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Kirandeep Kaur
Brigham Young University

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Does Dietary Behavior Mediate the Association Between Hedonic Hunger
and BMI in Overweight/Obese Adolescents?

Kirandeep Kaur

A thesis submitted to the faculty of
Brigham Young University
in partial fulfillment of the requirements for the degree of
Master of Science

Chad Jensen, Committee Chair
Kat Green
Lori Spruance

Department of Psychology
Brigham Young University

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ABSTRACT

Does Dietary Behavior Mediate the Association Between Hedonic Hunger and BMI in Overweight/Obese Adolescents?

Kirandeep Kaur
Department of Psychology, BYU
Master of Science

Heightened reward associated with palatable food, a construct referred to as hedonic reward, can promote excessive energy intake among adults. However, no known studies have examined the influence of hedonic reward on adolescents' eating behavior and weight status. The present study examined whether there was an association between hedonic hunger and weight status in overweight/obese adolescents and whether dietary behavior (caloric consumption) mediated this association. Baseline measures of body mass index, hedonic food reward, and dietary intake were collected from one hundred overweight and obese adolescents. Data were analyzed using mixture modeling. Mediation at varying levels of hedonic hunger was explored and three heterogeneous sub-classes were identified. Results indicated that for 65% participants there was a positive association between hedonic hunger and zBMI such that one unit of increase in hedonic hunger was associated with a 0.35 unit increase in zBMI. However, no conclusive evidence of caloric intake mediating the association between hedonic hunger and weight-status was found. Overall, our results suggest that exaggerated hedonic responses are associated with higher body mass in adolescents. These results provide a compelling argument that hedonic hunger can potentially override the homeostatic need for energy and may be associated with weight-gain.

Keywords: dietary behavior, hedonic hunger, body mass index, overweight adolescents

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Does Dietary Behavior Mediate the Association Between Hedonic Hunger and BMI in Overweight/Obese Adolescents?

The prevalence of pediatric obesity has risen in recent decades and has become a matter of grave concern. According to US Centers for Disease Control and Prevention (CDC, 2011), child and adolescent obesity is defined as a body mass index (BMI) at or above the sex-specific 95th percentile on BMI-for-age growth charts. In the United States, prevalence of obesity among adolescents has increased, an age wise trend analysis (2-19 years) over a 25-year period (1988 through 2014) estimates the prevalence of obesity in 2011-2014 as 17.0% (Ogden et al., 2016). Childhood obesity can lead to a variety of serious health consequences including early risk adult morbidity and mortality, premature death, type 2 diabetes, lipedema, cardiovascular diseases, asthma, sleep apnea, lower self-esteem, and psychological and social stress (Dev, McBride, Fiese, Jones, & Cho, 2013).

Childhood obesity is a complex condition, influenced by genetics, nutrition, physical activity, and environmental factors (Gurnani, Birken, & Hamilton, 2015). Eating behavior in humans is complex processes regulated by internal homeostatic processes, environmental, and social influences (Cappelleri et al., 2009). Homeostatic eating defines hunger as a biological state of acute energy deprivation and reflects an actual or approaching state of energy deprivation (Lowe & Butryn, 2007). Adaptations for survival motivated people to eat only when hungry. Schachter (1971) proposed that eating behavior of normal weight individuals are regulated by internal hunger and satiety cues whereas, for the obese individuals, food-relevant cues such as the time of the day (e.g., dinner time) or external cues like sight and the smell of palatable food is more influential in regulating eating behavior. Numerous behavioral and psychological factors have been shown to increase vulnerability to gain weight. Specifically, eating behavior patterns,

sensitivity to hedonic cues, the strength of hunger, and neurologic responses to rewarding properties of food or preference of foods have been shown to impact one's vulnerability to gain weight (Lipsky et al., 2016). Such dispositions can manifest through a behavioral or physiological process which promotes directs change of behavior (Blundell & Finlayson, 2004). However, homeostatic hunger models have ignored environment's role in predicting one's propensity to eat (Johnson, 2013).

