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Television food advertisement exposure and *FTO* rs9939609 genotype in relation to excess consumption in children

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Abstract

BACKGROUND/OBJECTIVE—Exposure to food advertisements may cue overeating among children, especially among those genetically predisposed to respond to food cues. We aimed to assess how television food advertisements affect eating in the absence of hunger among children in a randomized trial. We hypothesized that the Fat Mass and Obesity Associated Gene (*FTO*) rs9939609 single nucleotide polymorphism would modify the effect of food advertisements.

SUBJECTS/METHODS—In this randomized experiment, 200 children aged 9–10 years old were served a standardized lunch and then shown a 34-minute television show embedded with either food or toy advertisements. Children were provided with snack food to consume *ad libitum* while watching the show and we measured caloric intake. Children were genotyped for rs9939609 and analyses were conducted in the overall sample and stratified by genotype. A formal test for interaction of the food ad effect on consumption by rs9939609 was conducted.

RESULTS—172 unrelated participants were included in this analysis. Children consumed on average 453 (SD=185) kCals during lunch and 482 (SD=274) kCals during the experimental exposure. Children who viewed food advertisements consumed an average of 48 kCals (95% CI: 10, 85; $P=0.01$) more of a recently advertised food than those who viewed toy advertisements. There was a statistically significant interaction between genotype and food advertisement condition (P for interaction = 0.02), where the difference in consumption of a recently advertised food related to food advertisement exposure increased linearly with each additional *FTO* risk allele, even after controlling for BMI percentile.

CONCLUSIONS—Food advertisement exposure was associated with greater caloric consumption of a recently advertised food, and this effect was modified by an *FTO* genotype.

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Conflict of Interest Declaration

The authors declare they have no actual or potential competing financial interests related to the described work.

Supplementary information is available at The International Journal of Obesity's website.

Future research is needed to understand the neurological mechanism underlying these associations.

Introduction

Over one third of U.S. children are overweight or obese¹ putting them at higher risk for adverse health outcomes.²⁻⁴ Food intake must balance with energy expenditure and growth demands in order for children to maintain a healthy body weight. Complex neural regulatory feedback systems monitor available energy stores in the body in order to prompt feeding behaviors to match energy needs.⁵ While these homeostatic mechanisms play a critical role in maintaining energy balance, non-homeostatic (hedonic) pathways can also drive consumption of highly palatable foods.⁶ Such foods are known to activate the dopaminergic mesolimbic pathway that is responsible for detecting rewarding stimuli and motivating behaviors to repeat exposure to those stimuli.⁷ This pathway is also involved in classical conditioning,⁸ i.e. a learning process where a reward-related stimulus can lead to anticipation and motivation for that reward.⁹ Our current obesogenic environment is replete with food-related stimuli, or food cues, that may activate reward pathways and motivate overconsumption.

Food advertising is a highly pervasive source of exposure to food cues. The food industry spends \$1.79 billion marketing primarily energy-dense, nutrient-poor foods to U.S. children under 11 years old,¹⁰ resulting in an average viewing of 15 TV ads per day or 5,500 over each year.¹¹ Given the high prevalence of exposure, it is important to understand whether TV food ads prompt excessive caloric consumption in children.

Randomized studies assessing whether TV food advertising affects consumption in children have had mixed results. Some have shown significantly higher consumption when children view food ads compared to non-food ads.¹²⁻¹⁶ For example, Harris *et al.* showed 118 children, ages 7-11 years, two minutes of food or non-food ads embedded in a 14-minute cartoon and provided crackers to consume while watching the show.¹⁶ Children who viewed the food ads consumed an average of 28.5 g (133 kCals) more than those shown the non-food ads, even though the crackers were not advertised during the session. Others have not observed a main effect of food ad exposure on consumption;¹⁷⁻²¹ however, several of these studies reported effects of food ad exposure in subsets of participants (e.g. boys¹⁷ and children with maternal encouragement to be thin¹⁸), or when looking at specific foods (e.g. celebrity-endorsed foods¹⁹).

