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A Prospective Study of Adolescent Eating in the Absence of Hunger and Body Mass and Fat Mass Outcomes

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Abstract

Objective—Eating in the absence of hunger (EAH) refers to the consumption of palatable foods in a sated state. It has been proposed that EAH promotes excess weight gain in youth; yet, there are limited prospective data to support this hypothesis. We examined whether EAH at baseline predicted increases in body mass (BMI and BMIz) and fat mass (kg) 1 year later among adolescent boys and girls.

Design and Methods—EAH was assessed as adolescents' consumption of palatable snack foods following eating to satiety from an *ad libitum* lunch buffet. Parents also completed a questionnaire about their children's EAH. Body composition was assessed using air displacement plethysmography.

Results—Of 196 adolescents assessed for EAH at baseline, 163 (83%) were re-evaluated 1 year later. Accounting for covariates, which included respective baseline values for each dependent variable, race, height, age, sex, and pubertal stage, there were no significant associations between

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baseline observed or parent-reported EAH and change in adolescent BMI, BMIz or fat mass. Results did not differ by sex, child weight status, or maternal weight status.

Conclusions—We found no evidence to support the hypothesis that EAH is a unique endophenotype for adolescent weight or fat gain.

Keywords

eating in the absence of hunger; eating behavior; body mass index; fat mass; obesity; overweight; prospective; longitudinal

Introduction

Eating in the absence of hunger (EAH) refers to consumption of palatable foods in a sated state (1). EAH is objectively measured using a free-access procedure during which youth are given an opportunity to consume highly palatable snack foods after eating a meal to satiety (2, 3). Cross-sectional studies indicate that youth with overweight and obesity demonstrate greater EAH than non-overweight youth (3, 4, 5, 6, 7, 8, 9). Likewise, children and adolescents' fat mass and body mass index standard deviation scores (BMIz) positively correlate with observed EAH (4, 10, 11). Some, but not all, studies suggest the association between EAH and weight is sex-specific. Two investigations reported that boys with overweight and obesity engaged in greater EAH than non-overweight boys (12, 13). Girls with overweight and obesity have been found to engage in less (12, 13) or more EAH (1, 5, 10) than non-overweight girls.

EAH can also be assessed subjectively by questionnaire (14), and its association with weight is inconsistent. Youth's reports of their own EAH were positively associated with fat mass in one study (11), but not another (15). Parents of overweight adolescents reported that their children engaged in greater EAH than parents of non-overweight youth, and parental reports were positively associated with continuous measures of adolescent BMIz and fat mass (15). Notably, parent-reported EAH in response to external food cues, but not self-reported EAH, was associated with adolescents' observed EAH (15), suggesting parents may be more valid informants of their children's EAH in response to external cues. Existing questionnaire data on EAH and weight are cross-sectional.

Frequently it is presumed that EAH is a unique behavioral pathway, separate from overeating when hungry, that leads to excessive weight and fat mass gain; yet, prospective data are scant. The landmark study examining this hypothesis (8) enrolled a cohort of 171 non- Hispanic white girls and measured observed EAH repeatedly. Averaged across five, seven and nine years of age, observed EAH was positively associated with BMI change during the same time frame, after controlling for baseline BMIz (8). This association was significant only among girls with overweight mothers, but not among girls with non-overweight mothers. Analysis of the relationship between average EAH during childhood and BMI change during the same period suggests that EAH and BMI track together; however, it is unclear to what extent EAH serves as a prospective risk factor for excessive BMI gain, beyond initial BMI.. In a separate large, prospective study of Hispanic children (4-19y), observed EAH was related to youth's BMI cross-sectionally (6), but did not

significantly predict weight change after controlling for baseline BMI (7). Additional prospective data are required to evaluate the role of EAH as a predictor of undue weight and fat change in pediatric samples. Establishing whether EAH is a prospective risk factor for excessive gains in body mass may have implications for prevention, as intervening at an early time point could reduce obesity risk.

