HHS Public Access

Author manuscript

Appetite. Author manuscript; available in PMC 2017 October 01.

Published in final edited form as:

Appetite. 2016 October 1; 105: 385–391. doi:10.1016/j.appet.2016.06.012.

Power of Food Scale in association with weight outcomes and dieting in a nationally representative cohort of U.S. young adults

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Abstract

Food reward sensitivity may influence susceptibility to overeating in a permissive food environment, contributing to unintended weight gain and intentional weight loss behavior. This study examined associations of food reward sensitivity, assessed by the Power of Food Scale (PFS), with weight outcomes and dieting in a nationally representative cohort of U.S. emerging adults. Wave 5 (W5, 5th year of follow-up) respondents from the NEXT Generation Health Study were included (N=2202, W5 age=20.3±0.02 years). Baseline and W5 BMI, W5 weight status (normal weight=18.5≤BMI<25, overweight=25≤BMI<30, obese=BMI≥30), BMI change (W5-baseline BMI) and onset of overweight or obesity (OWOB) were calculated from self-reported height and weight. PFS (aggregate and 3 domain scores: food available, present, and tasted) and dieting for weight-loss were assessed at W5. Adjusted linear regressions estimated associations of PFS with W5 BMI and BMI change. Log-binomial regressions estimated associations of high W5 BMI (≥25), OWOB onset and dieting with PFS. Post hoc analyses estimated associations of PFS with W5 perceived weight status (overweight vs. about right or underweight). W5 BMI=25.73±0.32 kg/m^2, and OWOB onset occurred in 27.7% of participants. The PFS-food available score was associated with BMI change, β±SE=0.41±0.19. Other PFS scores were not associated with weight outcomes. Dieting prevalence was higher in participants with high versus low W5 BMI (61% versus 32%), and was positively associated with all PFS scores except the PFS-food tasted score, e.g., relative risk (RR) of dieting for PFS-aggregate=1.13, 95%CI [1.01-1.26]. Post-hoc analyses indicated perceived overweight was positively associated with PFS-food available, 1.12, [1.01-1.24], and PFS-food present, 1.13, [1.03-1.24]. PFS was positively related to dieting and perceived overweight, but not concurrent or change in weight status in a representative cohort of U.S. emerging adults.

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Conflict of Interest

The authors declare no conflict of interest.

Keywords

Adolescent; Young adult; Body Mass Index; Obesity; Overweight; Cohort Studies

1. Introduction

Young adulthood represents a critical period for obesity development. The prevalence of overweight and obesity increased by approximately 25%-75% over 10 years in participants aged 18-30 years at baseline in the CARDIA study (Lewis et al., 2000). Similarly, obesity prevalence doubled between the ages of 18 and 30 years in participants of the 1970 British Birth Cohort (Viner & Cole, 2006). Recent cross-sectional data indicate the prevalence of overweight and obesity among adults aged 20-39 years is nearly twice that of adolescents 12-19 years (Ogden, Carroll, Kit, & Flegal, 2014). Given the well-documented impact of obesity on multiple health indicators and outcomes (World Health Organization (WHO), 2000), research is urgently needed to better understand correlates of weight increases during the transition from adolescence to young adulthood ("emerging adulthood").

