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PEDIATRIC ORIGINAL ARTICLE

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

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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 were served a standardized lunch and then shown a 34-min 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 advertisement effect on consumption by rs9939609 was conducted.

RESULTS: About 172 unrelated participants were included in this analysis. Children consumed on average 453 (s.d. = 185) kcals during lunch and 482 (s.d. = 274) kcals during the experimental exposure. Children who viewed food advertisements consumed an average of 48 kcals (95% confidence interval: 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 body mass index 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. Future research is needed to understand the neurological mechanism underlying these associations.

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INTRODUCTION

Over one-third of US children are overweight or obese[1] 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] Although these homeostatic mechanisms have 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] that is, 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 US children under 11 years old,[10] resulting in an average viewing of 15 TV advertisements per day or 5500 over each year.[11] Given the high prevalence of exposure, it is important to understand whether TV food advertisements 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 advertisements compared with non-food advertisements.[12–16] For example, Harris *et al.*[16] showed 118 children, aged 7–11 years, 2 min of food or non-food advertisements embedded in a 14-min cartoon and provided crackers to consume while watching the show. Children who viewed the food advertisements consumed an average of 28.5 g (133 kcals) more than those shown the non-food advertisements, even though the crackers were not advertised during the session. Others have not observed a main effect of food advertisement exposure on consumption;[17–21] however, several of these studies reported effects of food advertisement exposure in subsets of participants (for example, boys[17] and children with maternal encouragement to be thin[18]), or when looking at specific foods (for example, celebrity-endorsed foods[19]).

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

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associated with common obesity in large genome-wide association studies.[22,23] Although 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 non-food images in brain reward regions compared with 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 advertisements.

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

PATIENTS 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 of 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, 1 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 (that is, 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 schedule for a study appointment at 1130 or 1230 hours. Children were instructed to eat a normal breakfast, but not to eat for the 2 h 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 Reference[28] 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-min TV show (Figure it Out!, Nickelodeon) that included 7.7 min of either food or toy advertisements,