The aim of the current study was to evaluate the impact of EAH on BMI, BMIz, and fat mass change prospectively in adolescent boys and girls. Adolescence is a salient developmental period to evaluate the role of eating behavior in body weight and composition change, as EAH increases as youth age (2, 5, 9, 16) and peaks during adolescence (6). We hypothesized that EAH would be a positive and significant predictor of change in BMI, BMIz, and fat mass. We evaluated observed EAH and parent-reported EAH as predictors. Given inconsistencies in EAH patterns by sex, child weight status, and maternal weight status, we tested these variables as moderators of the association between EAH and BMI, BMIz and fat mass change.

Methods

Participants and Procedure

Participants were healthy adolescent girls and boys ages 13 to 17 years recruited through flyers and listservs in the Washington, DC greater metropolitan area to take part in a study of eating behaviors (ClinicalTrials.Gov ID: NCT00631644). Exclusion criteria included: a major medical or psychiatric condition; use of medication affecting appetite or weight; pregnancy; active participation in weight loss treatment; or reported dislike of >50% of foods offered at the laboratory meals. The Institutional Review Board of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development approved the study protocol. Adolescents and parents/guardians provided written assent and consent, respectively. Adolescents were compensated up to $210 for their time. Participants were instructed to begin fasting at 10:00 PM the night prior to all of their visits. The following measurements were completed at baseline and again at a 1-year follow-up appointment at the National Institutes of Health Clinical Research Center.

Measures

Body composition—Participant's weight was measured to the nearest 0.1 kg with a calibrated digital scale. Height was determined with a calibrated wall stadiometer from the average of three measurements recorded to the nearest millimeter. BMI (weight in kg/[height in m^2]) and BMIz were calculated according to the Centers for Disease Control and Prevention 2000 standards (17). Fat mass and fat-free mass (kg) were estimated by applying the Siri equation to body density measurements from air displacement plethysmography (Life Measurement Inc., Concord, CA) obtained while participants were wearing underclothes as previously described (18, 19). Parents self-reported their height and weight, which were used to calculate parental BMI (17).

Puberty—Breast development was assigned during a physical examination by an endocrinologist or trained nurse practitioner according to the five stages of Tanner (20);

testicular volume (mL) was measured using a set of orchidometer beads as standards according to Prader (21). These measurements were used to categorize participants into pre-puberty (Tanner stage 1), early puberty (stage 2), mid-puberty (stage 3), late puberty (stage 4), or adult standard (stage 5).

Observed eating in the absence of hunger (EAH)—Adolescents were instructed to adhere to a fast (only water) initiated at 10:00 PM the night before their visit. As previously described (4), at 11:00 AM, each participant was served a multi-item buffet meal consisting of approximately 11,000 kcal of lunch-type foods that varied in macronutrient content (55% carbohydrate, 33% fat, and 12% protein; Supplemental Table 1). Each meal was served to individual participants in a private room. Youth were instructed: "Please eat until you are no longer hungry. Take as much time as you need, and open the door when you're done." Ratings of state hunger and fullness were ascertained immediately following the meal using visual analog scales (1 = *none* to 100 = *extremely*). Participants were led to a quiet room to read magazines devoid of food images. They then viewed a 4-min 20-sec film clip.[1] Following the film, each participant was provided with a 4,000 kcal array of palatable snack foods (Supplemental Table 1; (4). Consistent with the original EAH paradigm (1, 2, 6, 10, 16) and to provide a context in which to present a variety of novel, palatable food items, participants were instructed: "Please taste each of the foods. Rate your preferences for how much you like or dislike the foods on this rating form. Try to take at least two bites of each food. When you are done, eat as much of the foods as you would like and feel free to use any of the activities in the room." Activities included a handheld computer game, playing cards, magazines devoid of food content, word games, and drawing materials. Participants were left alone for 15 minutes. The EAH snack array was served 50 minutes after the initiation of the buffet lunch meal and approximately 30 minutes after the average teen finished eating. EAH was measured by calculating the difference in weight (g) of each snack before and after the eating period. Energy intake (kcal) was calculated with data from the U.S. Department of Agriculture National Nutrient Database for Standard Reference (Agricultural Research Service, Beltsville, MD) and food manufacturer nutrient information obtained from food labels.

