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Reward Capacity Predicts Leptin Dynamics During Laboratory-Controlled Eating in Women as a Function of Body Mass Index

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Abstract

Objective—The role of leptin in mesolimbic signaling non-food-related reward has been well established at the pre-clinical level, yet studies in humans are lacking. The present investigation explored the association between hedonic capacity and leptin dynamics, and whether this association differed by BMI class.

Methods—In this cross-sectional study of 75 women (42 with lean BMIs, 33 with obese BMIs), we measured serum leptin before/after meal consumption. Reward capacity was assessed using the Snaith-Hamilton Pleasure Scale (SHAPS). Multiple regression tested whether reward capacity was associated with leptin AUC, with an interaction term to test differences between lean (LN) and obese (OB) groups.

Results—The interaction of SHAPS by BMI group was robust (β=−.40, p=.005); among women with obesity, greater SHAPS score was associated with lower leptin AUC (β=−.35, p=.002, adjusted R-squared=.66). Among the lean group, the association was not statistically significant (β=−.16, p=.252, adjusted R-squared=.22). Findings were above and beyond BMI and age.

Conclusions—In this sample a robust, negative association between reward capacity and circulating leptin was stronger in women with obesity compared to lean counterparts. These findings suggest that despite likely leptin resistance, inhibitory leptin functioning related to non-food reward may be spared in women with obesity.

Keywords

leptin; reward capacity; pleasure; body mass index; women

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Introduction

Eating motivated by pleasure has distinct affective, behavioral, and clinical correlates compared to eating motivated by other factors, such as for coping with negative affect (1). By some accounts, differences in food-related pleasure thresholds between people with and without obesity may contribute to differences in food consumption (2). One underlying mechanism may be that people with obesity—compared to their leaner counterparts—experience blunted pleasure, and are less sated by comparable food consumption.

The neurobiological mechanisms supporting these divergent phenotypes include an number of hormone, cytokine, and peptide systems involved in appetite (e.g., leptin, ghrelin, orexins, insulin) that are sensitive to the reward value of a given food (3). Emerging evidence indicates a distinct inhibitory role of leptin, an adipocytokine, on reward signaling in mesolimbic regions, facilitated in part through expression of leptin receptors on dopaminergic (DA) neurons in the ventral tegmental area (VTA) (4). Pre-clinical studies report reduced dopaminergic (DA) neuron firing following peripheral leptin administration (4) and that leptin attenuates DA activity in response to food cues which is reversed by peripheral leptin pre-treatment (5). People with obesity who also have genetic leptin deficiency exhibit elevated ventral striatal activity in response to food cues which is normalized by leptin treatment (6). These findings indicate that leptin plays a role in inhibiting DA-modulated signaling in reward regions, and thus could be involved in reward processing at a broader level than food. However, to our knowledge, evidence for the association between general hedonic capacity and leptin, and the extent to which these factors might be divergently related according to BMI class, has not been reported.

The current study was designed to examine the association between self-reported (non-food-related) hedonic capacity and peripheral leptin dynamics in women characterized as with obesity or without (i.e., lean). We hypothesized that in light of leptin's inhibitory effect on reward signaling, ratings of reward capacity would be inversely associated with changes in leptin following a meal. Furthermore, considering evidence suggesting unique coupling between peripheral leptin and mesolimbic reward activity in people with obesity (7), we expected that this association would be stronger and potentially restricted to women with BMIs in the obese range.

Methods

We studied 75 women between 21 and 43 years of age: 42 in the lean BMI range (LN group; BMI: M=22.3 kg/m^2, SD=1.75; age: M=27.6, SD=3.41) and 33 in the obese range (OB group; BMI: M=37.9 kg/m^2, SD=5.87; age: M=31.3; SD=6.30), recruited from the Boston, Massachusetts area through advertisements. Exclusion criteria included current pregnancy or breastfeeding, neurological disease, diabetes, endocrine disorders, current major psychiatric disorders, significant change in weight within the last three months, current use of glucocorticoids, steroids, neuroleptics or stimulants. All participants provided written informed consent. All study procedures were approved by the Partners Healthcare Human Research Committee.