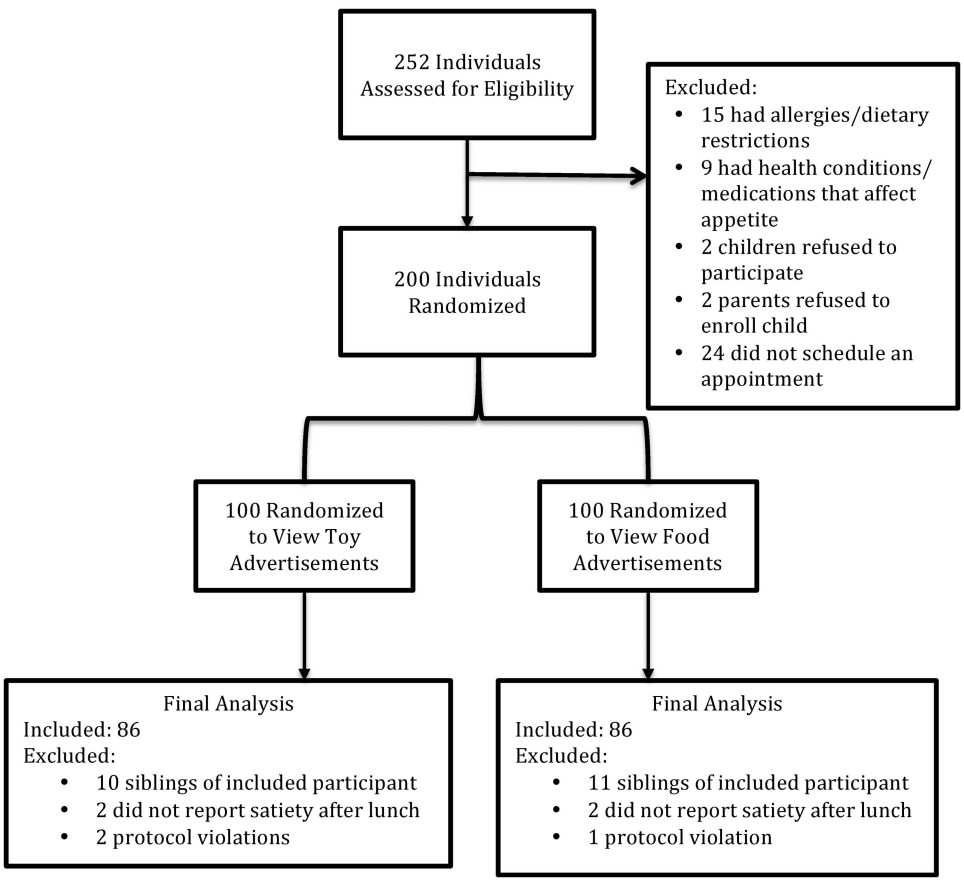

Figure 1. Participant flow diagram.

along with 3.1 min of neutral advertisements. The products advertised are listed in Supplementary Table S2.

Eating in the absence of hunger

Our outcome measure was caloric consumption after self-reported satiety, that is, 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 advertisement condition. Nutritional information for the snack foods is presented in Supplementary 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, UK). DNA was isolated using DDK-50 isolation kits (Isohelix). 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, MA, 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, Hamburg, 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 $\geqslant$85th– $<$95th percentile and obese was defined as $\geqslant$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 $\geqslant$120 min per 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] (for example, '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 X^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 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 advertisement 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 advertisement exposure and these variables on consumption. We conducted all analyses using SAS 9.4 (SAS Institute, Cary, NC, 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 (s.d.) of 1.4 (1.0) h 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) was similar to those of other studies[22] and were consistent with Hardy–Weinberg equilibrium (X^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 with 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 (Supplementary Table S3).

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

Participants consumed an additional 482 (s.d. = 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 advertisement exposure on the consumption of the food advertised during the session; children exposed to food advertisements consumed, on average, 44 additional calories of gummy candy than children exposed to toy advertisements ($P=0.02$). 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 advertisement 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 three hypothesis tests of consumption conducted (total food, advertised food and non-advertised food) and the association between food advertisement 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 advertisement exposure with advertised food consumption (*P* for interaction = 0.02, Figure 2). The magnitude of the association between food advertisement exposure and consumption increased linearly with each additional risk allele; the estimate (95% confidence interval) for the TT, AT and AA

Table 1. Selected characteristics of study participants ($n = 172$[a])

Variable	*Advertisement condition*		P-*value*[b]
	Toy (n = 86)	*Food (n = 86)*	
Age (years), mean (s.d.)	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)	
FTO 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[c]*,* N *(%)*			
Healthy weight: BMI < 85th percentile	63 (73)	69 (80)	0.41
Overweight: BMI 85th–94.9th percentile	8 (9)	8 (9)	
Obese: BMI ⩾95th percentile	15 (17)	9 (10)	
Moderate/vigorous physical activity (h per day), mean (s.d.)	2.4 (1.2)	2.1 (1.2)	0.15
Estimated daily energy requirement (kcals)[d], mean (s.d.)	2461 (475)	2316 (412)	0.03
TV and movie viewing (h per day), mean (s.d.)	1.4 (1.1)	1.3 (0.9)	0.59
Parental eating restriction (range: 1–5)[e], mean (s.d.)	3.0 (0.9)	3.0 (1.0)	0.79

Abbreviation: BMI, body mass index. [a]Participants missing race = 1, maternal education = 2 and TV/movie viewing = 1. [b]Calculated from an unpaired 2-tailed *t*-test with an equal variance assumption to analyze the difference in means or a X^2-test to analyze the difference in proportions when expected cell counts were above five and Fisher's exact test when expected cell counts were below five. [c]On the basis of U.S. Centers for Disease Control (CDC) 2000 growth charts.[31] [d]Calculated using measured height and weight, and reported sex, age and physical activity level using guidelines from the Institute of Medicine Guidelines.[32] [e]From the parental restriction subscale of the Child Feeding Questionnaire.[33]

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 advertisement exposure on consumption (data not shown).

