

Individual Differences in Striatum Activity to Food Commercials Predict Weight Gain in Adolescents

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Objective: Adolescents view thousands of food commercials annually, but little is known about how individual differences in neural response to food commercials relate to weight gain. To add to our understanding of individual risk factors for unhealthy weight gain and environmental contributions to the obesity epidemic, we tested the associations between reward region (striatum and orbitofrontal cortex [OFC]) responsivity to food commercials and future change in body mass index (BMI).

Methods: Adolescents ($N = 30$) underwent a scan session at baseline while watching a television show edited to include 20 food commercials and 20 nonfood commercials. BMI was measured at baseline and 1-year follow-up.

Results: Activation in the striatum, but not OFC, in response to food commercials relative to nonfood commercials and in response to food commercials relative to the television show was positively associated with change in BMI over 1-year follow-up. Baseline BMI did not moderate these effects.

Conclusions: The results suggest that there are individual differences in neural susceptibility to food advertising. These findings highlight a potential mechanism for the impact of food marketing on adolescent obesity.

Obesity (2014) **22**, 2544–2551. doi:10.1002/oby.20882

Introduction

Adolescents in the United States see ~6,000 food commercials each year (1). Most commercials promote calorie-dense, nutrient-poor foods (2) with fast-food restaurants advertising to adolescents more than any other company (3). Food marketing increases children's preferences for and consumption of commonly promoted unhealthy foods (4) and has been implicated as an important contributor to obesity (5). Viewing television advertising (commercial television), rather than television viewing per se (i.e., public television, videos, and DVDs), is positively associated with greater weight gain in children over 5-year follow-up, controlling for physical activity (6). Exposure to advertising for fast food and soft drinks is associated with weight gain over 3-year follow-up in overweight and obese (but not lean children), controlling for hours watching television (4). Yet, little is known about the underlying mechanisms through which food advertising contributes to obesity. One possibility is that repeated exposure to unhealthy food advertising activates brain reward regions in some individuals, prompting cravings for and increase consumption of these foods, contributing to unhealthy weight gain.

Bruce and associates (7) found that healthy-weight children exhibited greater activation in regions associated with motivational value (orbitofrontal cortex [OFC]) and processing of visual stimuli (e.g., occipital gyrus) in response to food logos compared to control logos. Further, obese versus healthy-weight children showed greater responsivity in somatosensory (postcentral gyrus) and reward-related (midbrain) regions to food logos compared with control images (8). Burger and Stice (9) found that adolescents showed greater activation in reward (putamen), gustatory (insula), and visual processing (occipital gyrus) regions in response to Coke soft drink advertisements compared to nonfood advertisements and that habitual Coke consumers versus nonconsumers showed greater activation in regions encoding salience/attention (posterior cingulate, precuneus) toward Coke logo's compared to control images. The only study (10) to examine neural response to food commercials, which used baseline data from the present study, found that adolescents showed elevated activity in regions implicated in motivational value (OFC), attention (anterior cingulate cortex), somatosensory response (postcentral gyrus), and visual processing (occipital gyrus) in response to food commercials versus nonfood commercials and a television program. However, no longitudinal research has examined the associations between neural response to food commercials and future weight gain.

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Funding agencies: This work was supported by National Institute of Diabetes and Digestive and Kidney Diseases grant (R01 DK80760, 8/09-7/14), Rudd Foundation, and the Robert Wood Johnson Foundation.

Disclosure: The authors declared no conflict of interest.

Additional Supporting Information may be found in the online version of this article.

Received: 30 April 2014; **Accepted:** 7 August 2014; **Published online** 24 August 2014. doi:10.1002/oby.20882

Although prospective research has not examined neural response to food commercials, two prospective fMRI studies found that activity in reward-related regions in response to food images is positively correlated with change in body mass index (BMI). One study (11) found that nucleus accumbens (NAcc) activity in response to food images was positively correlated with change in BMI over 6-month follow-up. Another study (12) found that activity in the putamen and OFC in response to palatable food images relative to unpalatable food images was positively correlated with change in BMI over 1-year follow-up in adolescents with a genetic propensity for greater dopamine signaling, but not in those without this genetic vulnerability; however, there were no main effects regarding BOLD response in brain regions to palatable food images and future weight gain in this study. These data suggest that individuals who exhibit greater responsivity in reward-related regions to food cues are more likely to gain weight, but the stimuli used in these studies were food images without branding/context, limiting ecological validity. An improved understanding of how neural response to food advertising is associated with weight gain should enhance our understanding of how and for whom food marketing increases the risk of obesity and may inform the design of obesity prevention programs and policies.