Following a screening visit at the Brigham and Women's Hospital Center for Clinical Investigation to ascertain height, weight, and other clinical variables, eligible participants were brought in for a main study visit during the follicular phase (days 1–10) of their menstrual cycle; participants (n=11 LN; n=7 OB) on oral contraceptives completed study procedures during the inactive (placebo) week of their regimen. On the morning of the visit, following an overnight (12 hr) fast, a saline-lock IV line was placed in the participant's non-dominant arm to enable serial blood draws. Participants completed other study procedures (not relevant to the current study), then were asked to consume a standardized meal, preceded and followed by blood draws. The meal contained 30% of the recommended daily caloric intake (varying according to each participant's basal metabolic rate/activity level, measured by the Harris-Benedict equation; with 18% calories from protein, 23% calories from fat, and 59% calories from carbohydrates). Blood samples were ascertained immediately prior to the meal (Time 0; T0), and 30 (T30), 60 (T60), and 120 (T120) minutes following meal commencement.

Following the meal, participants completed the Snaith-Hamilton Pleasure Scale (SHAPS) (8), a 14-item questionnaire measuring degree of pleasure experienced by the individual in the recent days on a 4–point Likert-type scale. Item examples include: "I would find pleasure in my hobbies and pastimes," "I would be able to enjoy a beautiful landscape or view," and "I would enjoy seeing other people's smiling faces." The alpha for the scale in this sample was .87. To reduce the possibility that associations with leptin AUC would be driven solely by food-related items, the three with food-, olfactory-, or gustatory-related content ("I would be able to enjoy my favorite meal," "I would find pleasure in the scent of flowers or the smell of a fresh sea breeze or freshly baked bread," and "I would enjoy a cup of tea or coffee or my favorite drink") were deleted. The reliability of this amended scale was .85, demonstrating strong construct validity. Thus we used the 11-item version to reduce the chance that potential associations would be driven by the food-related items. To capture the broadest extent of variability in responses, scores were calculated by summing the individual item responses (rather than the dichotomous scoring traditionally used for the SHAPS) and dividing by the number of items, yielding a minimum score of 1.0 (indicating low reward capacity) and a maximum of 4.0 (indicating high reward capacity), an approach used previously in assessing reward capacity in clinical and non-clinical populations (9).

Approximately 10–15 cc of blood was sampled at each time point, allowed to clot for 45–60 min, spun, aliquoted, and stored frozen at −80°C. Serum leptin was analyzed in duplicate with a commercial radioimmunoassay kit (LINCO Research, St. Charles, MS; inter-assay CV: 3.2–8.9%; intra-assay CV: 5.2–7.5%). Area under the curve for leptin was calculated using the trapezoidal method.

We computed means, standard deviations, skew, and kurtosis by BMI group (i.e., LN and OB). Differences between LN and OB groups on demographic (age, BMI, race/ethnicity, years of education) and primary variables of interest (SHAPS score, leptin AUC) were tested using Pearson X^2 or independent samples t-tests. Effect size differences between groups were estimated using Hedges' g (10), which is a less biased estimator than Cohen's d when —as is the current sample—sample sizes of groups are unequal, relatively small, and standard deviations between the group differ (11). Given age differences by BMI group, age

was retained as covariate in multivariate models. To minimize residual confounding, we included continuous BMI as an additional covariate. We inspected correlations among the study variables for LN and OB groups. Multiple regression modeling tested whether reward capacity (operationalized as the mean score on the SHAPS questionnaire) was associated with metabolic hormone dynamics (leptin AUC). The interaction term of reward capacity multiplied by BMI group tested whether the association was different for LN v. OB groups. We reported confidence intervals and effect size estimates (12). Data were analyzed using SPSS 23 (IBM Corp., Armonk, NY).

Results

Women in the lean v. obese range had lower BMIs and were slightly younger (see Table 1). Groups did not differ significantly ($p<.05$) on race (LN: 78.6% White, 7.1% Black, 9.6% Asian, 4.8% other; OB: 78.8% White, 12.1% Black, 3.0% Asian, 6.1% other), ethnicity (LN: 88.1% non-Hispanic; 11.9% Hispanic; OB: 93.9% non-Hispanic, 6.1% Hispanic), or educational attainment (LN: Less than an Associate degree, 4.8%, Associate degree or more, 95.1%; OB: Less than an Associate degree or less, 15.6%, Associate degree or more, 84.4%). There was no difference between groups in the proportion of women using hormonal contraceptives (LN: 11 out of 42; OB: 7 out of 33; $X^2=.25$, p=.62).