Genetic factors likely interact with environmental drivers of eating behavior and could affect how individuals respond to environmental cues to eat. A common variant in the *Fat Mass and Obesity Associated (FTO)* gene was the first genetic factor to be associated with common obesity in large genome-wide association studies.^{22,23} While the biological mechanism is yet unknown pediatric studies suggest that *FTO* may decrease satiety responsiveness and lead to excess consumption.²⁴⁻²⁶ Interestingly, one study (n=24) examined *FTO* rs9939609 in relation to brain response to food images in adult men.²⁷ The authors reported that, for the participants in a fasted state, *FTO* homozygous high-risk participants had a significantly greater response to food vs. nonfood images in brain reward

regions compared to homozygous low-risk participants. This past research motivated our hypothesis that children with the *FTO* rs9939609 high-risk allele would have heightened susceptibility to excess consumption after viewing food ads.

In this study, we tested the effect of food ad exposure on cued eating among children enrolled in a randomized trial and further explored whether a common variant in *FTO* modified that effect.

Subjects and Methods

Participants

We recruited 200 children through community fliers and a contact list from Children's Hospital at Dartmouth between July 2013 and February 2015 (Figure 1). Inclusion criteria included age 9 or 10 years, English fluency, absence of food allergies/restrictions, and absence of health conditions/medication use that may impact appetite or attention span. One caregiver accompanied each child to the visit. Participants were told that the study focused on how children process visual media. Caregivers and children provided written consent and assent, respectively, and Dartmouth's Committee for the Protection of Human Subjects approved all study protocols.

To limit the analysis to unrelated children, one sibling from each of 21 sibling pairs was excluded at random using a computerized random number generator. In addition, four children were excluded because they did not report satiety after lunch, and three were excluded because of protocol violations (i.e., health condition potentially affecting appetite, caregiver interaction with child during experiment). The final analysis sample thus consisted of 172 unrelated children. There were no significant differences between included and excluded children in terms of baseline covariates or consumption (data not shown).

Preload Lunch

Children and one parent were scheduled for a study appointment at 11:30 am or 12:30 pm. Children were instructed to eat a normal breakfast, but not to eat for the two hours prior to the appointment. During the appointment, children were provided lunch with their choice of main dish (macaroni and cheese, pizza bites, or chicken nuggets with ketchup), along with string cheese, carrots and dressing, apple slices, bread, butter, milk, and water. All meals were 1153–1183 kCals to help ensure that children would eat to satiety. Meals were balanced on macronutrients and contained 552–704 kCals of carbohydrates, 315–405 kCals of fat, and 152–188 kCals of protein. Foods were pre- and post-weighed, and nutrient labels and the USDA National Nutrient Database for Standard Reference²⁸ were used to calculate caloric consumption.

After lunch, each child was asked to assess his/her satiety with a 5-point Likert scale ranging from "I am very hungry" to "I am very full". Children who reported, "I am very hungry" or "I am a little hungry" were excluded from the analysis.

Food Advertisement Exposure

After lunch, all children viewed a 34-minute TV show (Figure it Out!, Nickelodeon) that included 7.7 minutes of either food or toy ads, along with 3.1 minutes of neutral ads. The products advertised are listed in Table S2.

Eating in the Absence of Hunger

Our outcome measure was caloric consumption after self-reported satiety, i.e., eating in the absence of hunger (EAH),^{29,30} during the experimental exposure. We provided four snack foods during the experimental exposure: gummy candy (546 kCals), cookies (692 kCals), chocolate (1000 kCals), and cheese puffs (536 kCals). Only the gummy candy was advertised during the food ad condition. Nutritional information for the snack foods is presented in Table S1. Food was pre- and post-weighed, and product nutrition information was used to calculate caloric consumption.

FTO rs9939609 Genotyping

Buccal cell swabs were collected before lunch and stored at room temperature with desiccant capsules (Isohelix, Kent, U.K.). DNA was isolated using DDK-50 isolation kits (Isohelix, Kent, U.K.). Genotyping for rs9939609 was conducted with real-time PCR and Taqman chemistry using the 7500 Fast Real-time instrument (primers and instrument from Thermo Fisher Scientific (Waltham, USA)). All samples were successfully genotyped and there was 100% genotyping consistency among the 10% blinded replicates.