Individual susceptibility to weight gain may be influenced by food reward sensitivity, the neurologic response to the rewarding properties of food stimuli (Davis, Strachan, & Berkson, 2004) leading to increased motivation to eat in the absence of metabolic need (Lowe & Butryn, 2007). Findings from functional magnetic resonance imaging (fMRI) studies have shown that greater sensitivity to food cues predicts increases in body weight (Stice, Yokum, Bohon, Marti, & Smolen, 2010; Sun et al., 2015; Yokum, Ng, & Stice, 2011) and body fat (Stice, Burger, & Yokum, 2015), as well as reduced success from participating in a weight-loss program (Murdaugh, Cox, Cook, & Weller, 2012). Further, sensitivity to food stimuli was associated with greater food intake in the absence of hunger (Nolan-Poupart, Veldhuizen, Geha, & Small, 2013), supporting the hypothesis that increased food reward sensitivity may underlie both unintentional overeating and weight gain. However, the generalizability of these neuroimaging studies may be limited by the small sample sizes and the homogenous sample characteristics (e.g., primarily White, female young adults). Larger studies have investigated food reward sensitivity using the Power of Food Scale (PFS) (Cappelleri et al., 2009; Lowe et al., 2009), a psychometric assessment of motivation to consume palatable foods in the environment. This measure has been positively associated with BMI over time in clinical weight-loss and bariatric-surgery patients (Carpenter, Wong, Li, Noble, & Heber, 2013; Schultes, Ernst, Wilms, Thurnheer, & Hallschmid, 2010; Ullrich, Ernst, Wilms, Thurnheer, & Schultes, 2013). However, studies in larger, non-clinical samples have yielded inconsistent findings regarding the association of PFS with BMI (Cappelleri et al., 2009; Lowe et al., 2009; Yoshikawa, Orita, Watanabe, & Tanaka, 2012), and have not examined associations with BMI change. The The association of the PFS with BMI and BMI change in a large, representative sample of US emerging adults has not been investigated.

Dieting, the act of restricting intake in order to produce weight loss, is paradoxically a reliable predictor of long-term weight gain (Lowe, Doshi, Katterman, & Feig, 2013; Neumark-Sztainer, Wall, Story, & Standish, 2012). Several explanations causally link dieting

to weight gain through metabolic and/or behavioral mechanisms (Mann et al., 2007; Neumark-Sztainer, Wall, Haines, Story, & Eisenberg, 2007; Rosenbaum, Kissileff, Mayer, Hirsch, & Leibel, 2010; Stice, Durant, Burger, & Schoeller, 2011). However, an alternative hypothesis is that dieting and weight gain are both influenced by factors underlying an individual's susceptibility to overeat (Hill, 2004; Pietilainen, Saarni, Kaprio, & Rissanen, 2012), such as heightened sensitivity to food cues (Ely, Childress, Jagannathan, & Lowe, 2014; Lowe & Butryn, 2007). While the relationship of dieting with PFS has not been previously examined, dietary restraint was positively associated with PFS in a large sample of predominantly White college students (Lowe et al., 2009), and with activation of brain reward regions during food intake in a small sample of adolescent females (Burger & Stice, 2011). However, dietary restraint is conceptually distinct from dieting since restrained eaters do not consistently consume less than non-restrained eaters (Stice, Fisher, & Lowe, 2004), and only a subset of restrained eaters report dieting to lose weight (Goldstein, Katterman, & Lowe, 2013). Identifying the psychosocial risk factors for dieting may help identify young adults most susceptible to excess weight gain and who would benefit most from early intervention.

The purpose of this study was to investigate the association of PFS with weight outcomes and dieting in a nationally representative cohort of emerging adults. We examine several indicators of body weight including concurrent BMI and weight status as well as BMI change and risk of onset of overweight and obesity (OWOB) over 5 years.

2. Materials and methods

2.1. Study Setting

Data come from the NEXT Generation Health Study, an on-going, observational prospective cohort study of multiple health indicators and behaviors in a nationally-representative sample of U.S. emerging adults. School districts were the primary sampling units, stratified by the nine major U.S. census divisions. Of137 selected schools, 81 (59%) agreed to participate. Classrooms were randomly selected within schools for inclusion. Baseline data were collected during the 2009-2010 school year, when students were in 10^{th} grade; however, due to timing of school approval for participation, baseline assessments of 260 of the participants occurred during the second wave (2010-2011, 11th grade). Schools with large percentages of African American students were oversampled to obtain reliable estimates for this subgroup; a sufficient number of Hispanic students were obtained to give reliable subgroup estimates without oversampling. Participants completed self-administered surveys annually; those completing the survey in wave 5 (W5, 79% retention, N=2202) were included in this study. Parents provided informed consent for their child's participation; youth provided assent (when <18 years of age) and consent (when ≥18 years). Procedures were approved by the institutional review board at the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development.