Leptin and BMI were positively correlated, more strongly so for OB (r=.75, p<.001) than LN (r=.50, p=.001). LN and OB groups did not differ on levels of reward capacity, but did differ in the associations of reward capacity with leptin AUC. Among the LN group, reward capacity was not statistically significantly associated with leptin AUC (r=−.15, p=.33). For women with obesity, higher reward capacity was correlated with lower leptin AUC (r=−.38, p=.03) in the laboratory task. Age was not correlated with any of the other variables in this sample for LN or OB groups.

The interaction of SHAPS by BMI group was robust (β=−.40, B=−2352, SE=805, p=.005, CI [−3959, −746]); among women with obesity, greater SHAPS score was associated with lower leptin AUC; among lean women, there was no statistically significant association. Findings were above and beyond BMI and age. Table 2 presents regression analyses stratified by BMI group. The key finding is that among women with obesity, greater reward capacity was associated with lower leptin AUC; this association did not hold among lean women (see Figure 1).

Discussion

In the present study, we demonstrate a novel association between leptin dynamics and general reward capacity in women, and provide preliminary evidence that this association is unique to women with BMIs in the obese range. Reward capacity was significantly associated with leptin changes in response to a meal in women with obesity, but not in lean women. This association remained robust in the group of women with obesity even after accounting for BMI and age. To our knowledge, these findings are the first to suggest that the inhibitory effect of leptin on reward processing in humans may extend beyond food-related hedonic experience to encompass pleasure associated with everyday situations.

Recent pre-clinical reports provide emerging mechanistic evidence for the role of leptin in reward functioning beyond food-related hedonic capacity. Central or peripheral leptin administration significantly attenuates cocaine-mediated reward processing (13), while obese, leptin-deficient ob/ob mice exhibit abnormal responses to standard reward tasks and to drugs of abuse (cocaine, opioids, and ethanol) compared to wild-type mice (14), suggesting leptin's blunting of reward capacity may extend to non-food substances and other primary rewards. The extent to which these functions systematically differ according to fat mass have not been well-explored in humans. Although leptin resistance (the inability of leptin to inhibit food intake and stimulate energy expenditure, despite elevated levels) is commonly manifested in obesity, the mechanisms of leptin resistance and specificity to various CNS targets remain to be fully elucidated. For example, exogenous vs. endogenous leptin appear to produce differential leptin resistance (15), a phenomenon which may be further promoted or inhibited by exposure to various dietary conditions (high-fat, low-fat, chow, etc.) (16). Further, some aspects of leptin action appear uniquely spared, including cardiovascular sympathetic effects (17, 18), possibly due to selective resistance to leptin in particular cell types, cellular and molecular signaling cascades following leptin receptor (LepR) binding, differential action on certain brain regions (19), altered free:bound leptin in circulation in obesity (20) resulting in differential desensitization, and reduced leptin transport across the blood brain barrier in obesity (21) [for a recent full review, see (22)]. Among the contributing factors to differential or selective leptin resistance which lend insight into the current data, recent evidence suggests that although diet-induced obesity (DIO) in rats is associated with leptin resistance as quantified by unchanged mRNA expression of POMC and NPY in the arcuate nucleus following ICV leptin administration, mesolimbic brain regions (including the VTA and NAcc) remain sensitive to leptin in DIO, but not in lean rats (23). These results suggest the intriguing possibility that leptin-induced food-related reward signaling is intact in DIO, although generalization of these findings beyond food-related appetite and reward in rodent models remain to be elucidated.

In line with these basic data, current findings demonstrating that our sample of women with obesity exhibited the expected inverse relationship between leptin AUC and reward capacity suggest that leptin inhibition of non-food rewards may be selectively spared, although with potentially negative implications for this population. Further studies on the mechanism behind the interactions between fat mass, peripheral leptin levels, and reward capacity, and the degree to which this impacts general well-being in individuals with obesity would assist in elucidating the complex systemic effects of leptin in humans. Additionally, given the complex role of catecholamines, such as dopamine, in immune responses (24), and recent data suggesting that obesity may be characterized by neuroinflammation (15, 16), investigation of the interactions between leptin, dopamine signaling, and peripheral and central inflammatory mediators would strengthen the understanding of the degree to which consumption of highly-rewarding, palatable diets (i.e., high-sugar, high-fat) predispose certain individuals to obesity, neuroinflammation in homeostatic and hedonic appetite circuitry, and impaired general reward capacity.