Parent-reported eating in the absence of hunger (EAH)—Parents described their perceptions of their children's EAH in response to external food cues using the Eating in the Absence of Hunger Questionnaire for Children – Parent Report about Child (11, 15). Items for this measure (e.g., eating beyond satiety because the food "looks, tastes or smells so good") are rated on a 5-point scale from 0 (*never*) to 4 (*always*). This subscale has demonstrated good internal consistency and temporal stability among children and adolescents (11). It has also demonstrated convergent validity with a laboratory measure of EAH among adolescents (15).

[1]The film clip was administered as part of a study evaluating the link between manipulations in state mood and EAH. Film clip exposure was controlled for in all analyses; its lack of significant association with any indicator of EAH is noted in the results.

Data Analysis

Analyses were conducted using IBM SPSS Statistics 22. All variables were normally distributed. Independent samples *t*-tests were used to compare youth who did and did not complete the 1-year follow-up. Independent samples *t*-tests and analyses of variance with post-hoc tests were used to examine group differences in the primary variables of interest by sex and weight status (0=non-overweight BMI<85th percentile, 1=overweight BMI≥85th percentile); a Mann-Whitney U Test and Kruskal-Wallis H Test were used to examine differences in median pubertal stage. Bivariate correlations, conducted separately for the baseline and 1-year time points, examined continuous associations of observed and parent-reported EAH with body measurements.

To test the primary hypotheses of interest, hierarchical regression models were used to determine if initial EAH predicted ΔBMI, ΔBMI*z*, and Δfat mass at the 1-year follow-up; separate models were conducted for observed and parent-reported EAH. We also examined the relationship between average EAH (from baseline to follow-up) and weight-related outcomes. All models adjusted for the baseline value of the associated dependent variable [grand mean centered; (22)], Δ age between baseline and follow-up (y), sex, race (0=non-Hispanic white, 1=other), baseline pubertal stage, Δ height (cm) between baseline and follow-up, film condition (0=neutral, 1=sad), and total energy intake (kcal) at the lunch meal. Two-way interactions with each indicator of EAH (grand mean centered) were evaluated for child sex, child weight status, and maternal weight status (0=non-overweight BMI kg/m^2 <25, 1=overweight BMI ≥25) (17). Additionally, we evaluated whether film condition moderated any observed effects of EAH on body outcomes. This variable had no main or interactional (*ps* =NS) effect on any outcome (data not shown). To control for multiple comparisons, the Benjamini-Hochberg procedure for false discovery rate (23) was applied to each family of analyses.

Statistical models in which missing values were imputed for all outcome variables and predictor variables were conducted to ensure that results were not related to attrition bias. Missing values were imputed using the fully conditional specification method and linear regression model type in SPSS. Five imputations with 100 iterations between successive imputations were performed, and pooled estimates were evaluated. Results did not differ in direction or significance from the non-imputed data and thus are not shown.

Results

Sample Characteristics

196 adolescents (39.5% overweight) and 179 parents (60.1% overweight) completed a baseline visit. Characteristics at baseline, as well as unadjusted group differences by sex and weight status, are shown in Table 1. Following the *ad libitum* lunch meal, adolescents reported low levels of hunger (Mean±SD 9.8±13.0, range 1-100) and high levels of fullness (66.0±23.3), confirming the meal was effective in inducing satiety. At baseline, adolescents consumed an average of 318±152 kcal (range 39.1-1042.6) during the EAH snacks array; fullness ratings were not associated with observed EAH (r = +.06, p =NS). In unadjusted analyses, boys demonstrated more EAH than girls (t = 2.36, p <.02), but there were no sex

differences in parent-reported EAH (t = -.79, p =NS). Observed EAH did not differ by adolescent weight status (t = -1.84, p =NS), but parent-reported EAH was higher among overweight versus non-overweight adolescents (t = -4.78, p <.001). In unadjusted analyses evaluating sex by weight status interactions among baseline data, observed EAH for non-overweight girls was lower compared to all other participants (p <.01), who did not differ from one another (p =NS). Parent-reported EAH was higher for overweight girls relative to the non-overweight girls and boys (p <.01), but did not differ from overweight boys (p =NS).

163 (83%) youth completed the 1-year follow-up. Most participants (n = 28; 85%) who did not complete a 1-year visit were lost to follow-up. Two were excluded because they enrolled in another intervention research study, and one was excluded after developing health problems. There were no differences between youth who did and did not complete a follow-up visit in any baseline demographic, energy intake, body or body composition variable (ps =NS).