2.2. Measures

Several weight outcomes were investigated in order to obtain a comprehensive understanding of the association of PFS with body weight. Body mass index (BMI, kg/m^2)

at baseline and W5 was calculated from self-reported height and weight. Baseline weight status for adolescents was calculated using sex- and age-specific Centers for Disease Control BMI percentiles (%ile) (underweight=BMI%ile<5, normal weight=5≤BMI%ile<85, overweight=85≤BMI%ile<95, obese=BMI%ile≥95) (Ogden & Flegal, 2010). W5 weight status was calculated using CDC BMI cutoffs for adults>20 years of age (underweight=BMI<18.5, normal weight=18.5≥BMI<25, overweight=25≥BMI<30, obese=BMI≥30) (American College of Cardiology/American Heart Association Task Force on Practice Guidelines, 2014). BMI change (kg/m^2) was calculated as W5–baseline BMI. Participants were additionally classified as having high W5 BMI (BMI≥25 versus BMI<25). Onset of overweight or obesity (OWOB) was defined as moving to a higher risk BMI category between baseline and W5. For this variable, participants with baseline underweight and normal weight BMI were coded as OWOB onset if W5 weight status was overweight or obese, and participants with baseline overweight BMI were coded as OWOB onset if W5 weight status was obese. In line with predefined BMI risk categories for adults (American College of Cardiology/American Heart Association Task Force on Practice Guidelines, 2014), participants with baseline obese BMI were coded as OWOB onset if W5 BMI exceeded 35 kg/m^2 (“class II obesity”) and exceeded baseline BMI.

Past year dieting in W5 was assessed with the question, “How often have you gone on a diet during the last year? By “diet” we mean changing the way you eat so you can lose weight.” Response options included *never*, *1-4 times*,*5-10 times*,*more than 10 times*,*I am always dieting*. This question was used in adolescents participating in the Project EAT study (Neumark-Sztainer et al., 2007). Consistent with previous use, the variable was dichotomized as having gone on a diet versus never having gone on a diet in the past year.

Food reward sensitivity was measured at W5 using the previously validated Power of Food Scale (PFS) (Lowe et al., 2009). The measure consists of 15 items assessing appetite for and motivation to consume palatable foods 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 (Cappelleri et al., 2009). This construct is hypothesized to contribute to excessive dietary intake relative to metabolic needs.” Responses are measured on a 5-point Likert scale ranging from *do not agree at all* to *strongly agree*. The measure is represented by three domain scores and an aggregate score (Cappelleri et al., 2009). Domain scores are calculated as the mean of the items representing the corresponding domain: food available (e.g., “I find myself thinking about food even when I am not physically hungry”); food present (e.g., “If I see or smell a food I like, I get a powerful urge to have some”); and food tasted (e.g., “Just before I taste a favorite food, I feel intense anticipation”). The aggregate score is calculated as the mean of the three domains. There was high internal consistency of PFS items in the current sample (food available α=0.85, food present α=0.87, food tasted=.84, all items=0.94).

Covariates include baseline sociodemographic characteristics and W5 height and vigorous physical activity. Participants reported sex and race/ethnicity at baseline. The previously-validated Family Affluence Scale (Currie et al., 2008) was calculated based on participant survey responses regarding household car and computer ownership, family vacations, and bedroom sharing , and ranges from 0 (*low affluence*) to 7 (*high affluence*). Parent-reported

educational attainment was ascertained during the consent process and categorized as high school graduate or less/some college/bachelors or graduate degree. Participants reported previous-week vigorous physical activity (hours per week) at W5, which was included as a covariate given its associations with body weight (Westerterp, 2010) and eating behaviors (Bild et al., 1996; Lipsky et al., 2015). Participants were asked “How many hours a week do you usually engage in vigorous physical activity so much that you get out of breath or sweat”, with responses ranging from none to 7 hours or more.