Major strengths of this study include a focus on the leptin-reward capacity association in human subjects; a well-controlled laboratory setting; and novel use of a well-validated assessment of reward capacity (SHAPS) to examine non-food reward in humans. We were

limited by a cross-sectional design, which prevents causal inferences, restriction to female participants, and reliance on BMI rather than a more precise measurement of fat and lean mass, which would be more closely linked to leptin levels. Additionally, as the SHAPS is a self-report questionnaire, it may represent a biased measure of reward capacity. However, because reward capacity is a subjective phenomenon, starting with a self-report measure is a sensible choice. Future research could benefit from including an implicit measure of reward capacity. We also acknowledge that other factors, including but not limited to genetics, neural circuitry sensitivity to various non-food rewards, recent exercise, and current mood, for example, may influence non-food reward capacity. As these were not addressed in the current study, we cannot exclude these as potential confounders.

In conclusion, here we report for the first time a robust inverse relationship between hedonic capacity and leptin dynamics in women with BMIs in the obese range. In addition to testing for replication, future studies may benefit from utilizing longitudinal designs, including male participants to explore sex and gender differences, employing other measures of general hedonic capacity (e.g., more objective or more implicit), objectively measuring fat and lean mass, and exploring potential mediators of the identified effects. Finally, given the social and clinical implications of these findings—suggesting individuals with obesity who exhibit elevated leptin are vulnerable to reduced ability to experience pleasure—once confirmed in other samples, additional investigations focused on leptin-based therapies and their effects on reward capacity in individuals with obesity are needed to mitigate the potential harmful long-term consequences of reduced reward capacity on overall well-being and quality of life.

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Acronyms

BMI	body mass index
LN	lean
OB	obese
SHAPS	Snaith-Hamilton Pleasure Scale
AUC	area under the curve
DA	dopamine
VTA	ventral tegmental area

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Study Importance Questions

1) What is already known about this subject?

- Leptin decreases food intake in animal models and in humans, partially through influencing reward processing around food.
- Pre-clinical data suggest leptin inhibits dopamine signaling and may influence non-food reward.
- Similar studies in humans, linking leptin and non-food reward, have not been reported.

2) What does your study add?

- Regression modeling shows statistically significant inverse associations between general reward capacity and postprandial leptin.
- This relationship was specific to women with BMIs in the obese range.
- Leptin inhibition may extend beyond food-related hedonic experience to encompass pleasure associated with other daily associations.