If only governed by homeostatic mechanisms, humans would avoid overeating behaviors and each would be at their ideal weight (Saper, Chou, & Elmquist, 2002). In modern times, well-nourished populations are consuming food for hedonic reasons rather than acute energy deprivation (Lowe & Butryn, 2007). It appears that eating for pleasure, a behavior often referred to as hedonic hunger, and not the need of calories may drive excess consumption of food. Lowe and Levine (2005) defined hedonic hunger as a subjective state of pleasure experienced when consuming high-energy foods which produce eating in the absence of hunger. Hedonic models of hunger explain that "environment" is the catalyst for eating when not hungry, and people will often eat simply because the food is available, even in the absence of homeostatic hunger (Birch, Fisher, & Davison 2003; Johnson, 2000; Lowe & Levine 2005; Painter, Wansink, & Hieggelke, 2002). Similarities are drawn between eating for pleasure and compulsive gambling. Just as compulsive gamblers are occupied with the thoughts and urges of indulging in gambling even in the absence of cues, so are some individuals who are occupied with the urges, feelings and thoughts of eating, even when not hungry (Lowe & Butryn, 2007). Furthermore, the obesogenic environment is full of inexpensive food, which decreases the cost associated with hedonic eating (Papies, Stroebe, & Aarts, 2007) and environmental factors have also restricted opportunities for physical activity (Snoek, Van Striem, Janssens, & Engels, 2007). Hedonic hunger also explains

why many individuals experience frequent thoughts, feelings, and urges about food even when they are not hungry and these experiences may or may not have occurred due to exposure to food-related cues (Lowe & Butryn, 2007; Papies et al., 2007).

Understanding the influence of hedonic eating on adolescent's eating behavior is an important research aim because this construct has potential to contribute to obesity and other health complications in adolescents (De Cock et al., 2016). Adolescence is often marked by unhealthy eating habits, such as low consumption of dairy products, fruit, vegetables, and grains, and a high intake of energy-dense snacks, and overconsumption of sugar-sweetened beverages. Adolescent's quality of diet declines with a decrease in the consumption of fruits, vegetables and milk (Campbell et al., 2007; Murray et al., 2015). Moreover, overweight adolescents are less likely to eat breakfast and they may skip meals, as a way of managing their weight (Boutelle, Neumark-Sztainer, Story, & Resnick, 2002). Living in an obesogenic environment is especially challenging for adolescents as they learn to manage increased autonomy over eating choices (Basset, Chapman, & Beagan, 2008; Stok et al., 2015). Additionally, adolescents with sensitivity to environmental food cues report higher unhealthy snack consumption and frequent use of self-regulatory strategies designed to reduce unhealthy snack intake (Stok et al., 2015).

This line of research suggests that hedonic hunger may predict motivation for and consumption of high-energy foods. Hedonic eating may explain, in part, why weight management interventions have been minimally effective in achieving long-term weight loss among obese children and adolescents (Laurent & Sibold, 2016). A weight management intervention exploring hedonic hunger and food cue reactivity among adults concluded that before weight loss, greater hedonic hunger was associated with a greater attentional bias to high-calorie food cues. Whereas, weight-loss was associated with less receptiveness to high-calorie

food cues. Overall, change in hedonic hunger predicted weight-loss (Mead, Ahern, Halford, Harrold, & Boyland, 2015). In a similar study, Schütz, Bower, & Ferguson (2015) found that scores on Power of Food (PFS) scale, a measure of hedonic hunger, predicts the probability of everyday snacking behavior. A significant interaction was noted between PFS and effects of internal and external cues on the likelihood of snacking. Another study by O’Neil, Theim, Boeka, Johnson, & Miller-Kovach (2012) examined whether a commercially-available weight loss program is associated with the changes in hedonic hunger (measured using PFS), and whether these changes are associated with weight loss. Reported decreases in hedonic hunger were associated with better weight loss. When it comes to adolescents, whether hedonic food reward processes are associated with weight status has only been studied peripherally. For example, a recent study suggested that overweight children have a higher food cue sensitivity, they attend strongly to unhealthy foods, and they have a bias towards (palatable) unhealthy food (Smeets, Charbonnier, van Meer, van der Laan, & Spetter, 2012). In a study exploring the association between PFS, weight status, and dieting, among emerging adults, (Lipsky et al., 2016). PFS was not related to multiple measures of excess weight and weight gain. De Cock et al. (2016), concluded that sensitivity to reward was positively associated with unhealthy snacks intake among obese and overweight emerging adults. However, this relationship was only partially mediated by external and emotional eating. In a recent study with adolescents, Bejarano and Cushing (2018) investigated hedonic hunger’s function as a state and trait and examined the relationship among dietary motivation, hedonic hunger, and food intake. They concluded that hedonic hunger demonstrates both state and trait properties and this conceptualization influences consumption of food differently. Altogether this suggests that hedonic hunger predicts higher

intake of unhealthy food with an upward shift in weight, in addition to treatment response in weight control interventions.