DISCUSSION

In this randomized study, we observed a significant interaction between the *FTO* rs9939609 genotype and food advertisement 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 advertisement exposure on overall cued eating, we did find that exposure to food advertisements influenced the consumption of a recently advertised food. During just 34 min of TV viewing, children who viewed a show with embedded food advertisements, including one advertisement for gummy candy, ate an average of 48 more kcals of gummy candy than children who viewed toy advertisements. 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 advertisements 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] 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] *FTO* rs9939609 may regulate dopamine (D2)-dependent reward learning,[38] 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 advertisements (systematically reviewed by Boyland *et al*.[39]).[12–21] Of these studies, five reported a main effect of food advertisement exposure.[12–16] A series of school-based experiments in the United Kingdom found that children consumed more after viewing a TV program proceeded by 8–10 food advertisements compared with toy advertisements.[13–15] While compelling, the non-naturalistic presentation of advertisements 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 advertisements during more naturalistic commercial breaks, as we did in our study, and showed increased consumption related to food advertisement 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.[39] 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 advertisement for a celebrity-endorsed potato chip increased consumption of that specific chip,

Table 2. Unadjusted associations between participant characteristics and consumption during lunch preload and in the absence of hunger ($n = 172$[a])

Variable	*$\hat{\beta}$ (95% CI) for consumption (Δ kcals per unit predictor)*[b]			
		Eating in the absence of hunger phase		
	Lunch preload	*All foods*	*Food advertised in session*	*Foods 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[c]	3 (−30, 36)	12 (−37, 61)	14 (−9, 36)	−2 (−45, 42)
Household income[c]	5 (−24, 34)	−21 (−64, 22)	−5 (−25, 15)	−16 (−55, 22)
BMI: 10 percentile increase[d]	21 (12, 31)	35 (21, 49)	8 (1, 15)	27 (15, 40)
Moderate/ vigorous physical activity (h per day)	6 (−17, 29)	−28 (−62, 7)	−7 (−22, 9)	−21 (−52, 9)
Expected daily energy requirement (per 100 kcals)[e]	17 (11, 22)	21 (12, 30)	2 (1, 6)	18 (10, 25)
TV/movie viewing (h per day)	9 (−20, 37)	21 (−20, 62)	0 (−20, 19)	21 (−15, 58)
Parental eating restriction (1 pt. increase)[f]	4 (−25, 33)	51 (9, 94)	8 (−11, 28)	43 (5, 81)

Abbreviations: BMI, body mass index; CI, confidence interval. [a]Participants missing race = 1, maternal education = 2 and TV/movie viewing = 1. [b]From a series of linear regressions with the food advertisement exposure or participant characteristics as the independent variable and consumption (all foods, advertised food or non-advertised foods) as the dependent variable. [c]Treated as ordinal with categories shown in Table 1. [d]On the basis of U.S. Centers for Disease Control (CDC) 2000 growth charts.[31] [e]Calculated using Institute of Medicine guidelines.[32] [f]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 $^{\beta}$ (95% CI) for consumption (Δ kcals per unit predictor)*[a]		
	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)[b]	22 (13, 30)	3 (−1, 8)	18 (10, 26)
Parental eating restriction (1 pt increase)[c]	50 (10, 90)	8 (−12, 27)	42 (6, 78)

Abbreviation: CI, confidence interval. [a]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 non-advertised foods) as the dependent variable. [b]Calculated using measured height, weight and reported sex, age and physical activity level using guidelines from the Institute of Medicine Guidelines.[32] [c]From the parental restriction subscale of the Child Feeding Questionnaire (range: 1–5).[33]

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 advertisement 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 and cheese puffs). In addition, although only 1 advertisement for the gummy candy was shown, 6 of the 20 advertisements shown were for candy and those advertisements may have also influenced the consumption of the gummy candy. Future research is necessary to better understand the observed specificity of the advertising effect on consumption.