2.3. Statistical analysis

Multiple imputation by chained equations, assuming missing-at-random (Buuren & Groothuis-Oudshoorn, 2011; Little & Rubin, 2002) was used for missing variables. The algorithm iteratively imputes missing variables by estimating its distribution conditional on other variables. Fifty imputed datasets were generated using IVEware (Ann Arbor, MI). StataSE version 14 (College Station, TX) was used for all analyses; each dataset was analyzed separately and the results were combined using Rubin's rule (Little & Rubin, 2002).

Baseline sample characteristics, overall and by W5 BMI and dieting status, were summarized using means or proportions and linearized standard errors. *F*-tests were used to examine bivariate differences in sample characteristics. Linear regression models estimated associations of W5 PFS with W5 BMI and BMI change, adjusted for covariates (baseline sex, race/ethnicity, family affluence and parent education, and W5 age, height and vigorous physical activity). Log-binomial regression models controlling for the same covariates estimated relative risk of high W5 BMI, OWOB onset and dieting associated with PFS. This analytic approach provides a more accurate risk estimate than logistic regression for common outcomes (Cummings, 2009). Survey estimation methods accounted for the complex sampling design.

3. Results

3.1. Sample characteristics

Approximately half the sample was female and non-Hispanic white, and 43.9% were overweight or obese at W5 (**Table 1**). OWOB onset occurred in over one quarter of the participants. In W5, nearly half the sample reported going on a diet to reduce body weight at least once over the past year. The proportion of participants with high W5 BMI reporting dieting (61%) was nearly double that of participants with low W5 BMI (32%). BMI change from baseline, and OWOB onset were significantly higher in dieters than non-dieters.

3.2. Associations of PFS with continuous W5 BMI and BMI change (W5 – baseline)

Mean±linearized SE for the PFS scores were 1.84±0.02, 2.23±0.03, 2.09±0.03, and 2.05±0.02 for the food available, food present, food tasted, and aggregate scores, respectively.

In linear regression models adjusted for baseline sociodemographics and W5 height and vigorous physical activity, W5 BMI was not associated with PFS scores (**Table 2**). BMI change was positively associated only with the food available domain.

3.3. Associations of PFS with odds of high W5 BMI, risk of OWOB onset and dieting

In log-binomial regression models adjusted for baseline sociodemographics and W5 height and vigorous physical activity, PFS scores were not associated with high W5 BMI or OWOB onset (**Table 3**). The point estimates for of the associations of high W5 BMI and OWOB onset with the food available, food present and aggregate PFS scores were greater than 1, while those of the food tasted domain were less than 1, although none of the estimates were statistically different from the null. Dieting was positively associated with the food available, food present and aggregate PFS scores, but was not associated with the food tasted score.

3.4. Post hoc analyses: Associations of perceived weight status with PFS

Given the positive associations of dieting with weight outcomes and PFS, and the non-significant association of PFS with weight outcomes, we investigated whether PFS was instead associated with subjective weight status (i.e., perceived overweight). Participants were asked in W5, “At this time, do you feel you are...”, with response options including “very underweight”/”somewhat underweight”/”about the right weight”/”somewhat overweight”/”very overweight”. The variable was dichotomized as perceived overweight (“somewhat overweight” or “very overweight”) versus lower perceived weight status (“about the right weight”, “somewhat underweight” or “very underweight”).

Bivariate correlations of weight perception with sample characteristics are shown in the supplementary material. Log-binomial regressions estimated RR of perceived overweight associated with PFS domain and aggregate scores, controlling for baseline sociodemographics and W5 height and vigorous physical activity. Perceived overweight was positively associated with the food available, RR=1.12, 95%CI [1.01, 1.24], p=0.04, and food present, 1.13, [1.03, 1.24], p=0.009) scores. Associations with the food tasted, 0.95, [0.86, 1.04], p=0.26, and aggregate, 1.08, [0.97-1.20], p=0.17, scores did not reach statistical significance.