The purpose of the current study was to examine whether there is an association between hedonic hunger and weight status in overweight/obese adolescents and determine if this association is mediated by dietary behavior, such as total calorie consumption. This study is unique because no published research has examined effects of hedonic hunger on adolescent weight status and no studies have examined whether dietary behavior mediates the association between hedonic hunger and weight status. We hypothesized a positive association between hedonic hunger and weight status (zBMI). Moreover, we hypothesized that this association would be mediated by dietary behavior (i.e., total calories,). If our hypotheses are confirmed, this study may provide evidence that adolescent weight control interventions should be designed accounting for hedonic reward sensitivity to increase the effectiveness of interventions for individuals with varying levels of hedonic hunger. Furthermore, our results may also suggest that screening for hedonic reward prior to beginning weight loss interventions may be an essential part of pre-treatment assessment.

Method

Participants

One hundred over-weight and obese (BMI % ile $\geq 85 < 95$) adolescent participants (ages 12-20; M age = 16.8, $SD = 2.5$) comprised the study sample. Participants were recruited using advertisements in pediatrician's office, recreation facilities and schools. Participants were drawn from three studies, two of which were randomized clinical trials of weight control interventions and the other examined effects of sleep duration on neural food reward and inhibition processes. All measures included in this study were completed at baseline (pre-treatment) so there were no

differences across groups on current assessments due to intervention. The eligibility criteria for participation, included, 1) the adolescent was between ages 12 -20, 2) the participant's body mass index percentile was classified in the overweight or obese range (BMI %ile ≥ 85), 3) the participant could speak and read English, 5) the participant did not have any diagnosable serious mental illness which could preclude participation (e.g., severe autism), 6) the participant was not taking medication prescribed for weight control, and 7) the participant had not undergone and did not plan to undergo bariatric surgery. All individuals meeting these criteria were deemed eligible to participate in the study.

Measures

Body mass index. Weight was measured using a digital scale (Seca 217, measured to the tenth of a pound) and height was measured using a portable stadiometer (seca 869, measured to the eighth of the inch). BMI percentiles (BMI%) for age and sex were calculated using the standard formula ($BMI = [\text{weight (kg)}]/[\text{height (m)}]^2$; Keys, Fidanza, Karvonen, Kimura, & Taylor, 1972). BMI was converted to BMIZ as recommended by the Centers for Disease Control (Centers of Disease Control and Prevention [CDC], 2011)

Hedonic food reward. Participants completed the Power of Food Scale (PFS) (Lowe et al., 2009). This measure is a 15-item questionnaire assessing appetite for and motivation to consume palatable food on a five-point Likert-type scale ranging from 1 (do not agree at all) to 5 (strongly agree) in three domains: (1) when food is available but not physically present; (2) when food is physically present but has not been tasted; and (3) when food has been tasted but not yet consumed. These three domain scores are aggregated to create a total score. The scale has good internal consistency (Cronbach' alpha from 0.81 to 0.91) and test-retest reliability (Cappelleri et al., 2009). With a community sample of preadolescents and adolescents, Cronbach's alpha for

the total scale and three subscales ranged from 0.86 to 0.95 (Mitchell, Cushing, & Amaro, 2016). PFS is stable construct for measuring hedonic hunger, that is not substantially affected by daily variations in hunger (Witt, Raggio, Butryn, & Lowe, 2014).

Diet. The Automated Self-Administered 24-hour Dietary Assessment Tool for Children (ASA-24 Kids) assessed dietary behavior. The ASA-24 is an Internet-based automated 24-hour diet recall assessment developed to understand the influences of nutritional status and assessment of dietary behavior change on health. Concordance rates suggest 47.8% match in recall between interviewer-administered and self-administered 24-hour diet recall (Baranowski et al., 2014). The literature suggests comparable report quality of self-administered dietary recall to the interviewer-administered dietary recall using ASA-24 (Hughes, Summer, Ollberding, Berken, & Kalkwarf, 2017). ASA-24 provides a valid estimate for intakes of a wide variety of dietary variables (Yuan et al., 2017).