Accordingly, the current study examined the associations between reward-related neural response to food commercials and change in BMI over 1-year follow up in adolescents varying from lean to obese. Because obese versus lean individuals have shown greater reward region responsivity to unhealthy food images (13), we tested whether baseline BMI moderates any relations between reward region response to food commercials and change in BMI.

Methods

A more detailed description of the sample and measures are discussed in Gearhardt et al. (10) which presents cross-sectional results from the present study.

fMRI media paradigm

At baseline, participants (M age = 15.2 ± 1.1 ; M baseline BMI = 26.9 ± 5.4 ; 17 females) were scanned while viewing a video of the television show “Mythbusters” edited to include 20 food commercials and 20 nonfood commercials. Participants rated hunger on a visual analog scale pre- and postscan. Food commercial recall, recognition, liking, and familiarity were assessed after the scan (10).

Body mass index

BMI (kg/m^2) was used to reflect adiposity at baseline and 1 year after fMRI scan. Height was measured to the nearest millimeter and weight was assessed to the nearest 0.1 kg after removal of shoes and coats (see Supporting Information).

Pubertal development

At baseline, adolescents were asked to report on their state of pubertal development using a standardized series of line drawings of youth at various states of pubertal development (10,14).

Energy intake

We used the 60-item Block Food Frequency Questionnaire (BFFQ) (15), which inquires about the frequency of consumption of specific

food types, to assess total energy intake over the past 2 weeks at baseline and 1-year follow-up (see Supporting Information).

fMRI data acquisition, preprocessing, and analysis

A detailed description of the fMRI data acquisition, preprocessing, and analyses are provided elsewhere (Supporting Information). Activation in response to food commercials was assessed by contrasting BOLD response during food commercials versus nonfood commercials and during food commercials versus the television show. Because there were 20 food commercials and 20 nonfood commercials, we used 20 randomly selected segments of the show for the second contrast. Contrast images were constructed within each participant. Consistent effects across subjects were tested using the contrast images in one-sample random effects t tests, controlling for hunger as hunger modulates neural response to food images (16). Morphology-based regions of interests (ROIs) were generated using the Montreal Neurological Institute (MNI) average adult MRI template. We identified the striatum and OFC ROIs using results of previous prospective fMRI studies which found that greater BOLD activation in these regions to food images is associated with weight gain (11,12). For the striatum, we created an ROI that included bilateral caudate, putamen, and NAcc. For the caudate and putamen, we used the automated anatomical labeling atlas (17). Due to the lack of a NAcc ROI in the automated anatomical labeling atlas, we used a spherical ROI (10-mm diameter spheres) that was built centered at MNI coordinates $x = -9$, $y = 6$, $z = -4$ (left NAcc) (11,18) and $x = 9$, $y = 6$, $z = -4$ (right NAcc) (18). Due to the relatively large size of the OFC, we also used a spherical ROI (10-mm diameter spheres) for this region. The ROI was centered at the MNI coordinates $x = 36$, $y = 27$, $z = -15$ (12) and $x = -36$, $y = 27$, $z = -15$. Figure 1 shows the identified ROIs.

Mean activity (parameter estimates) was extracted from these ROIs at the individual level using MarsBar (<http://www.marsbar.sourceforge.net>) and exported to SPSS. Regression analyses were performed testing the associations between activity in the *a priori* ROIs and BMI at 1-year follow-up separately for food commercials versus nonfood commercials and for food commercials versus the television show. Baseline BMI was included as a control variable so that the models functionally predicted change in BMI (19). Sex, pubertal development, and change in energy intake over 1-year follow-up were also included as covariates in the analyses. Exploratory whole-brain analyses were conducted in which we examined the correlations between BOLD response to the food commercials (relative to the control stimuli) and change in BMI. The significance level for these analyses was a threshold of P uncorrected < 0.005 combined with a minimum cluster of $(k) \geq 22$, equal to $P < 0.05$ whole-brain corrected (see Supporting Information).