Covariates

We measured children's weight (to the nearest 0.1 kg) and height (to the nearest 0.5 cm), using a digital scale and stadiometer (Model 597KL, Seca, Hamberg, Germany). We calculated body mass index (BMI) percentile using U.S. Center for Disease Control (CDC) 2000 age- and sex-specific distributions.³¹ Healthy weight was defined as <85th percentile, overweight was defined as 85th – <95th percentile and obese was defined as 95th percentile.

Estimated daily energy requirement (EER) was calculated according to Institute of Medicine guidelines using child's sex, measured height and weight, and caregiver-reported daily average physical activity.³² Caregivers answered, "How much time does your child spend doing physical activity such as running around, climbing, biking, dancing, swimming, playing sports, etc.?" separately for school or weekend days. A single weighted average was created and categorized as "sedentary" for <30, "low active" for 30–<60, "active" for 60–<120, and "very active" for 120 min/day.

Caregivers reported their child's race and ethnicity, their highest level of education, their spouse's highest level of education, and their household income category. Caregivers also reported their child's typical number of hours spent watching TV or movies on a weekend day and school day, and responses were used to create a single weighted average. "Parental eating restriction" was calculated as an average of caregiver responses to the restriction subscale questions of the Child Feeding Questionnaire,³³ (e.g., "I decide what my child eats"

answered on a scale of: 1=disagree, 2=slightly disagree, 3=neutral, 4=slightly agree, and 5=agree).

Statistical Analysis

We compared participant characteristics by study condition using unpaired, 2-tailed *t*-tests for continuous measures and χ^2 or Fisher's Exact tests for categorical measures, as appropriate. Next, we examined unadjusted associations between participant characteristics and caloric consumption during the lunch and EAH phases using linear regression; consumption during the EAH phase was computed for total foods and separately for foods that were and were not advertised during the experiment. We then estimated the effect of the experimental condition on EAH for total foods, advertised food, and non-advertised foods separately using multivariable linear regression adjusted for EER and all covariates related to EAH at the $P < 0.10$ threshold. We did not include sex and BMI percentile as covariates because they were used to calculate EER; however, we conducted a sensitivity analysis to determine whether adding those variables to the final models influenced findings. To address whether the *FTO* rs9939609 genotype modified the effect between food ad exposure and EAH, we included a multiplicative interaction term between the exposure and genotype in the adjusted models and used a Wald test to determine the significance of the interaction term.

To assess the robustness of our findings, we repeated the final models after excluding participants who were above the 90th percentile for EAH consumption (>840 kCals). We also performed analyses stratified by sex and weight status, because some previous studies found interactions between food ad exposure and these variables on consumption. We conducted all analyses using SAS 9.4 (SAS Institute, Cary, USA).

Results

The analysis included 172 children who were equally distributed across study conditions (Table 1). Approximately half of the children were male, and they were mostly white (86%) and non-Hispanic (97%). Twenty-three percent of children were overweight or obese, which is slightly lower than the New Hampshire rate of 26%. The mothers of the children were generally highly educated with 78% obtaining at least a college degree. Children watched an average (SD) of 1.4 (1.0) hours of TV per day. Baseline characteristics were balanced across experimental conditions with the exception of EER, which was 145 kCals higher in the toy vs. food arm of the study ($P = 0.03$).

The frequency of *FTO* rs9939609 genotype frequencies (36% TT, 48% AT, and 16% AA) were similar to those of other studies²² and were consistent with Hardy-Weinberg equilibrium (χ^2 test P value = 0.93). There was a strong relationship between the *FTO* genotype and adiposity; the rate of overweight/obesity was 18% among low-risk (TT) children compared to 44% among the highest-risk (AA) children ($P < 0.01$). In our sample, 21% of heterozygotes were overweight/obese, a rate similar to that for homozygous low-risk participants ($P = 0.68$). The *FTO* genotype was not associated with any other child, caregiver or household characteristic at the $P < 0.05$ significance level (Table S3).

Participants across both experimental conditions consumed an average (SD) of 453 (185) kCals during lunch. Being male, having a higher BMI, and having a higher EER were associated with higher caloric consumption at lunch (Table 2).

Participants consumed an additional 482 (SD: 274) kCals during the EAH phase. Baseline characteristics associated with greater total EAH consumption were being male, increased BMI percentile, EER and parental eating restriction (Table 2). There was a statistically significant main effect of the food vs. toy ad exposure on the consumption of the food advertised during the session; children exposed to food ads consumed, on average, 44 additional calories of gummy candy than children exposed to toy ads ($P = 0.02$). There was no main effect of experimental condition on total consumption or on foods not advertised during the experiment.