Baseline Associations between EAH and Body Measurements

When examining continuous measures of baseline BMI, BMIz, and fat mass, both BMI (r = .19, p <.02) and BMIz (r = +.24, p <.001) were correlated with observed EAH, although fat mass was not (r = +.13, p =NS). BMI, BMIz, and fat mass were correlated with parent-reported EAH (rs = +.26 to +.36, ps <.002).

One-year Follow-up EAH Comparisons by Sex and Weight Status

At the 1-year follow-up, adolescents consumed an average of 323±149 kcal (range 96.3-1074.0) during the EAH snacks array. Boys and girls did not differ in observed (t = 1.86, p =NS) or parent-reported EAH (t = -.32, p =NS) at follow-up, in unadjusted analyses. However, follow-up observed (t = -2.29, p <.02) and parent-reported (t = -3.38, p <.001) EAH was higher for overweight versus non-overweight youth. The interaction between sex and weight status for observed EAH at 1-year was non-significant (p =NS). Parent-reported EAH was higher for overweight boys and girls relative to non-overweight boys and girls (p <.01). At 1-year, BMIz was correlated with observed EAH (r = +.21, p <.01), although BMI (r = +.14, p =NS) and fat mass were not (r = +.09, p =NS). BMI, BMIz, and fat mass all correlated with parent-reported EAH (rs = +.24 to +.29, ps <.003) at 1-year. Both observed EAH and parent-reported subjective EAH were moderately consistent over the two time points, with bivariate correlation coefficients of .51 and .54, respectively.

Prospective Associations between Observed EAH and Changes in BMI, BMIz, and Fat Mass

After controlling for covariates, there were no significant associations between observed EAH at baseline and ΔBMI, ΔBMIz, or Δfat mass (ps =NS; Table 2). Interactions between observed EAH and sex, child weight status, and maternal weight status were also non-significant (ps =NS). In regards to observed EAH expressed as an average of baseline and follow-up, neither main effects nor interactions with sex, child weight status, or maternal weight status were significant for any body measurement outcome (ps =NS).

Prospective Associations between Parent-Reported EAH and Changes in BMI, BMIz, and Fat Mass

After controlling for covariates, there was no significant association between baseline parent-reported EAH in response to external cues and ΔBMI, ΔBMIz, or Δfat mass (*ps* =.NS; Table 3). Interactions between parent-reported EAH and sex, child weight status, and maternal weight status were also non-significant (*ps* =NS). None of the results for observed or parent-reported EAH changed when also controlling for participants' state fullness ratings after the baseline lunch array, baseline fat-free mass (kg), or change in fat-free mass from baseline to 1-year (data not shown).

Discussion

Despite significant cross-sectional associations between EAH and BMIz at both baseline and follow-up, neither observed nor parent-reported EAH significantly predicted changes in adolescents' BMI, BMIz, or fat mass over the course of one year. Results remained non-significant when EAH was defined as a single eating episode or expressed as an average of two repeated measures obtained at baseline and follow-up. Interactions with sex, child weight status, and maternal weight status were also non-significant. These data are inconsistent with one prior prospective evaluation in which a significant association was identified between average observed EAH and BMI gain among young girls of overweight mothers (8). However, the current data are consistent with a second evaluation conducted in Hispanic children and adolescents that found null prospective effects of observed EAH on weight gain (7).

Consistent with prior cross-sectional data (15), parents rated overweight adolescents' EAH in response to external cues higher than parents of non-overweight youth at both baseline and 1-year. Further, overweight youth were observed engaging in more EAH at the 1-year follow-up, and a continuous measure of BMIz was related to observed EAH within time, at both intervals. Yet, these cross-sectional variations in EAH did not translate into prospective increases in BMI, BMIz, or fat mass.