Results

Of the total sample ($N = 30$), we were unable to collect objective height and weight data at 1-year follow-up for six subjects. For four of these subjects, self-reported height and weight data at 1-year follow-up was used to calculate BMI. Measured baseline BMI correlated highly with self-reported BMI at 1-year follow-up for the participants with both data ($r = 0.98$, $P < 0.05$). Two subjects did not provide any 1-year follow-up data. We used maximum likelihood imputation to

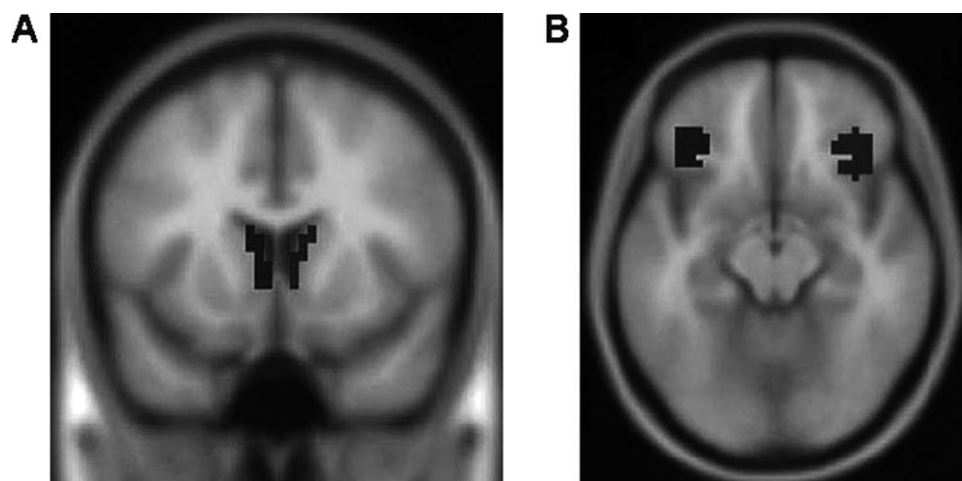


Figure 1 Depiction of the identified ROIs for (A) striatum and (B) orbitofrontal cortex.

estimate BMI for these latter two subjects because this method provides more accurate parameter estimates than listwise deletion or alternative method for handling missing data (e.g., mean substitution) (20). Subjects who did not provide objective weight data at 1-year follow-up did not differ with respect to sex ($F_{1,29} = 2.2, P = 0.2$), age ($F_{1,29} = 0.9, P = 0.4$), or baseline BMI ($F_{1,29} = 0.0, P = 0.9$) compared to those with complete measured BMI data. Weight gain over 1-year follow-up (M BMI change = 1.0 ± 1.4) was significant ($t(29) 3.6, P < 0.001$) and remained significant after excluding subjects without objective weight data ($t(23) = 3.6, P < 0.01$). There were no significant sex differences on baseline BMI and change in BMI and no significant differences between lean, overweight, and obese individuals on change in BMI (Supporting Information). Baseline BMI was not significantly correlated with activation in the *a priori* ROIs (Table 1).

Associations between brain response to food commercials and change in BMI

Activity in the striatum ROI in response to food commercials > nonfood commercials (partial $r = 0.57$; Figure 2A) and in response to food commercials > television show (partial $r = 0.51$; Figure 2B) correlated positively with increases in BMI over 1-year follow-up (Table 2). Identified mean response in the constructed striatum ROI was mainly located in the caudate (Figure 2). This effect was replicated with whole-brain analyses (Table 3). There were no significant correlations between activity in the OFC ROI in response to food commercials versus the control stimuli and change in BMI (Table 2). The effects of the OFC ROI remained nonsignificant when excluding the striatum ROI from the model (food commercials > nonfood commercials: partial $r = -0.03$; food commercials > television show: partial $r = 0.19$). We also did not find any significant correlations between OFC activity in response to food commercials relative to the control stimuli and change in BMI with whole-brain analyses (Table 3). Baseline BMI did not significantly moderate the associations between striatum and OFC activity in response to food commercials and change in BMI (Table 2). The positive correlation between activity in the striatum ROI and change in BMI remained significant when the six subjects without directly measured

follow-up BMI data were excluded from the analyses (food commercials > nonfood commercials: partial $r = 0.57, P < 0.01$; food commercials > television show: partial $r = 0.46, P < 0.05$).