In analyses adjusted for EER and parental eating restriction (Table 3), the association between food ad exposure and consumption of the advertised food remained similar ($\beta = 48$ kCals, $P = 0.01$). There were no main effects for total consumption ($P = 0.21$) or consumption of foods not advertised ($P = 0.98$). We conducted a Bonferroni correction of the significance level for the 3 hypothesis tests of consumption conducted (total food, advertised food, non-advertised food) and the association between food ad exposure and consumption of advertised food was still significant at the $P < 0.017$ level.

In a sensitivity analysis that also included sex and BMI percentile in the final models, results were not substantially changed. Findings also remained unchanged when the analysis was restricted to individuals below the 90th percentile of EAH consumption. In addition, findings were unchanged in analyses restricted to white participants.

There was a significant interaction between the *FTO* rs9939609 genotype and food ad exposure with advertised food consumption (P for interaction = 0.02) (Figure 2). The magnitude of the association between food ad exposure and consumption increased linearly with each additional risk allele; the estimate (95% CI) for the TT, AT, and AA genotypes were -3 ($-64, 59$), 59 ($4, 115$), and 125 ($16, 233$) kCals, respectively. Stratum-specific estimates also increased linearly when considering total foods rather than only the advertised food, though the estimates were not significant, nor was the interaction. There were no significant interactions between participant sex or weight status with food ad exposure on consumption (data not shown).

Discussion

In this randomized study, we observed a significant interaction between the *FTO* rs9939609 genotype and food ad exposure on the consumption of a recently advertised food. Our results suggest that the *FTO* obesity-risk allele may confer children with a predisposition to heightened consumption in response to food cues. Although we did not find evidence of a generalized effect of food ad exposure on overall cued eating, we did find that exposure to food ads influenced the consumption of a recently advertised food. During just 34 minutes of TV viewing, children who viewed a show with embedded food ads, including one ad for gummy candy, ate an average of 48 more kCals of gummy candy than children who viewed

toy ads. Moreover, that consumption occurred immediately after children reported eating lunch to satiety, reflecting excessive intake. The implications of such findings are concerning given the frequent exposure that children have to TV food ads and that the majority of television food advertising is for energy-dense, nutrient poor foods.³⁴

Food cues such as those present in food advertising are thought to drive non-homeostatic pathways of food consumption via their incentive-motivational properties acquired via classical conditioning.^{9,35} The nucleus accumbens, part of the dopaminergic reward pathway of the brain, is the likely functional interface between motivational food cues and consumptive behaviors,³⁶ and functional neuroimaging studies have observed activation of the nucleus accumbens in response to food advertisements.^{37,38} In addition, studies also suggest that the neural reward response to food cues is greater for participants with *FTO* rs9939609 obesity risk alleles, independent of adiposity.^{27,38} *FTO* rs9939609 may regulate dopamine (D2)-dependent reward learning,³⁹ so the increased responsiveness to food cues may be a result of heightened prior conditioning. We hypothesize that genetic differences in the neural reward response to food cues underlies our observed behavioral findings; however, further research is needed to better understand genetic differences in the effect of food cues on overconsumption.

Ten studies performed by five unique research groups have explored measured food consumption in children during or after viewing TV food ads (systematically reviewed by Boyland in 2016⁴⁰).^{12–21} Of these studies, five reported a main effect of food ad exposure.^{12–16} A series of school-based experiments in the U.K. found that children consumed more after viewing a TV program preceded by 8–10 food ads compared to toy ads.^{13–15} While compelling, the non-naturalistic presentation of ads in a single block before the show may have suggested the study goals to participants and thereby influenced behavior. However, two other studies presented the ads during more naturalistic commercial breaks, as we did in our study, and showed increased consumption related to food ad exposure.^{12,16} The remaining five studies did not find a main effect of food advertising exposure on consumption.^{17–21}