4. Discussion

The objective of this study was to examine associations of food reward sensitivity, assessed by the Power of Food Scale (PFS), with weight outcomes and dieting in a nationally representative cohort of 10th graders followed prospectively. Contrary to the hypothesis, PFS domain and aggregate scores were not associated with concurrent or longitudinal weight outcomes except for a positive association of the food available domain with BMI change from baseline to W5 (5 years after baseline). However, dieting was positively associated with all PFS domain and aggregate scores other than a null association with the food tasted domain score. These findings suggest that while PFS is related to efforts to reduce weight, this measure generally does not explain the variability in objective relative weight (BMI or a priori BMI risk categories) in U.S. emerging adults.

Previous findings regarding the association of PFS with body weight are conflicting. Several studies in clinical populations have shown lower PFS and body weight in obese bariatric surgery patients relative to obese control patients who have not undergone surgery (Schultes et al., 2010; Ullrich, Ernst, Wilms, Thurnheer, Hallschmid, et al., 2013; Ullrich, Ernst, Wilms, Thurnheer, & Schultes, 2013). One study demonstrated approximately parallel decreases in PFS and weight change after surgery, and a later increase in both measures from 18-24 months follow-up(Cushing et al., 2014). These findings have led researchers to hypothesize that the impact of bariatric surgery on weight change may be causally mediated through PFS. The relationship between PFS and body weight in the general population is less consistent. PFS was not correlated with BMI in a sample of Japanese young adults (Yoshikawa et al., 2012), a sample of Canadian young adult women (Vainik, Neseliler, Konstabel, Fellows, & Dagher, 2015), an international sample of healthy college students (Lowe et al., 2009), or a small sample of healthy overweight and obese U.S. women (Appelhans et al., 2011). In contrast, PFS was positively associated with BMI in a sample of Estonian young adult women (Vainik et al., 2015). Cappelleri et al. reported no association of PFS with BMI in a clinic sample but positive associations of PFS domain and aggregate scores with BMI in a more general population of older (approximately 50 years of age) U.S. adults (Cappelleri et al., 2009). The lack of association of PFS with any concurrent or longitudinal weight outcome in the current study of a large, nationally representative cohort of US emerging adults suggests a need to explore additional explanatory factors in this population.

To our knowledge, this is the first study to investigate and provide evidence of a relationship of PFS with dieting; a one unit increase in the PFS aggregate score was associated with a 13% increased risk, 95%CI [1%, 26%], of reducing intake in order to lose weight. This is consistent with previous studies demonstrating a positive correlation between PFS and dietary restraint (Lowe et al., 2009), a construct that is associated with dieting (DelParigi et al., 2007). The present study also demonstrated a positive bivariate association of dieting with weight status. Taken together, these findings demonstrate that dieting is positively associated with both PFS and weight status, despite little evidence of an association of PFS with weight outcomes. One confounding factor may be the purported circular association of dieting and weight gain, whereby weight gain leads to dieting, which can lead to either weight loss or gain (Hofmann, Adriaanse, Vohs, & Baumeister, 2014). This dynamic association of weight change with dieting along with individual factors contributing to dieting initiation (French, Story, Downes, Resnick, & Blum, 1995) may obscure an overall association of PFS with weight outcomes.

