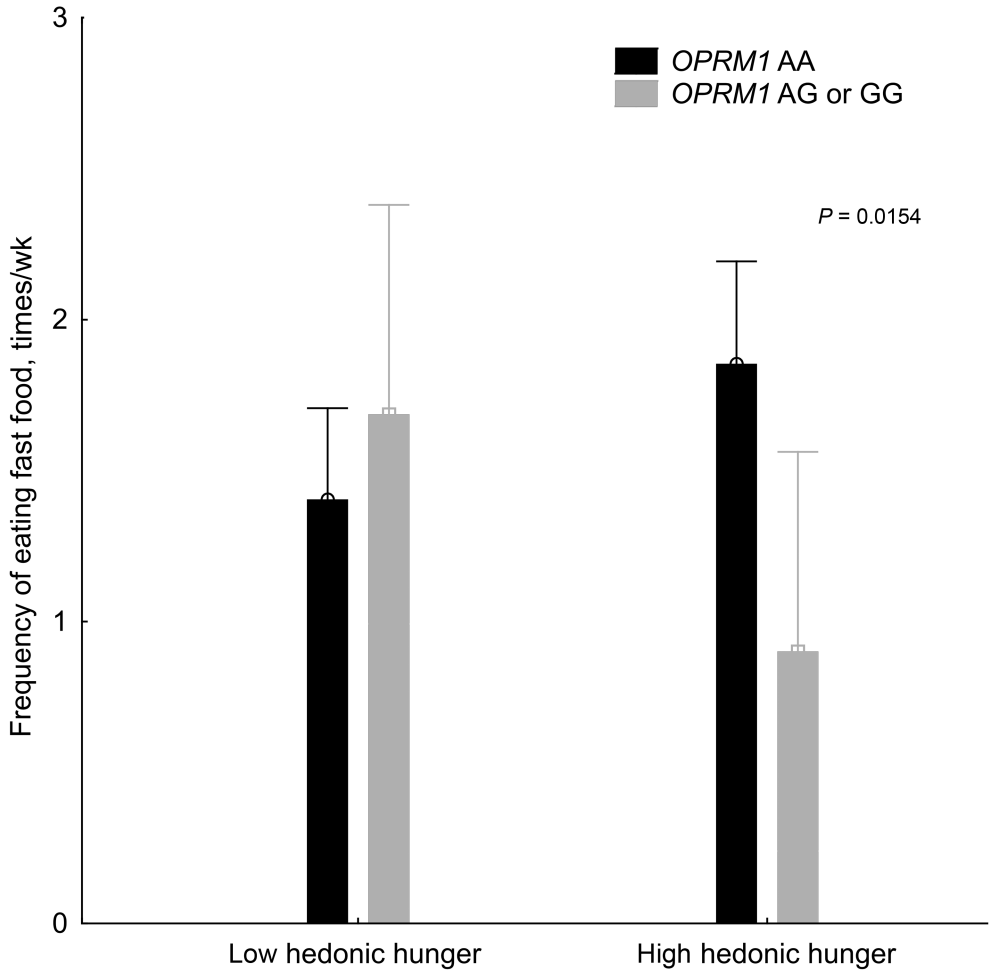

FIGURE 1 Interaction between *OPRM1* and hedonic hunger on high-fat food intake (linear regression model) in people aged 20–40 y. Low hedonic hunger was defined as a total PFS score below the median value (3.07), and high hedonic hunger was defined as a total PFS score equal to or above the median value. The results are shown as adjusted means and their 95% CIs. Means were adjusted for sex, physical activity (as a categorical variable), and BMI group (<25 compared with ≥25 kg/m^2). *OPRM1*, opioid receptor mu 1 gene; PFS, Power of Food Scale.

$P < 0.001$). Moreover, in that study, as in ours, total PFS, PFS1, and PFS2 values—but not PFS3—differed by BMI (30). Interestingly, we showed that PFS3 was inversely associated with being overweight or obese. Although this result might seem incorrect at first, our findings on the relations of PFS2 and PFS3 with body weight are in line with previous research, showing that obesity is associated with increased motivation to eat, rather than with pleasure while eating (31, 32). Similar associations between components of hedonic hunger and obesity have been demonstrated by Schultes et al. (30). There were no differences between normal-weight and obese patients in PFS3, and gastric bypass patients had significantly lower scores than did the nonobese control subjects. Taken together, it seems that PFS2 tends to have a positive relation with BMI, whereas PFS3 has a negative relation with BMI, which may explain the nonconcordant results concerning relations between BMI and hedonic hunger.

TABLE 3 Multivariate logistic regression of factors associated with being overweight or obese in people aged 20–40 y[1]

Variables	Adjusted OR (95% CI)[2]	*P*
Model 1		
Total PFS	1.43 (1.03, 2.01)	0.0335
OPRM1, minor allele	0.79 (0.42, 1.46)	0.4454
High-fat food intake	0.99 (0.97, 1.01)	0.5538
Model 2		
PFS1[3]	1.17 (0.82, 1.68)	0.3779
PFS2[3]	1.89 (1.37, 2.61)	0.0001
PFS3[3]	0.61 (0.41, 0.87)	0.0082
OPRM1, minor allele	0.68 (0.36, 1.29)	0.2346
High-fat food intake	0.99 (0.97, 1.01)	0.3631

[1] $n = 330$. *OPRM1*, opioid receptor mu 1 gene; PFS, Power of Food Scale.
[2] The regression models were adjusted for gender.
[3] PFS1, PFS2, and PFS3 are measures of hedonic hunger in the cases of food available, food present, and food tasted, respectively.

This is the first study that we know of to examine hedonic eating and the intake of different types of high-fat food measured in a real-time manner. Moreover, the study considered the role of polymorphisms of the *OPRM1* gene in the relation between hedonic hunger, high-fat food intake, and BMI. On the other hand, one limitation of this study is that the food-frequency approach we used to estimate high-fat food intake may fail to accurately reflect the amount of food consumed. Generally, using applications for mobile devices in research can pose challenges, including technical problems with using the app, which we observed occasionally. Also, the compliance rate in our study was moderate—we were able to use data from 330 out of 421 individuals—which may have produced bias toward

the null and reduced our power to detect differences. We also did not perform correction for multiple testing, because our study is exploratory in nature. Moreover, the conclusions cannot be generalized to the overall population, because we excluded smokers and people on weight-reduction diets, among others.

In conclusion, intake of healthy and sweet high-fat food, as well as snacks and fast food, is associated with hedonic hunger in people aged 20–40 y. *OPRM1* genotype can modify the relation between hedonic hunger and fast-food intake. Its minor allele is associated with lower fast-food intake in people with higher hedonic hunger. The aggregated hedonic hunger score and PFS2 are associated with increased BMI, whereas PFS3 is inversely associated with being overweight or obese.

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Data Availability

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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