Procedure

Interested individuals were given a contact phone number for study personnel who provided details regarding study participation and screened potential participants for eligibility. Subsequently, an in-person meeting was scheduled to complete the informed consent process. At this visit, study personnel discussed the benefits and risks of the study with the participant and answered questions regarding study participation. Participants were offered the opportunity to provide informed consent/assent for their participation and when required parents were offered the opportunity to provide informed consent for their child to participate. All the measures used in the present study were completed at the baseline visits. Therefore, the different study procedures did not affect performance on the assessments relevant to this study.

Data Screening

Prior to data analysis, primary study variables were inspected for data accuracy, missing values, and conformity to the assumptions of multivariate statistical analysis using STATA Version 16. Descriptive statistics including means, standard deviations and interquartile ranges were used to examine the distribution of study variables. Box plots and histograms were evaluated to identify univariate outliers. Univariate outliers were fenced to their median +/- two interquartile ranges (IQR) to reduce outliers' influences on their respective distributions. Next, a missing values analysis was conducted using Little's Missing Completely at Random (MCAR) Test and separate variance t-tests for all variables with 5% or more cases missing. The MCAR test was not significant ($\chi^2 = 0.242$, $df = 4$, $p = 0.993$) and missing values were not significantly related to the dependent variable so MCAR was assumed. Bivariate scatterplots were examined for departures from homoscedasticity. No significant departures were noted. Histograms and probability plots examining skewness and kurtosis were evaluated to determine normality in variable distributions. All three primary study variables, namely zBMI, hedonic hunger, and total caloric consumption were not normally distributed. Since Bayesian estimation does not assume or require normal distributions of data, no transformations were conducted (van de Schoot et al., 2013). Finally, distributions were evaluated for multivariate outliers and multicollinearity. Caloric intake, sugar intake, and total fat had significantly high correlations. Since caloric intake was highly collinear with sugar and fat consumption, only total calories were considered in the dietary analyses for this study.

Analytic plan

To evaluate our study hypotheses, we tested for mediation at varying levels of hedonic hunger using mixture modeling. Mixture modeling allowed us to identify heterogeneous subgroups classified by different mean levels of hedonic hunger, referred to as classes in the literature. Mixture modeling enhances our understanding by identifying similarities among key variables and their relationships with dependent variables. For this study, mixture modeling segmented classes depending upon the levels of hedonic hunger and its association with other variables. Bayesian estimation was used because of our relatively small sample size (Muthén & Asparouhov, 2012; van de Schoot et al., 2013). Without informative priors, the maximum likelihood estimates were used as priors by default (van de Schoot et al., 2013).

Results

Descriptive Statistics

Sixty-one percent of the participants were female. Seventy-one percent of the participants were overweight and 28% were obese. Non-Hispanic Whites represented 79% percent of the sample. (Table 1) Average baseline level of hedonic hunger was 35.25 ($SD = 14.35$, $Min: 12$, $Max: 84$). The average baseline zBMI was 1.4 ($SD = .44$, $Min .46$, $Max 3.2$). Participants reported their average baseline caloric consumption as 1890 kcal ($SD = 889.9kcal$, $Min 34kcal$, $Max 6036.3 kcal$; Table 2).

Table 1
Demographic Characteristics of Participants

Demographic Variable	n =101	%
Gender		
Male	39	38.6
Female	62	61.4
Body Mass Index (BMI)		
Overweight (BMI $\geq 85 \leq 94$)	67	71.3
Obese (BMI ≥ 95)	27	28.7
Race/Ethnicity		
White/ Not Hispanic	79	78
Hispanic/Latino	11	10.9
Asian	3	2.9
Native American	1	1.0
Other	6	5.9
Approximate Family Income		
\$21,000-\$30,000	0	0.0
\$31,000-\$40,000	4	3.9
\$41,000-\$50,000	9	8.9
\$51,000-\$59,000	17	16.8
\$60,000-\$69,000	14	13.8
\$70,000-\$79,000	4	3.9
>\$80,000	42	41.5
	<i>Mean</i>	<i>SD</i>
Age (years)	16.87	2.56

Note: M= mean, SD= standard deviation, four participants did not report BMI

Table 2

Summary of Pearson Correlations for Total Fat, Sugar, Calories, Hedonic Hunger, and BMIz.