The fact that the advertising effect was only observed for an advertised food differs from the majority of previous studies on cued eating that have demonstrated that the effect of advertising extends to non-advertised foods, a “beyond-brand” effect.⁴⁰ Unlike our study, however, most of those previous studies only provided participants with foods that were not advertised during the experimental session and did not also provide them the choice of an advertised food. Only one other study provided participants with the choice between an advertised and non-advertised food; in that study, exposure to an ad for a celebrity-endorsed potato chip increased consumption of that specific chip, but did not impact consumption of chips with a generic label.¹⁹ Like our study, that study also did not observe a significant difference in consumption of the non-advertised food across study condition. We do not deem our study findings a contradiction of a beyond-brand effect, but consider them evidence of a preference for consuming advertised food when provided, a behavior that was not captured in most previous studies. The specificity of our food ad effect could also relate to the particular characteristics of the gummy candy, such as macronutrient composition, that differ from those of the other provided foods (cookies, chocolate, cheese puffs). In

Our study was also limited by its examination of a single genetic obesity risk factor, rs9939609. This polymorphism has shown one of the strongest associations with child obesity, however, so understanding potential mechanisms of its action are of both scientific and public health importance. This is also one of the first studies to examine genetic predisposition to reactivity to food cues and the first examination of this research area in children. Our study cannot rule out the possibility that unmeasured confounders, like other genetic loci related to excess consumption, were unbalanced between study arms and could have contributed to the association we report. Future, larger studies are necessary to study multiple genetic obesity risk factors related to food cue reactivity.

Conclusion

In this randomized experimental trial, exposure to TV food ads was associated with increased caloric consumption of a recently advertised food in children who had already eaten a meal to satiety, and that association was modified by the *FTO* rs9939609 obesity-risk allele. Future research is needed to understand the neurological mechanism underlying these associations and whether other genetic risk factors also influence reactivity to food cues. Given the high exposure that children have to ads marketing unhealthy foods, the observed cued eating may have a substantial impact on children's dietary choices.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Study concept and design: Gilbert-Diamond, Lansigan, Kelley, Heatherton, Sargent

Acquisition of data: Gilbert-Diamond, Lansigan, Rapuano

Analysis and interpretation of data: Gilbert-Diamond, Emond, Sargent

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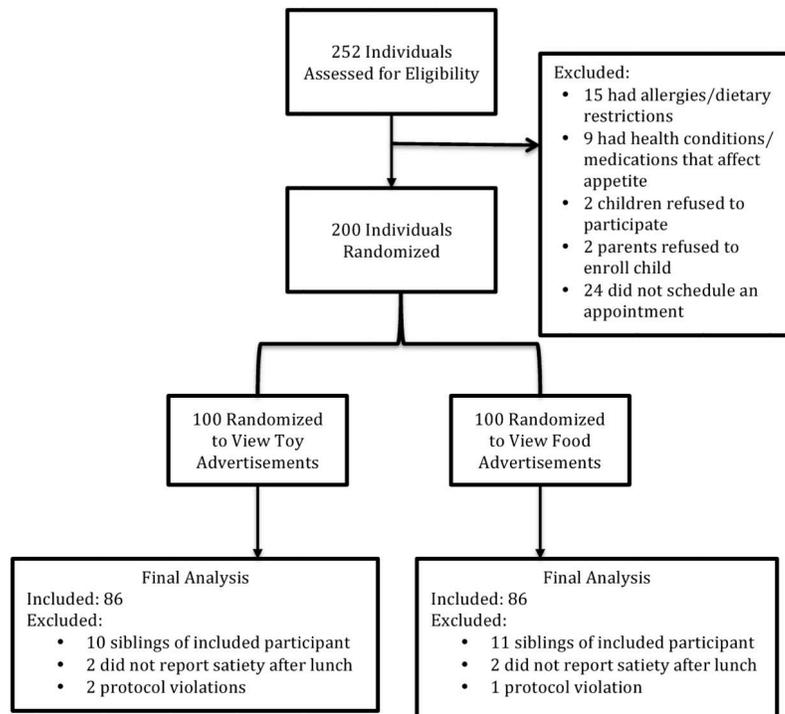