A number of possibilities may account for the lack of significant prospective findings. One distinct possibility is that EAH is not an eating behavior phenotype (11) that uniquely promotes excessive increases in BMI or fat mass during adolescence. EAH may already have sufficiently impacted body weight during childhood (2, 3) such that baseline body weight or fat mass accounts for any effect it might exert during adolescence. Another alternative is that EAH is a related characteristic of other distinct eating patterns that promote excessive weight gain. Prospective literature illustrates that loss of control eating patterns predict excessive weight gain among children and adolescents (24, 25, 26, 27, 28). EAH demonstrates significant overlap with loss of control and other forms of disinhibited eating (11, 14), but in and of itself may not lead to excess gain among adolescents. Post-hoc power analyses suggest that a sample size of over 600 children would be required for the observed effect of EAH on change in fat mass to reach statistical significance and thousands more would be needed to observe a significant effect of EAH on BMI change. These analyses lend further support to the notion that EAH may not be a significant, independent contributor to adolescents' growth trajectory. EAH could be more predictive of excess

weight and fat gain during other developmental stages, such as preadolescence (11). EAH appears to peak around the age of 13 (6) and the current study included adolescents with an average age of 15 years, the majority of whom were in late puberty. Thus it is possible that not recruiting younger participants may have attenuated any potentially significant association between EAH and changes in BMI, BMIz or fat mass. It may be necessary to study larger cohorts with more variation in development to separate the weight gain that is due to interrelated factors such as EAH and baseline body composition.

Null findings in the current study may also be attributed to the length of the follow-up period. Previous studies reporting a link between excess weight gain and pediatric disinhibited eating examined changes over a three to five year period (8, 24, 25, 26, 28). The current study and Butte et al. (7) examined weight-related changes over the course of one year. It is possible that a longer follow-up interval is required to detect the impact of EAH on body weight gain, as well as on changes in weight status. Additionally, although the EAH paradigm we utilized was very similar to prior studies (1, 2, 6, 10, 16), it differed from other prospective studies (7, 8) in that our lunch meal was larger and designed to eliminate, not just reduce, hunger (4). Although meal types across studies demonstrated convergent validity with one another, slight differences in post-meal intake [~70 kcal; (4)] may account for some of the disparate findings. As part of a separate study assessing the link between negative affect and EAH, participants in our study were also exposed to a brief film clip prior to the snack array. Although this manipulation did not have a significant effect on any of our findings, it may have influenced observable EAH to some degree. The current study also only evaluated eating behavior over a 60-minute time frame. Some youth may have compensated for their caloric intake later in the day, thereby minimizing effects on body weight (5, 29, 30, 31). Future studies are needed to observe the temporal associations between EAH and compensatory intake as these associations may be critical to the identification of youth vulnerable to excess weight gain (32).

A strength of the current study is its use of a well-validated laboratory paradigm to assess EAH (4). However, data from this paradigm represent only a single instance of EAH in response to external food cues. Future studies should use ecological momentary assessment to evaluate whether EAH in youth's natural environments in response to a variety of cues (e.g., food availability, negative affect) translates into significant changes in weight. Further, some studies (12, 13) suggest that a social desirability bias may lead overweight youth, who frequently receive strong messages to lose weight (33), to consume less energy in the laboratory than they would in their natural environments. Finally, results from the current study may not generalize to clinical samples, who may engage in more frequent EAH.

In conclusion, neither observed nor parent-reported EAH in response to external food cues was associated prospectively with changes in BMI, BMIz, or fat mass at a 1-year follow-up among a healthy sample of adolescent boys and girls. These findings are in contrast to cross-sectional data. At least among adolescents, we find no evidence that EAH is an independent predictor of weight or fat gain.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Study Importance Questions

Question #1: What is already known about this subject?

- In the majority of studies, youth with overweight and obesity demonstrate more eating in the absence of hunger (EAH) relative to non-overweight youth.
- Child and adolescent EAH is positively correlated with body mass and fat mass in cross-sectional studies.
- Data are limited and mixed regarding the prospective association between EAH and changes in body and fat mass.

Question #2: What does your study add?

- The current study prospectively tested the hypothesis that baseline EAH is an independent predictor of subsequent changes in body mass and fat mass.
- Contrary to hypotheses, neither objective nor parent-reported EAH was independently associated with changes in adolescents' body mass or fat mass.
- The findings do not support the hypothesis that EAH represents a unique endophenotype for adolescent obesity.