Measure	<i>Sugar</i>	<i>Calorie</i>	<i>Hedonic Hunger</i>	<i>BMIz</i>	<i>M</i>	<i>SD</i>
Total Fat	.428**	.890**	.008	.148	35.25	14.34
Sugar	--	.675**	-.041	-.029	109.79	71.69
Calorie		--	-.024	.117	1890.51	889.95
Hedonic Hunger			--	.177	35.25	14.34
zBMI				--	1.43	.445

Note. *M* = mean; *SD* = standard deviation; *BMIz* = *BMI* z-score; *Calorie* measured in kilos, *Hedonic hunger* measured as total score on *Power of Food Scale (PFS)*

** $p < .01$

Mixture Modeling

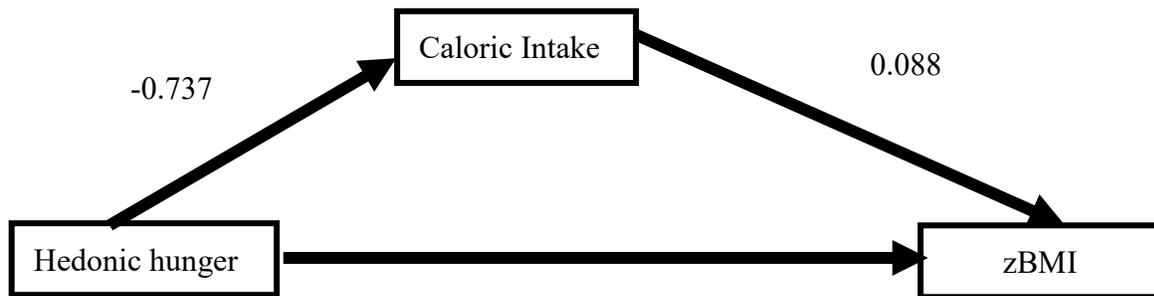
A three-class model provided the best fit to the data and a posterior predictive value ($ppp = 0.381$) indicated an acceptable fit. The classification of the model was good with an entropy of 0.88 (entropy above 0.80 is considered as an indication of good classification). The Model results are displayed in Table 3 and Figure 1. The first latent class comprised 19% ($n = 19$) of the cases, the second latent class had 65% ($n = 65$) of the cases. The third latent class had 16% ($n=16$). The first class ($n=19$, $M = 44.96$) indicated no direct effect ($\beta = -0.419$, $p = .140$; $CI = -1.097, 0.239$) of hedonic hunger on zBMI and no indirect effect ($\beta = -0.036$, $p = 0.45$; $CI = -0.565, 0.633$) was observed. However, there was a significant negative association between hedonic hunger and caloric intake ($\beta = -0.737$, $p = 0.018$; $CI = -0.867, -0.145$). In the second class ($n = 65$, $M = 29.86$) a direct effect ($\beta = 0.355$, $p = 0.018$; $CI = 0.016, 0.654$) of hedonic hunger on zBMI was noted. There was a positive association between hedonic hunger and zBMI; more specifically, with every one unit increase in hedonic hunger, there was a 0.35 points increase in zBMI. No indirect effect was noted, ($\beta = -0.007$, $p = 0.043$; $CI = -0.125, 0.054$). The

third class ($n = 16$, $M = 44.50$) had a significant direct association between hedonic hunger and zBMI ($\beta = -0.68$, $p < .01$, CI = 0.68, 1.35) such that with every one unit of increase in hedonic hunger the zBMI decreased by 68 units. A lifestyle intervention study with obese children suggested a decrease in zBMI as small as 0.18 results in differences in metabolic disease risk (Reinehr, Kleber, & Toschke, 2009). Hedonic hunger was positively associated with caloric intake in class 3 ($\beta = 0.52$, $p < .01$, CI = 0.24, .78), with every one unit increase in hedonic hunger there was an increase of 52 kilocalories daily. Additionally, there was a strong positive relationship between caloric intake and BMI ($\beta = 1.00$, $p < .01$, CI 0.68, 1.35), indicating that with every one thousand unit increase in calorie intake there was one unit increase in BMI. A test for mediation in class 3 using mixture modeling indicated significant indirect effect ($\beta = 0.56$, $p = .02$, CI .022, .017). 79% of variance in the zBMI was explained by the mixture model of mediation. Overall there was a negative association between hedonic hunger and zBMI; however, when mediator was involved the relationship changed, and indicated a positive association, indicating suppression.