To gain a deeper understanding of the positive associations between activity in the striatum ROI in response to food commercials relative to the control stimuli and change in BMI, we tested whether striatum ROI activity correlated with prescan hunger, change in hunger levels over the scan, change in energy intake over 1-year follow-up, and food commercial recall, recognition, liking, and familiarity. Activity in the striatum ROI in response to food commercials > nonfood commercials was significantly correlated with food commercial recognition ($r = 0.43, P < 0.05$), but not with hunger ($r = 0.24$), changes in hunger ($r = -0.28$), change in energy intake ($r = -0.14$), food commercial recall ($r = -0.03$), liking ($r = -0.03$), and familiarity ($r = -0.14$). The positive correlation between striatum ROI activity in response to food commercials > nonfood commercials and change in BMI remained significant ($r = 0.50, P < 0.05$) when including food commercial recognition in the model. There were no significant correlations between activity in the striatum ROI in response to food commercials > television show and hunger ($r = -0.00$), change in hunger ($r = -0.08$), change in

TABLE 1 Correlations between baseline BMI and BOLD activation in response to food commercials

Variables	1	2	3	4	5
1. Baseline BMI	-				
<i>Food > nonfood commercials</i>					
2. Striatum	-0.07	-			
3. Orbitofrontal cortex	-0.09	0.34	-		
<i>Food commercials > television show</i>					
4. Striatum	0.06	0.52**	-0.21	-	
5. Orbitofrontal cortex	-0.07	0.36*	0.17	0.03	-

*Correlation is significant at $P < 0.05$. **Correlation is significant at $P < 0.01$.

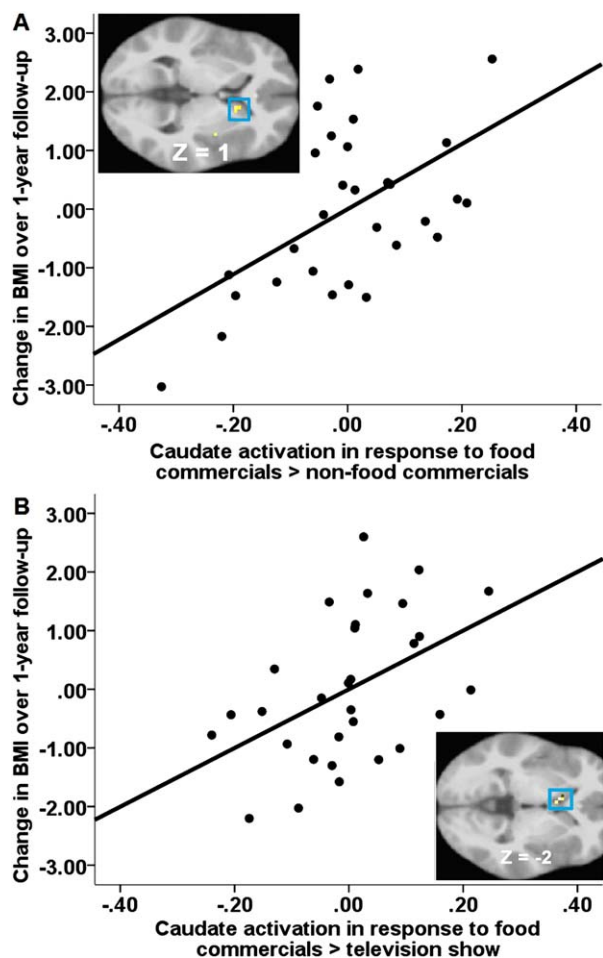


Figure 2 Partial regression plots showing the positive associations between (A) BOLD response in the caudate in response to food commercials > nonfood commercials and (B) BOLD response in the caudate in response to food commercials > television show and increases in BMI over 1-year follow-up, while controlling for baseline BMI. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

energy intake ($r = 0.11$), food commercial recall ($r = 0.18$), recognition ($r = 0.03$), liking ($r = -0.10$), and familiarity ($r = 0.13$).