Another potential explanation for the findings is that dieting may lead to increased food reward sensitivity. Food reward sensitivity is hypothesized to be a stable trait (Lowe et al., 2009) despite evidence of modification due to bariatric surgery. While the association of dieting with PFS has not been examined, one study reported greater activation of reward circuitry while viewing highly palatable food images in participants with a history of dieting for weight loss (historical dieters) as compared with nondieters (Ely et al., 2014); activation of reward circuitry was simultaneously lower in current dieters than historical dieters. Relatedly, cross-sectional studies examining the impact of dieting on cravings have demonstrated higher food cravings (i.e. desire to eat specific foods in the absence of hunger)

in dieters versus non-dieters (Massey & Hill, 2012), whereas experimental studies consistently show significant decreases over time in food cravings in participants following diets to lose weight relative to controls (Batra et al., 2013; Lappalainen, Sjoden, Hursti, & Vesa, 1990; Martin, O'Neil, & Pawlow, 2006). Although food cravings are distinct from the concept of food reward sensitivity, studies demonstrating higher state cravings for desired food in participants with higher PFS scores (Forman et al., 2013; Rejeski et al., 2012) suggest that decreased food cravings would not occur in the context of increasing food reward sensitivity. Taken together, this evidence suggests that the observed positive association of PFS with dieting in this sample is more likely due to greater food reward sensitivity preceding intentions to restrict food intake to lose weight.

After observing positive associations of dieting with PFS and weight status in the absence of an association of PFS with weight outcomes, we conducted post hoc analyses to examine whether these findings could be explained by an association of PFS with perceived overweight, which has been positively associated with dieting (Liechty & Lee, 2015; Quick et al., 2014). These analyses demonstrated that two of the three PFS domain scales (food available and food present) were positively associated with RR of perceived overweight. The relation of PFS to perceived rather than objective weight status suggests that food reward sensitivity correlates with individuals' weight perception and dieting across the range of BMI.

We also considered whether the absence of an association of PFS with weight outcomes may be due to limitations of the PFS measure. Vainik and colleagues (2015) have argued that PFS reflects the middle along a continuum of an underlying "uncontrolled eating" construct ranging from no overeating on one extreme to food addiction or bingeing on the other, and may not represent food reward sensitivity per se. In their analysis, the PFS was found to have an underlying two-factor structure, and these factors (one representing loss of control eating and the other reflecting enjoyment of food) demonstrated opposing associations with BMI. As a post-hoc sensitivity analysis, we examined whether these findings were applicable in our sample by conducting an exploratory factory analysis, and found almost the identical two-factor structure reported by Vainik and colleagues. However, both factor scores were unassociated with objective weight outcomes in our study. Nonetheless, the strikingly similar findings from exploratory factor analysis in these two large, distinct samples suggest that additional psychometric evaluation of the PFS may be warranted. Another consideration is that while the validity of the PFS with respect to dietary restraint, emotional eating and external eating has been documented (Lowe et al., 2009), only two studies have examined the correlation of PFS with brain imaging data, which may be the gold standard measure of the underlying construct. One study demonstrated a positive correlation of intensity of magnetoencephalography responses to viewing food images with scores on the food available and food present domain scores as well as the aggregate PFS score (Yoshikawa, Tanaka, Ishii, & Watanabe, 2013). Another study demonstrated an association of high PFS aggregate scores with greater brain activity in response to food cues in a fed state (Rejeski et al., 2012), suggesting that PFS is correlated with brain activity associated with eating motivation specifically in the absence of metabolic need. Notwithstanding the potential weaknesses of the PFS, the fact that PFS was associated with dieting and perceived weight status in the current study suggests that the absence of an

association with objective weight outcomes may be related to unmeasured confounding or moderating factors.