Figure 1.
Participant Flow Diagram

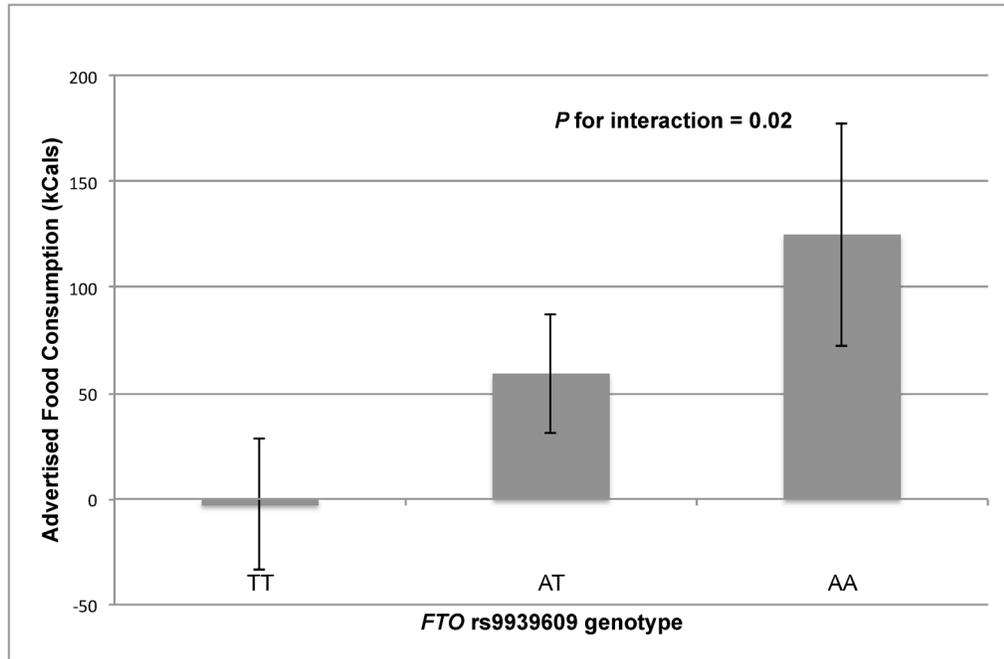


Figure 2. *FTO* rs9939609 stratum specific estimates (\pm standard error bars) of the association between food ad exposure and advertised food consumption after adjustment for estimated daily energy requirement, which was calculated using Institute of Medicine guidelines.³²

Table 1

Selected characteristics of study participants (n=172¹).

Variable	Advertisement Condition		P Value ²
	Toy (n=86)	Food (n=86)	
Age (years), mean (SD)	9.9 (0.6)	9.9 (0.6)	0.62
Male, N (%)	44 (51)	40 (47)	0.54
Race, N (%)			
White	70 (81)	78 (91)	0.08
Non-white	16 (19)	8 (9)	
Ethnicity, N (%)			
Hispanic	4 (5)	2 (2)	0.68
Non-Hispanic	82 (95)	84 (98)	
<i>FTO</i> rs9939609, N (%)			0.63
TT	34 (40)	28 (33)	
AT	39 (45)	44 (51)	
TT	13 (15)	14 (16)	
Maternal Education, N (%)			
Some High School or High School Diploma	4 (5)	1 (1)	0.53
Some Post-High School or Associates Degree	16 (19)	14 (16)	
College Graduate	22 (26)	27 (31)	
Professional or Graduate School	42 (49)	43 (50)	
Household Income, N (%)			
< \$25,000	2 (2)	3 (3)	0.61
\$25,000 – \$64,999	19 (22)	19 (22)	
\$65,000 – \$144,999	40 (47)	41 (48)	
\$145,000 – \$224,999	13 (15)	17 (20)	
> \$225,000	12 (14)	6 (7)	
Weight Status ³ , N (%)			
Healthy Weight: BMI <85 th percentile	63 (73)	69 (80)	0.41
Overweight: BMI 85 th –94.9 th %tile	8 (9)	8 (9)	
Obese: BMI 95 th percentile	15 (17)	9 (10)	

Variable	Advertisement Condition			P Value ²
	Toy (n=86)	Food (n=86)	Food (n=86)	
Moderate/Vigorous Physical Activity (hrs/day), mean (SD)	2.4 (1.2)	2.1 (1.2)	2.1 (1.2)	0.15
Estimated Daily Energy Requirement (kCals) ⁴ , mean (SD)	2461 (475)	2316 (412)	2316 (412)	0.03
TV and movie viewing (hours/day), mean (SD)	1.4 (1.1)	1.3 (0.9)	1.3 (0.9)	0.59
Parental Eating Restriction (range: 1–5) ⁵ , mean (SD)	3.0 (0.9)	3.0 (1.0)	3.0 (1.0)	0.79

¹Participants missing race=1, maternal education=2, TV/movie viewing = 1

²Calculated from an unpaired 2-tailed *t* test with an equal variance assumption to analyze the difference in means or a χ^2 test to analyze the difference in proportions when expected cell counts were above 5 and Fisher's Exact test when expected cell counts were below 5.