Table 3
Latent Class Wise Bayesian Estimates of Mediation Analysis

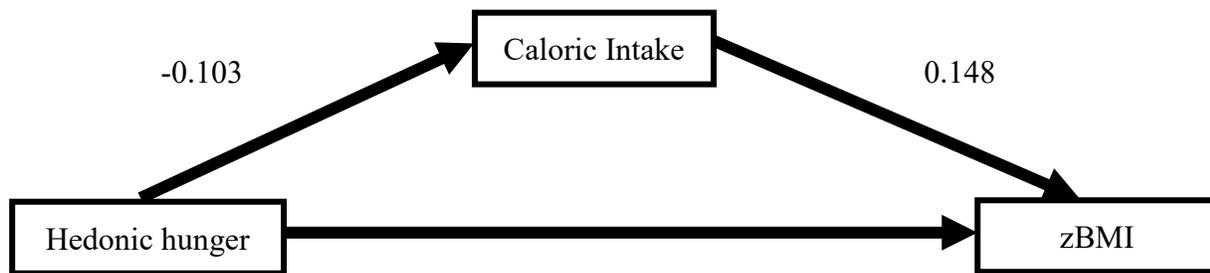
	β	S.E	p	95% CI
Class 1				
Direct	-0.419	0.356	0.140	-1.097, 0.239
Indirect	-0.036	0.291	0.450	-0.565, 0.633
Class 2				
Direct	0.355	0.162	0.018	0.016, 0.654
Indirect	-0.007	0.043	0.357	-0.125, 0.054
Class 3				
Direct	-0.678	0.149	0.000	-1.011, -0.408
Indirect	0.516	0.262	0.022	0.017, 1.056

*Note. Direct effect hedonic hunger -> zBMI
Indirect effect hedonic hunger-> calories ->zBMI*



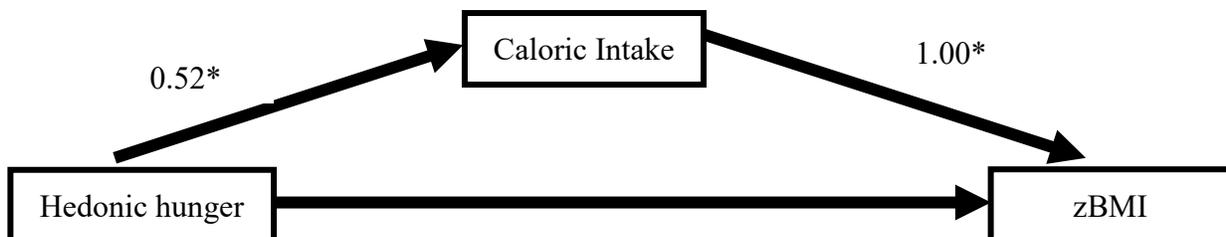
Direct effect = -0.419 ; Indirect effect = 0.025

Figure 1. Mixture mediation model for first latent class (n = 19) demonstrating the association between hedonic hunger and zBMI by caloric intake * $p \leq .05$, ** $p \leq .01$



Direct effect = 0.355*; Indirect effect = -0.00

Figure 2. Mixture mediation model for second latent class (n=65) demonstrating the association between hedonic hunger and zBMI by caloric intake * $p \leq .05$, ** $p \leq .01$



Direct effect = -0.68**; Indirect effect = 0.52*

Figure 3. Mixture mediation model for third latent class (n=16) demonstrating the association between hedonic hunger and zBMI mediated by caloric intake * $p \leq .05$, ** $p \leq .01$