Interpretation of these findings should consider the relative strengths and limitations of the study. One limitation is that PFS was assessed only at W5, concurrent with W5 self-report height and weight, but after the change from baseline occurred. If PFS reflects a stable trait, it would be expected to have the same value regardless of measurement timing. If instead PFS varies over time, e.g.,(Witt, Raggio, Butryn, & Lowe, 2014), this may introduce bias or imprecision in the estimates of its association with BMI change. Another limitation is the lack of a measure of the exposure to highly palatable foods in the environment. While the obesigenic environment is ubiquitous in the U.S. (Piernas & Popkin, 2010; Young & Nestle, 2002), the ability to test for differences, which may interact with PFS to influence eating behavior, is an important area for future study. Additionally, PFS is a self-reported measure of food reward sensitivity, which may have limited our ability to detect a true association with weight outcomes. However, objective assessment through brain imaging was not feasible in the context of this large, national cohort. BMI was similarly calculated from self-reported height and weight, which may have introduced bias. On the other hand, self-reported and measured BMI are highly correlated (r=0.91) (Goodman, Hinden, & Khandelwal, 2000), and self-reported BMI is strongly associated with health outcomes in this population (Pietrobelli et al., 1998; Steinberger et al., 2005). Notable strengths include the use of a large, contemporary, nationally representative sample of emerging adults including sufficient participants to produce generalizable estimates for several subgroups. As well, the prospective assessment of body weight prevents recall bias that may lead to misclassification of weight change. The use of multiple imputation additionally strengthens the internal validity of these findings relative to complete case analysis. Finally, the measurement of (and adjustment for) a number of potential confounders supports the validity of the estimates of the associations of the primary variables of interest.

5. Conclusions

Overall, findings from this study indicate that PFS was not related to multiple measures of excess weight and weight gain in a general population of U.S. emerging adults, suggesting the need for additional research to examine whether alternative self-report or objective measures of food reward sensitivity can account for the variance in these outcomes in this population. Higher PFS scores were associated with more frequent dieting and greater risk of perceived overweight, indicating that PFS is more sensitive to subjective evaluation of excess weight and intentional weight loss behaviors. Future research is warranted to investigate whether PFS can be used to identify young adults across the range of BMI who would benefit most from interventions to improve weight control.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research (contract number HHSN275201200001I) was supported in part by the intramural research program of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD), and the National Heart, Lung and Blood Institute (NHLBI), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), and Maternal and Child Health Bureau (MCHB) of the Health Resources and Services Administration (HRSA), with supplemental support from the National Institute on Drug Abuse (NIDA). Intramural researchers were responsible for the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication. The authors thank Ms. Katie Dempster for her contributions to this work.

Abbreviations

PFS	Power of Food Scale
fMRI	functional magnetic resonance imaging
OWOB	overweight and obesity
%ile	percentile
CDC	Centers for Disease Control and Prevention
W5	wave 5

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Table 1

Sample characteristics overall and by wave 5 (W5) BMI and dieting status

	Overall (N=2201)		W5 BMI <25 kg/m2		W5 BMI ≥ 25 kg/m2			Dieters		Non-dieters		
	Mean or Proportion	Linearized SE	Mean or Proportion	Linearized SE	Mean or Proportion	Linearized SE	P^2	Mean or Proportion	Linearized SE	Mean or Proportion	Linearized SE	P^2
Sociodemographics												
Sex (% female)	59.2	1.8	59.8	2.7	58.4	2.8	.74	70.1	2.0	29.3	2.0	<.001
Age, W5 (y)	20.3	0.02	20.3	0.03	20.3	0.03	.61	20.2	0.03	20.3	0.03	.03
Family Affluence Score	5.5	0.1	5.6	0.1	5.3	0.1	.02	5.5	0.1	5.5	0.1	.90
Race/ethnicity (%)												
Non-Hispanic white	60.7	5.4	66.2	6.2	53.6	5.2	.06	58.9	5.7	62.2	5.5	.04
Non-Hispnic black	13.6	3.4	11.8	3.7	15.9	3.4		12.9	3.3	14.2	3.7	
Hispanic	20.3	3.9	17.9	3.8	23.2	4.5		23.5	4.1	17.6	3.9	
Other	5.4	1.0	4.0	1.0	7.2	2.6		4.7	1.4	6.0	1.3	
Parent education (%)												
HS diploma/GED or less	32.8	3.1	34.1	3.8	31.1	3.7	.28	30.4	3.6	34.7	3.5	.52
Some coll, tech school, AD degree	38.4	2.0	35.6	2.8	42.1	2.8		39.4	2.4	37.6	2.8	
Bachelor's or graduate degree	28.8	3.3	30.3	4.7	26.9	2.8		30.1	3.2	27.7	4.1	
Anthropometrics												
BMI, W5 (kg/m^2)	25.73	0.3	21.8	0.1	30.8	0.4	-	27.8	0.5	24.1	0.2	<.001
Weight category, W5 (%)			-	-	-	-	-					
Underweight (BMI<18.5)	3.8	0.7						1.9	1.1	5.3	0.9	<.001
Normal weight (18.5≤BMI<25)	52.3	2.1						38.4	2.7	63.6	2.5	
Overweight (25≤BMI<30)	26.0	1.4						32.8	2.4	20.6	2.3	
Obese (BMI≥30)	17.9	1.5						26.9	2.2	10.5	1.2	
BMI change (W5-baseline)	2.2	0.2	0.9	0.2	4.0	0.3	<.001	2.7	0.3	1.9	0.2	.007
Overweight/obesity onset[2] (%)	27.7	2.0	0	0	63.1	3.4	-	36.1	2.7	20.9	2.4	<.001
Dieter (% yes)	44.9	2.1	32.2	2.3	61.0	2.8	<.001					
Vigorous physical activity, W5 (hours/week)	2.6	0.1	2.7	0.15	2.43	0.2	.16	2.6	0.1	2.6	0.1	.80