Table 1

Baseline and 1-year demographic, anthropometric, and eating in the absence of hunger characteristics for the total sample and by sex and weight status

Characteristic*	Total Sample (*N*=196 baseline; *N*=163 1-year)	Non-Overweight Boys (*n*=48 baseline; *n*=39 1-year)	Overweight Boys (*n*=21 baseline; *n*=17 1-year)	Non-Overweight Girls (*n*=70 baseline; *n*=61 1-year)	Overweight Girls (*n*=56 baseline; *n*=44 1-year)
Baseline					
Age (years)	15.3±1.4 (13.0-18.0)	15.3±1.3 (13.0-18.0)[a]	14.9±1.3 (13.4-17.3)[a]	15.5±1.4 (13.1-17.8)[a]	15.3±1.5 (13.0-18.0)[a]
Pubertal stage (median)	4 (1-5)	4 (1-5)[a]	4 (2-5)[a]	4 (1-5)[a]	4.5 (2-5)[b]
Height (cm)	165.3±8.1 (147.3-189.9)	171.1±9.7 (151.6-189.9)[a]	168.8±7.5 (155.4-184.5)[a]	161.9±5.7 (147.3-177.8)[b]	163.4±6.2 (152.2-177.8)[b]
Weight (kg)	67.3.7±20.3 (35.9-153.0)	60.3±10.8 (38.7-89.2)[a]	76.6±11.3 (56.4-102.3)[b]	54.1±6.5 (35.9-65.3)[c]	86.8±24.1 (55.0-153.0)[d]
BMI (kg/m^2)	24.6±7.1 (16.3-58.9)	20.5±2.1 (16.3-24.9)[a]	26.8±2.3 (23.3-31.0)[b]	20.6±2.0 (16.3-24.8)[a]	32.3±8.2 (22.7-58.9)[c]
BMIz score	.7±1.02 (-2.2-2.8)	.05±.7 (−2.2-1.0)[a]	1.6±0.3 (1.1-2.0)[b]	.07±.6 (−1.5-1.0)[a]	1.8±.5 (1.0-2.8)[b]
Fat mass (kg)	19.2±14.4 (2.3-86.5)	8.2±3.7 (2.3-20.9)[a]	22.8±6.3 (10.7-35.9)[b]	13.2±4.0 (4.0-23.7)[c]	34.8±16.7 (15.2-86.5)[d]
Observed EAH (kcal)	318.3±152.0 (39.1-1042.6)	360.0±171.6 (118.4-1042.6)[a]	349.1±178.1 (39.1-861.5)[a]	266.3±111.3 (63.6-632.0)[b]	344.5±158.7 (82.1-821.8)[a]
Parent-reported EAH	2.3±0.7 (1.0-4.3)	2.2±0.7 (1.0-3.5)[a,b]	2.5±0.6 (1.5-3.3)[a,c]	2.1±0.7 (1.0-4.3)[b]	2.7±0.7 (1.0-4.0)[c]
1-year follow-up					
BMI (kg/m^2)	24.8±6.6 (16.6-54.6)	21.4±2.3 (16.9-26.5)[a]	26.8±3.2 (21.6-31.3)[b]	21.4±2.1 (16.6-25.0)[a]	32.1±8.0 (19.1-54.6)[c]
BMIz	.7±1.0 (-1.9-2.7)	.08±.8 (−1.9-1.3)[a]	1.5±.6 (.3-2.1)[b]	.1±.7 (−1.9-1.1)[a]	1.7±.6 (−.3-2.7)[b]
Fat mass (kg)	19.1±13.5 (1.1-75.6)	8.9±4.3 (1.1-21.6)[a]	22.3±9.6 (8.7-34.8)[b]	14.1±4.9 (−1.9-1.1)[c]	33.8±15.3 (9.7-75.6)[d]
Observed EAH (kcal)	323.3±149.3 (96.3-1074.0)	336.9±149.4 (102.9-712.5)[a]	388.8±138.8 (181.3-612.7)[a]	283.7±112.1 (96.3-515.0)[a]	348.4±194.4 (108.3-1074.0)[a]
Parent-reported EAH	2.2±0.6 (1.0-4.0)	2.1±0.6 (1.0-3.0)[a]	2.4±0.6 (1.0-3.5)[b]	2.1±0.6 (1.0-3.8)[a]	2.4±0.7 (1.0-4.0)[b]