³Based on U.S. Centers for Disease Control (CDC) 2000 growth charts³¹

⁴Calculated using measured height and weight, and reported sex, age, and physical activity level using guidelines from the Institute of Medicine Guidelines³²

⁵From the parental restriction subscale of the Child Feeding Questionnaire³³

Table 2

Unadjusted associations between participant characteristics and consumption during lunch preload and in the absence of hunger (n=172)¹

Variable	β (95%CI) for consumption (kCals/unit predictor) ²		
	Lunch Preload	All Foods	Eating in the Absence of Hunger Phase Food advertised in session Food not advertised in session
Food advertisement exposure	-8 (-64, 48)	19 (-63, 102)	44 (7, 81) -24 (-98, 49)
FTO rs9939609 (per risk allele)	33 (-7, 73)	42 (-18, 102)	17 (-11, 44) 25 (-28, 79)
Male	123 (71, 176)	116 (35, 197)	15 (-23, 52) 102 (29, 174)
Age in years	8 (-40, 56)	-35 (-106, 36)	-22 (-54, 10) -13 (-76, 50)
Maternal Education ³	3 (-30, 36)	12 (-37, 61)	14 (-9, 36) -2 (-45, 42)
Household Income ³	5 (-24, 34)	-21 (-64, 22)	-5 (-25, 15) -16 (-55, 22)
BMI: 10 percentile increase ⁴	21 (12, 31)	35 (21, 49)	8 (1, 15) 27 (15, 40)
Moderate/ Vigorous Physical Activity (Hrs/Day)	6 (-17, 29)	-28 (-62, 7)	-7 (-22, 9) -21 (-52, 9)
Expected Daily Energy Req. (per 100 kCals) ⁵	17 (11, 22)	21 (12, 30)	2 (1, 6) 18 (10, 25)
TV/movie viewing (hours/day)	9 (-20, 37)	21 (-20, 62)	0 (-20, 19) 21 (-15, 58)
Parental Eating Restriction (1 pt. increase) ⁶	4 (-25, 33)	51 (9, 94)	8 (-11, 28) 43 (5, 81)

¹ Participants missing race = 1, maternal education = 2, TV/movie viewing = 1

² From a series of linear regressions with the food ad exposure or participant characteristics as the independent variable and consumption (all foods, advertised food, or non-advertised foods) as the dependent variable

³ Treated as ordinal with categories shown in Table 1

⁴ Based on U.S. Centers for Disease Control (CDC) 2000 growth charts³¹

⁵ Calculated using Institute of Medicine guidelines³²

⁶ From the parental restriction subscale of the Child Feeding Questionnaire (range: 1-5)³³

Table 3

Adjusted associations with consumption in the absence of hunger (n=172)

Variable	Adjusted β (95%CI) for consumption (kCals/unit predictor) ¹		
	Total consumption	Food advertised during session	Foods not advertised during session
Food Advertisement Condition	43 (-34, 120)	48 (10, 85)	-5 (-74, 65)
Estimated Daily Energy Requirement (per 100 kCals) ²	22 (13, 30)	3 (-1, 8)	18 (10, 26)
Parental Eating Restriction (1 pt increase) ³	50 (10, 90)	8 (-12, 27)	42 (6, 78)

¹ From a series of multiple linear regressions that included the food advertisement condition, estimated daily energy requirement, and parental eating restriction as independent variables and consumption (all foods, advertised food, or nonadvertised foods) as the dependent variable.

² Calculated using measured height, weight, and reported sex, age, and physical activity level using guidelines from the Institute of Medicine Guidelines³²

³ From the parental restriction subscale of the Child Feeding Questionnaire (range: 1-5)³³