Exploratory whole-brain analyses identified positive associations between activity in the caudate and regions outside of our hypothesized ROIs (e.g., cuneus, insula, and amygdala) with change in BMI (Table 3).

Discussion

Elevated striatal (i.e., caudate) responsivity to food commercials relative to control stimuli showed large correlations with weight gain over 1-year follow-up. The magnitude of these effects sizes ($M r = 0.54$) is much larger than the effects for established risk factors for future weight gain, such as parental obesity, which has typically shown only moderate predictive relations to future weight gain (r 's 0.18-0.21) (21,22). The caudate appears to encode incentive motivation (23) and reward valuation (24). Elevated caudate responsivity has been linked to increased craving (25) and greater palatable-food

intake (26). The present findings extend prior evidence that elevated striatum response to food images predicted weight gain (11), implying that a similar phenomenon occurs with food commercials.

One possible explanation for the current findings is that some individuals have an elevated reward region responsivity that is biologically based, which may render them more vulnerable to food cue induced cravings for the types of foods shown in the commercials, resulting in greater caloric intake and weight gain. Brain imaging studies have found that a genetic propensity for elevated signaling capacity of dopamine-based reward circuitry is associated with greater reward region responsivity (27,28). Elevated striatum activation in response to palatable food images (relative to unpalatable food images) predicted weight gain in individuals with a genetic propensity for greater dopamine signaling, but not in those without this genetic propensity (12). Therefore, it is possible that the positive association between striatal responsivity to food commercials and future weight gain is partially determined by genetic variability related to dopamine signaling. A second possible explanation for the current findings is that participants with a history of eating the advertised foods undergo a conditioning process in which the brand logos, food images, and even the restaurant settings shown in the commercials become associated with subjective reward from consuming these foods, prompting cravings for and increase consumptions of these food, resulting in weight gain. For these individuals, the food cues appearing in the commercials more readily activate reward regions because of this conditioning history. This interpretation is suggested by animal experiments in which previously neutral cues that are repeatedly paired with palatable food intake eventually develop the capacity to activate brain reward regions via a learning mechanism (29). In support, striatum activity in response to food commercials relative to nonfood commercials was positively correlated with food commercial recognition. This latter result also dovetails with studies that found that striatal activity is associated with successful recognition and active evaluation of salient events, e.g.(30). However, this effect did not occur for the contrast food commercials versus television show, suggesting that this effect was not robust and limits the confidence that can be placed in this finding.

Baseline BMI did not moderate the prospective relations. However, the positive interactions between baseline BMI and striatum activation in response to food commercials versus nonfood commercials ($r = 0.42$) and food commercials versus television show ($r = 0.35$) predicting BMI increases were medium effect sizes, implying that the associations between striatal responsivity, and future weight gain were stronger for heavier youth. The nonsignificance of these interactive effects is most likely due to the small sample size.

Change in energy intake was not significantly correlated with neural activation and change in BMI over 1-year follow-up. However, these null findings should be interpreted with caution because reported energy intake on food frequency measures correlates only weakly with doubly labeled water estimates of actual intake (31).

Stice et al. (11) found that OFC activity in response to food images was positively correlated with change in BMI over 1-year follow-up in adolescents with a genetic propensity for greater dopamine signaling. Further, OFC activity in response to a cue signaling the impending presentation of palatable food images is positively correlated with future weight gain (32). A potential explanation for nonsignificant OFC effect in the present

TABLE 2 Summary of hierarchical regression analysis for variables predicting change in BMI