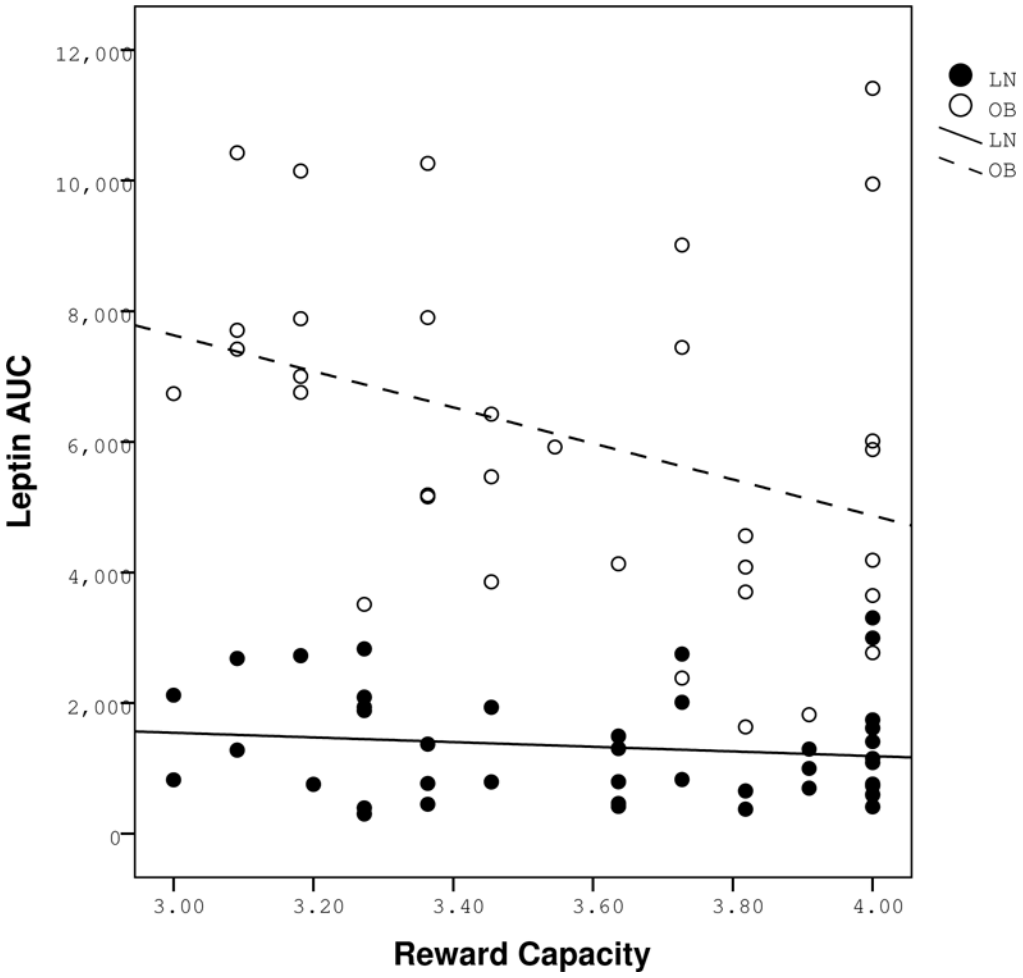

Figure 1. Effect of Reward Capacity on Leptin AUC is Modified by BMI Group

Scatterplot demonstrating the relationship between postprandial serum leptin levels (Leptin AUC) and SHAPS score (Reward Capacity), with data markers and fit lines stratified by group according to the legend.

Table 1

Descriptive Statistics

	Lean (n = 42)				Obese (n = 33)							
Variable	***M***	***SD***	**Min**	**Max**	***M***	***SD***	**Min**	**Max**	***t***	***p***	**95% CI [LL, UL]**	**Hedges' *g***
Predictor												
Reward Capacity	3.62	0.34	3.00	4.00	3.56	0.34	3.00	4.00	.67	.51	[−0.11, 0.21]	−0.18
Outcome												
Leptin AUC	**1325**	**831**	**303**	**3305**	**6073**	**2628**	**1635**	**11421**	**−9.92**	**<.001**	**[−5710, −3785]**	**2.57**
Covariates												
Age	**27.6**	**3.4**	**22**	**38**	**31.3**	**6.30**	**21**	**43**	**−3.05**	**.004**	**[−6.15, −1.26]**	**0.76**
BMI	**22.3**	**1.8**	**19.2**	**25.6**	**37.9**	**5.87**	**30.1**	**52.0**	**−14.73**	**<.001**	**[−17.7, −13.4]**	**3.78**

Note. **Bold** indicates non-overlapping confidence intervals of mean differences in values of lean (LN) v. obese (OB) participants. LL = lower limit; UL = upper limit. Positive g values indicate larger values for OB v. LN women in this sample. Variables for which Levene's test for equality of variances showed $p < .05$, the *t*-value not assuming equal variances in comparing OB v. LN s was used (i.e., Leptin AUC, age, and BMI).

Table 2

Reward Capacity Associated with Leptin AUC during Laboratory-Controlled Eating, Stratified by BMI Status

Outcome = Leptin AUC										
Lean	**Reduced**					**Full**				
Predictor Variable	***B***	***SE***	**β**	**CI [LL, UL]**	***p***	***B***	***SE***	**β**	**CI [LL, UL]**	***p***
Age	10.0	34.9	.041	[−60.6, 80.6]	.78	13.7	34.9	.06	[−57.0, 84.4]	.697
BMI	**240**	**68.0**	**.51**	**[102, 377]**	**.001**	**243**	**67.7**	**.51**	**[106, 380]**	**.001**
Reward Capacity	—	—	—	—	—	−394	338	−.16	[−1079, 292]	.252
Adjusted R-Squared	.21					.22				
Obese	**Reduced**					**Full**				
Predictor Variable	***B***	***SE***	**β**	**CI**	***p***	***B***	***SE***	**β**	**CI**	***p***
Age	−31.0	50.3	−.074	[−134, 72]	.54	−52.2	43.8	−.13	[−142, 37]	.24
BMI	**336**	**54.0**	**.75**	**[226, 446]**	**<.001**	**333**	**46.5**	**.74**	**[238, 428]**	**<.001**
Reward Capacity		—	—	—		**−2757**	**816**	**−.35**	**[−4426, −1088]**	**.002**
Adjusted R-Squared	.54					.66				

Note. **Bold** indicates statistically significant parameters. LL = lower limit; UL = upper limit.