Discussion

There is abundant evidence that the omnipresence of highly palatable food activates a hedonic pattern of eating (Davis et al., 2011; Johnson, 2013; Lowe & Butryn, 2007; Morris, Beilharz, Maniam, Reichelt, & Westbrook, 2015; Papies et al., 2007). Among overweight individuals, exaggerated hedonic responses constitute a behavioral risk for excessive energy intake (Blundell & Finlayson, 2004; Hall, Hammond, & Rahmandad, 2014). Given the debilitating long-term health complications associated with obesity, adolescence is a focal period for understanding eating behaviors that may favor the development of obesity. There is a dearth of literature on the association between hedonic hunger and increase in the BMI among adolescents. A better understanding of individual differences is important to illuminate the causes of obesity and identify potential solutions. The current study explored whether there is a positive association between hedonic hunger and zBMI and whether this relationship was mediated by caloric intake. Using a mixture model of mediation, it was hypothesized that mediation could occur at certain levels of hedonic hunger.

Consistent with the first hypothesis, results demonstrated a positive association between hedonic hunger and zBMI. For the majority of our participants (65%) hedonic hunger was positively associated with BMI (see Table 3). Across the 65% of participants, zBMI increased by 0.35 units for every one unit increase in hedonic hunger. This finding demonstrates that hedonic eating can override the homeostatic need for energy in response to rewarding food items and potentially stimulate weight gain. Consistent with the larger literature in pediatric obesity, our findings reiterate that hedonic hunger in adolescents could contribute weight gain (Drewnowski & Specter, 2004; Fisher & Birch, 2002; Hill & Peters, 1998; Swinburn, Egger, & Raza, 1999). Eating in the absence of hunger is said to promote obesity in girls as young as 7 years old (Fisher

& Birch, 2002). Our results contribute to the growing literature on hedonic hunger and obesity among adolescents that exaggerated hedonic responses promote excessive energy intake.

Our second hypothesis that the association between hedonic hunger and zBMI would be explained by caloric intake, was built on previous literature on adult obesity (Mead et al., 2015; O'Neil et al., 2012). However, results of the current study did not demonstrate a similar relationship among adolescents. Caloric intake suppressed the association for 16% of participants, hence caloric intake only tentatively mediated the association between hedonic hunger and weight status. Since no known study has examined this relationship before, our findings may be limited by lack of supporting evidence. Shrout and Bolger (2002) explained that suppression occurs when the indirect effect has the opposite sign of the direct effect. Conger (1974) defined suppressor variable as “one which increases the predictive validity of another variable by its inclusion in a regression equation.” Such models are also called “inconsistent mediation models” (MacKinnon, Fairchild, & Fritz, 2007). Overall, it is unclear why and how this effect could occur, however in our case, it could be because a suppressor variable may substantially correlated with the dependent variable (Maassen & Bakker, 2001), suggested by high covariance between caloric intake and zBMI.

Specific to a sub-group comprising 16% of our sample, there was strong covariance between caloric intake and zBMI. This is a consistent phenomenon among adolescents; each additional serving of sugar-sweetened drink consumed is significantly associated with an increase in BMI and frequency of obesity odds (Ludwig, Peterson, & Gortmaker, 2001). In a review on a trajectory towards weight gain and obesity in adolescents, Rosenheck (2008) found strong evidence between adolescents increased fast-food intake and associated caloric intake leading to weight gain.

The absence of mediation may also be explained by the lack of temporal precedence in our study. Hedonic hunger likely exerts influence on BMI over a lengthy time period and assessing these constructs over time may be a more ecologically valid method. Shrout and Bolger (2002) suggested that as the causal process becomes more distal, the size of the effect typically gets smaller, potentially because the influence of the independent variable is likely to get transmitted through additional links in the causal chain, or might get affected by random factors. For example, some individuals might consume food in the absence of short-term energy deficit, not because of the palatability of food, but because of potential scarcity in the future. Furthermore, according to the homeostatic – hedonic model of eating (Lowe & Levine, 2005) the appetitive motivation among overweight individuals can occur simply because of energy deficiency or evolutionary needs. This implies that the food consumed in the absence of physiological hunger prevents the development of future physiological hunger; resulting in increased consumption and storage of extra energy as body fat.