Variable	<i>B</i>	<i>B</i> (SE)	β	Partial <i>r</i>	ΔR^2
<i>Food commercials > nonfood commercials</i>					
Step 1					0.94
Baseline BMI	1.0	0.1	0.97	0.97***	
Sex	0.2	0.6	0.02	0.07	
Pubertal development	0.5	0.5	0.06	0.22	
Change in total energy intake	0.0	0.0	0.00	0.00	
Step 2					0.02
Striatum	4.9	1.5	0.16	0.57**	
Orbitofrontal cortex	-2.0	1.5	-0.07	-0.28	
Step 3					0.00
Striatum \times baseline BMI	0.9	0.4	0.13	0.42	
Orbitofrontal cortex \times baseline BMI	-0.3	0.3	-0.06	-0.23	
<i>Food commercials > television show</i>					
Step 1					0.94
Baseline BMI	1.0	0.1	0.97	0.97***	
Sex	0.2	0.6	0.02	0.07	
Pubertal development	0.5	0.5	0.06	0.22	
Change in total energy intake	0.0	0.0	0.00	0.00	
Step 2					0.02
Striatum	5.5	2.0	0.13	0.51**	
Orbitofrontal cortex	1.2	1.1	0.05	0.22	
Step 3					0.00
Striatum \times baseline BMI	1.0	0.6	0.09	0.35	
Orbitofrontal cortex \times baseline BMI	-0.2	0.3	-0.04	-0.17	

** $P \leq 0.01$; *** $P \leq 0.001$.

study is that we had a smaller sample ($N = 30$) relative to the previous studies ($N = 44$ and 35 , respectively), reducing sensitivity.

Despite the lack of OFC effects, exploratory whole-brain analyses identified positive correlations between regions implicated reward (caudate, insula, and amygdala) (24, 33), motor (anterior cerebellum and supplementary motor area) (34, 35), memory (middle temporal gyrus, parahippocampal gyrus) (36), emotional- (temporal lobe) (37), and cognitive (posterior cerebellum) (34) processing in response to food commercials and change in BMI over 1-year follow-up. Thus, individuals who potentially evaluate the food featured in the commercials as more rewarding and access memories related to prior experiences of consuming the advertised foods may be more susceptible to weight gain in response to food marketing. Additionally, increased activation in motor regions in response to food cues has been implicated in planning to acquire or consume food (38). Thus, greater activation of the motor regions in response to food commercials may be related to an increased tendency to seek out the calorie-dense foods featured in the advertisements, which may contribute to weight gain. Curiously, we observed that some peaks in visual regions (left middle occipital gyrus, left cuneus) (39) and the cerebellum (right posterior cerebellum) regions were positively related to weight gain, but other peaks in visual regions (right middle occipital gyrus, right superior occipital gyrus, and bilateral lingual gyrus) and cerebellum (left posterior cerebellum) were negatively related to this outcome. This may imply that these different regions of the visual cortex and cerebellum

encode somewhat different processes, or that some of these peaks are chance findings, despite the correction for multiple testing.

Although past studies have found that obese versus lean individuals show greater striatal activation in response to pictures of palatable foods versus control pictures (13), baseline BMI did not significantly correlate with striatal and OFC responsivity to food commercials nor did BOLD response differ between obese and healthy-weight adolescents at baseline (10). The lack of significant correlations between BMI and striatal effects may be the result of the stimuli used in the current study. Previous studies have used stationary food- and nonfood pictures. Our study is the first to investigate neural response to unhealthy food commercials shown in the context of a television show. The food commercial blocks included periods of time during which no food was shown (e.g., food logos and people dining). Thus, food commercials may show a weaker ability to activate striatal and OFC regions than food images in isolation because of the greater heterogeneity of the former. To our knowledge, no studies have compared neural response to food images with neural response to food commercials. Future studies should determine the differences in neural activation between the two types of food stimuli. Alternatively, the small number of obese ($n = 11$) and lean ($n = 10$) individuals in the current sample, which is smaller than the average sample size ($N = 28$) from studies that found hyper-responsivity of these reward regions to food images, may have limited sensitivity to detecting striatal activation differences.

TABLE 3 Exploratory whole-brain analyses of correlations between BOLD activation to food commercials versus nonfood commercials and food commercials versus television show and BMI over 1-year follow-up