BMI- body mass index, W5- wave 5 (5 years after baseline)

[1]F-test of overall significance

[2]Indicates participants whose BMI moved to a higher risk category between baseline and W5.

Table 2

Estimates from linear regression models[1] examining associations of W5 BMI and BMI change (W5-baseline) with Power of Food Scale (PFS) domain and aggregate scores, adjusted for baseline demographics (sex, race/ethnicity, Family Affluence Scale, parent education) and W5 height and vigorous physical activity

	W5 BMI (kg/m^2)			BMI change (W5 - baseline, kg/m^2)		
	β	SE	*P*	β	SE	*P*
PFS						
Domain 1, food available	0.32	0.29	.30	0.42	0.19	.04
Domain 2, food present	0.34	0.26	.25	0.21	0.16	.21
Domain 3, food tasted	−0.15	0.21	.48	0.03	0.11	.81
Aggregate[2]	0.22	0.30	.47	0.26	0.18	.16

BMI- body mass index, W5- wave 5 (5 years after baseline)

[1] Separate models estimated associations with PFS aggregate and domain scores.

[2] PFS scores range from 0-5. The aggregate score is calculated as the mean of the three domain scores.

Table 3

Relative risk (RR) and 95% confidence intervals (CI) of high W5 BMI (BMI≥25 kg/m^2), onset of overweight and obesity (OWOB) and dieting associated with PFS domain and aggregate scores, adjusted for baseline demographics (sex, race/ethnicity, Family Affluence Scale, parent education) and W5 height and vigorous physical activity

	High W5 BMI			OWOB onset			Dieting		
	RR	**95%CI**	***P***	**RR**	**95%CI**	***P***	**RR**	**95%CI**	***P***
Power of Food Scale									
Domain 1, food available	1.08	[0.90, 1.30]	.42	1.20	[0.97, 1.48]	.10	1.15	[1.02, 1.29]	.02
Domain 2, food present	1.09	[0.97, 1.23]	.13	1.15	[0.96, 1.39]	.14	1.12	[1.03, 1.22]	.01
Domain 3, food tasted	0.89	[0.77, 1.03]	.12	0.94	[0.79, 1.13]	.50	1.05	[0.96, 1.14]	.27
Aggregate[2]	1.03	[0.88, 1.19]	.74	1.12	[0.91, 1.37]	.29	1.13	[1.01, 1.26]	.03

W5- wave 5 (5 years after baseline)

[1]PFS aggregate score is calculated as mean of the three domain scores.