Recent advances in the field recommend considering the individual differences in the dynamic interaction between hedonic, homeostatic and cognitive feedback systems (Hall et al., 2014). For example, energy inefficient; who do not gain weight despite frequent overeating, are particularly deserving of study unlike the energy efficient individuals (Blundell & Finlayson, 2004). Another factor is the “pull” obesity-prone individuals feel from the obesogenic environments (Davis, Strachan, & Berkson, 2004). The pull may be greater when individuals are gaining weight than when their weight stabilizes at an obese level. Additionally, there is literature suggesting that the interaction between diminished inhibitory control capacities and sensitivity towards the rewarding properties of food can predict weight-gain among young adult females (Appelhans et al., 2011; Nederkoorn, Houben, Hofmann, Roefs, & Jansen, 2010).

Similarly, self-regulatory competency may also serve as a moderator of the negative impact of being sensitive to the food environment on eating behavior (French, Epstein, Jeffery, Blundell, & Wardle, 2012; Stok et al., 2015) decisions about when to eat are modulated by homeostatic hunger, as well as the availability of the food, the social context, and one's response to food cues (Hall et al., 2014). Since we did not explore the individual differences in the intake and expenditure of energy, it is possible that an interaction between hedonic hunger, homeostatic eating and individual differences in cognitive capacities could explain the association better.

Conclusions and Future Directions

We conclude that hedonic hunger is associated with increased sensitivity towards the rewarding properties of food and change in weight status for a subset of adolescents. This research adds to the growing body of research that heightened responsiveness to food environments drives unhealthy eating practices leading to weight gain among adolescents. Our study contributes to the widely growing field of hedonic hunger and obesity by exploring the role of caloric intake as a mediator. Our study provides preliminary evidence that caloric intake did not mediate the association between hedonic hunger and weight status. Caloric intake suppressed the association; tentative mediation suggests that some teenagers may experience an exaggerated hedonic response to palatable food, deriving more pleasure from eating and as a result consumed more calories resulting in an upward shift of weight. Whereas, others might have a diminished ability to experience pleasure from food and hence consume food to satisfy an optimum level of stimulation. Overall, our results are commensurate with Lowe et al., 2009 that some individuals are more psychologically sensitive to the obesogenic environment than others. Future research would benefit by considering the mediating as well potential suppressing variables; a theory that

takes into account mediating and suppressing variables is more complete than a theory that examines only the former (Rucker, Preacher, Tormala, & Petty, 2011).

Changes in hedonic hunger during weight-loss intervention are well established in adult weight-loss literature (O'Neil, et al., 2012; Schultes, Ernst, Wilms, Thurnheer, & Hallschmid, 2010). Our study suggests that it is important to consider hedonic hunger in adolescent weight control interventions because it may influence dietary behavior and weight outcomes for some individuals. Individuals with hedonic obesity may respond better to behavioral and lifestyle modification, including traditional behavioral therapy (Yu et al., 2015).

Limitations

The present study should be interpreted within the context of some limitations. The PFS quantifies hedonic hunger as a trait, measuring a tendency toward preoccupation with food despite the absence of short-term energy deficit. However, it is likely that this trait interacts with exposure to food cues. Moreover, PFS does not contain items measuring actual food consumption or overeating. Vainik, Neseliler, Konstabel, Fellows, & Dagher, (2015), argued that PFS reflect a continuum of an “uncontrolled eating” construct ranging from no overeating on one extreme to food addiction on the other, and it may not represent food reward sensitivity per se. Furthermore, the items on the PFS scale reflect a preoccupation with delicious foods, thus a high-scoring PFS participants experience more difficulty than a low-scoring participant to inhibit their urges to eat highly palatable food (Lowe et al., 2016).

Lastly, our sample comprises developing adolescents and risk factors for childhood obesity change with age (Ambrosini, 2014). The association between an energy-dense, high-fat, low-fiber dietary pattern and adiposity diminishes between 7 and 15 years of age (Ambrosini,

Emmett, Northstone, & Jebb, 2014). Further studies examining the influence of hedonic hunger at various developmental stages are warranted.

The cross-sectional nature of the study limits our ability to make causal claims. Additionally, social desirability could have impacted the self-report measure of hedonic hunger and caloric intake. Moreover, self-reported food consumption data are subject to limitations, such as reactivity and accuracy of reporting serving size. Use of additional measures to validate self-reported data can add substantial value. In the light of suppression, future research should explore calorie intake as a moderated mediator.

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