Contrast and region	BA	k	Z value	MNI coordinates
<i>Food > nonfood commercials</i>				
Positive correlation with change in BMI				
Caudate		37	3.82	-12, -7, 22
Caudate			3.45	-6, -1, 10
Anterior cerebellum		40	3.61	-3, -49, -14
Anterior cerebellum			3.08	-3, -58, -11
Middle occipital gyrus		52	3.53	-39, -76, 31
Middle temporal gyrus	39		3.35	-45, -61, 22
Cuneus	31	35	3.36	-3, -70, 16
Cuneus			3.01	-6, -85, 22
Insula		27	3.25	-45, -16, 10
Insula			3.17	-51, -22, 10
Superior frontal gyrus	9	24	3.24	-21, 41, 37
Middle temporal gyrus		24	3.07	51, -55, 16
Supplemental motor area		22	3.06	-6, -10, 55
Negative correlation with change in BMI				
Middle occipital gyrus*		186	4.35	27, -76, 31
Superior occipital gyrus	7		3.77	24, -73, 45
Superior parietal lobe			3.44	24, -64, 55
Lingual gyrus*	17	170	3.90	9, -91, -2
Lingual gyrus			3.31	-3, -79, -8
Lingual gyrus	18		3.14	-18, -79, -17
Middle occipital gyrus*		93	3.68	-27, -79, 19
Superior occipital gyrus			3.45	-24, -82, 31
Middle occipital gyrus			3.28	-27, -88, 4
<i>Food commercials > television show</i>				
Positive correlation with change in BMI				
Parahippocampal gyrus*		67	4.45	-30, -43, -5
Amygdala*		47	4.21	33, -7, -23
Thalamus			3.42	9, -28, 10
Temporal pole		23	4.04	45, 11, -26
Middle temporal gyrus			3.30	54, 5, -20
Posterior cerebellum		129	4.03	24, -49, -44
Anterior cerebellum			3.59	15, -55, -29
Posterior cerebellum			3.30	12, -46, -50
Supplemental motor area		36	3.96	-15, -37, 52
Parahippocampal gyrus		34	3.74	30, -43, -8
Parahippocampal gyrus			3.52	36, -28, -14
Anterior cerebellum		35	3.63	-12, -55, -29
Anterior cerebellum			3.58	-3, -58, -11
Middle temporal gyrus*		50	3.49	45, -67, 22
Middle temporal gyrus	39		3.01	42, -61, 16
Middle temporal gyrus		34	3.40	-42, -67, 16
Negative correlation with change in BMI				
Superior parietal lobe	7	33	3.96	30, -70, 49
Posterior cerebellum		39	3.22	-21, -79, -23

Analyses controlled for hunger and baseline BMI.

BA = Brodmann areas; k = cluster size.

*Significant at $P < 0.005$ with a cluster size (k) = 22; $P < 0.05$, whole-brain corrected.

It is important to consider the limitations of this study. First, the small sample size may have resulted in false negative findings regarding the nonsignificant association between follow-up BMI and the OFC, as well as the lack of significant correlations between baseline BMI and striatal and OFC responsivity. Second, 1-year follow-up BMI data were missing for two subjects and for four subjects, we used self-reported height and weight data to calculate 1-year follow-up BMI, which may have resulted in biased estimates of weight gain. However, our results remained significant when excluding these subjects from the analyses and reported BMI was highly correlated with measured BMI ($r = 0.98$), suggesting that the missing and self-reported BMI data had limited impact. Nonetheless, findings should be considered provisional until replicated in a larger sample using objective measures for assessing BMI. Third, data on energy expenditure might have facilitated the interpretation of our results as individual differences in physical activity may contribute to changes in weight over time (40), although self-reports of exercise have low validity (31). Finally, we prioritized external validity using food and nonfood commercials that are more frequently advertised to adolescents; future studies should contrast the effects of exposure to unhealthy versus healthy food commercials, to better isolate the effects of exposure to unhealthy food commercials.

The finding that elevated reward region response to food commercials is associated with greater weight gain is a unique contribution to the literature, as it suggests that there are individual differences in neural vulnerability to food commercials that appear to identify youth at risk for excess weight gain. In combination with established risk factors of weight gain during adolescence, such as sedentary behavior (40) and parental obesity (21,22), elevated reward-related response to commercials may be an important contributor and a potential target for prevention and intervention programs. Future studies should explore how individual differences (e.g., weight status, genetic markers), and environmental factors (e.g., the proximity to fast food restaurants) determine who will develop hyper-responsivity of reward regions to food advertising that increases risk for future weight gain. **O**

Acknowledgments

We thank the Lewis Center for Neuroimaging at the University of Oregon for assistance in data collection.

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