The implications of our findings for weight gain are unclear given that we did not find a significant difference in total caloric consumption related to advertisement exposure, though we did observe a positive trend for the association. Given the high inter-individual variability in total consumption, our study may have been underpowered to test the effect of food advertisement exposure on this outcome. Unlike four of the five studies that found a significant main effect, our study did not measure consumption in the same children under both conditions. Although a within-participant design may have been more powerful, we chose not to use such a design because of our concerns that participant awareness of study goals could influence their behavior. We posited that children would be more likely to remain naive to the study goals when they were randomized to a single-experiment condition. A larger study will be necessary to more definitively assess food advertisement exposure's effect on total consumption.

Our finding that parental eating restriction of child eating was positively related to total consumption recapitulate results from other groups.[40–42] It is possible that children develop an increased preference for restricted foods.[43] Alternatively, parents may impose more restrictive feeding practices in response to a child's tendency to overeat. Longitudinal studies are necessary to clarify the directionality of this relationship.

This study has the strength of controlling for initial satiety level, thereby enabling us to measure EAH. Indeed, a surprising finding was that children consumed approximately the same number of calories during the EAH and lunch phases, suggesting the inability

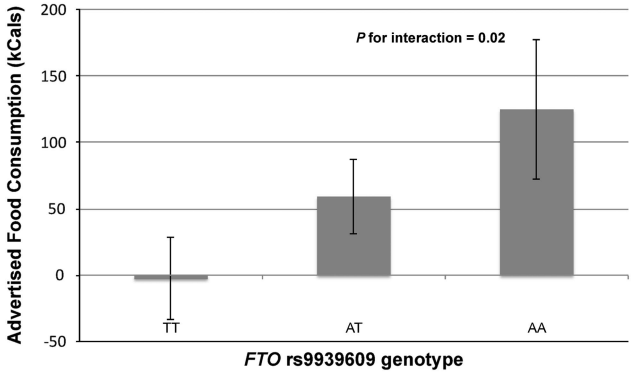

Figure 2. *FTO* rs9939609 stratum-specific estimates (± s.e. bars) of the association between food advertisement exposure and advertised food consumption after adjustment for estimated daily energy requirement, which was calculated using Institute of Medicine guidelines.[32]

of children to regulate caloric intake when presented with highly palatable foods. Through this EAH paradigm, we were able to demonstrate that recent food advertisement exposure prompted children to consume a recently advertised food even when they were full.

Our study was limited in that it measured one instance of cued eating in a laboratory setting, and it is unknown whether children compensate for increased short-term intake by modifying long-term intake, thus, mitigating any effects on excess weight gain. Also, the generalizability of our laboratory-based findings to the home environment is unknown. However, studies suggest that children frequently eat snacks while watching TV.[44] Furthermore, studies also suggest that TV advertisements prompt children to request the purchase of advertised foods,[45,46] which may relate to an increased availability of those foods at home. Thus, it is plausible that exposure to TV food advertisements among children at home may indeed relate to cued eating.

Although TV is still a primary mechanism for advertising to children,[11] food companies are increasingly using other marketing tactics.[10] For example, advergames, internet games that promote brand recognition have been shown to increase short-term caloric consumption in children.[47,48] In addition, the use of celebrity endorsements[19] and character tie-ins[49] is concerning and warrants further research.

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, such as other genetic loci related to excess consumption, were unbalanced between study arms and could have contributed to the association we report. In the 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 advertisements 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 advertisements marketing unhealthy foods, the observed cued eating may have a substantial impact on children's dietary choices.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHOR CONTRIBUTIONS

Dr G-D had full access to the data and takes full responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: G-D, RKL, WMK, TFH, JDS. Acquisition of data: G-D, RKL, KMR. Analysis and interpretation of data: G-D, JAE, JDS. Drafting of the manuscript: G-D, JAE. Critical revision of the manuscript for important intellectual content: All the authors. Statistical analysis: G-D, JAE. Obtaining funding: G-D, TFH, JDS. Administrative, technical or material support: RKL. Study supervision: G-D.

ADDITIONAL CONTRIBUTIONS

Participating children and their caregivers enabled this research study to happen. Horacio Romero Castillo, Archana Ramanujam and other study staff members contributed significantly to running this study. Margaret R Karagas, Department of Epidemiology, Geisel School of Medicine at Dartmouth, obtained additional funding for this study.

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