*Values presented are mean ± standard deviation (range), unless otherwise noted as median (range); means followed by different letters on the same line are considered significantly different; BMI = body mass index; EAH = eating in the absence of hunger

Table 2

Associations between observed EAH at baseline and changes in BMI, BMIz, and fat mass at 1-year

Outcome	Steps	Variable Entered	β [a]	*SE*	*b* [b]	R^2 [c]	ΔR^2
BMI 1 yr	Step 1	BMI baseline	−.03	.02	−.12	**.17	**.17
		Δ Age	−.77	.76	−.08		
		Sex	−.13	.33	−.04		
		Race	−.45	.27	−.14		
		Puberty	−.39	.15	*−.21		
		Δ Height	−.16	.03	**−.41		
		Film clip	.17	.24	.05		
		Buffet lunch meal intake	<.001	<.001	.13		
	Step 2	Observed EAH	<.001	.001	.01	**.17	<.001
BMIz 1 yr	Step 1	BMIz baseline	−.07	.03	*−.19	**.16	**.16
		Δ Age	−.04	.17	−.02		
		Sex	−.01	.07	−.02		
		Race	−.05	.06	−.07		
		Puberty	−.08	.03	*−.19		
		Δ Height	−.03	.01	**−.37		
		Film clip	.03	.06	.04		
		Buffet lunch meal intake	<.001	<.001	.10		
	Step 2	Observed EAH	<.001	<.001	.08	**.17	.01
Fat Mass 1 yr	Step 1	Fat mass baseline	.02	.03	.06	.05	.05
		Δ Age	2.30	2.35	.08		
		Sex	.49	.98	.05		
		Race	−1.03	.77	−.12		
		Puberty	−.14	.44	−.03		
		Δ Height	−.16	.10	−.16		
		Film clip	.95	.72	.11		
		Buffet lunch meal intake	.001	.001	.09		
	Step 2	Observed EAH	.002	.002	.06	.16	.003

[a] β = unstandardized regression coefficient at each step

[b] *b* = standardized regression coefficient at each step

[c] R^2 = proportion of variability in the dependent variable accounted for by model; BMI = body mass index; EAH = eating in the absence of hunger

* p < .05

** p < .001

Table 3

Associations between parent-reported EAH at baseline and changes in BMI, BMIz, and fat mass at 1-year

Outcome	Step	Variable Entered	β [a]	*SE*	*b* [b]	R^2 [c]	ΔR^2
BMI 1 yr	Step 1	BMI baseline	−.03	.02	−.13	**.22	**.22
		Δ Age	1.28	.73	.14		
		Sex	−.12	.32	−.04		
		Race	−.49	.25	−.16		
		Puberty	−.36	.15	−.21		
		Δ Height	−.17	.03	−.47		
		Film clip	.07	.23	.02		
		Buffet lunch meal intake	.001	<.001	.17		
	Step 2	External EAH	−.08	.18	−.04	**.22	.001
BMIz 1 yr	Step 1	BMIz baseline	−.06	.03	−.17	**.20	**.20
		Δ Age	.09	.16	.05		
		Sex	.02	.07	.02		
		Race	−.07	.06	−.11		
		Puberty	−.07	.03	−.20		
		Δ Height	−.03	.007	−.43		
		Film clip	.002	.05	.003		
		Buffet lunch meal intake	<.001	<.001	.16		
	Step 2	External EAH	−.03	.04	.04	**.20	.002
Fat Mass 1 yr	Step 1	Fat mass baseline	−.02	.03	−.06	.07	.07
		Δ Age	3.43	2.34	.13		
		Sex	.17	1.00	.02		
		Race	−1.09	.76	−.13		
		Puberty	−.02	.44	.01		
		Δ Height	−.17	.09	−.18		
		Film clip	.74	.72	.09		
		Buffet lunch meal intake	.001	.001	.09		
	Step 2	External EAH	−.52	.53	−.09	.07	.01

[a] β = unstandardized regression coefficient at each step

[b] b = standardized regression coefficient at each step

[c] R^2 = proportion of variability in the dependent variable accounted for by model; BMI = body mass index; EAH = eating in the absence of hunger

** $p < .001$