Emotional eating and instructed food-cue processing in adolescents: an ERP study

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Abstract

We examined the P3 (250 to 500 ms) and Late Positive Potential (LPP; 500 to 2000 ms) event-related potentials (ERPs) to food vs. nonfood cues among adolescents reporting on emotional eating (EE) behavior. Eighty-six adolescents 10-17 years old were tested using an instructed food versus nonfood cue viewing task (imagine food taste) during high-density EEG recording. Self-report data showed that EE increased with age in girls, but not in boys. Both P3 and LPP amplitudes were greater for food vs. nonfood cues (food-cue bias). Exploratory analyses revealed that, during the LPP time period, greater EE was associated with a more positive food-cue bias in the fronto-central region. This heightened fronto-central food-cue bias LPP is in line with a more activated prefrontal attention system. The results suggest that adolescents with higher EE may engage more top-down cognitive resources to regulate their automatic emotional response to food cues, and/or they may exhibit greater reward network activation to food cues than do adolescents with lower EE, even in the absence of an emotional mood induction.

1. Introduction

1.1 Emotional Eating

Food is a primary reinforcer for many species, driving goal directed behavior. In modern society, with an abundance of food for many, humans eat not only for nutrition and to satisfy hunger, but also for pleasure and to alleviate negative emotions and reduce stress (Greeno & Wing, 1994; Rutledge & Linden, 1998). This latter function, referred to as *emotional eating* (EE), describes a tendency to eat and overeat when experiencing negative mood states, even in the absence of physiological hunger (Arnow, Kenardy, & Agras, 1995). Various mechanisms have been proposed to contribute to EE. Negative emotions reduce inhibitory control, thus making people more likely to eat unhealthy foods. Eating elevates positive mood. Thereby it becomes a self-reinforcing habit for blunting negative emotions (Hayaki, 2009). Additionally, long-term exposure to stress can cause malfunction of the hypothalamic-pituitary-adrenal (HPA) axis, thus dysregulating food consumption and metabolism (Yau & Potenza, 2013), and leading to long-term health concerns (Tryon, Carter, Decant, & Laugero, 2013).

EE is closely related to several eating disorders and obesity (Pinaquy, Chabrol, Simon, Louvet, & Barbe, 2003). Also, EE is a major contributor to binge eating (Eldredge & Agras, 1996) and is associated with emotional problems such as anxiety and depression (Van Strien, Schippers, & Cox, 1995). Compared to men, women are at higher risk for EE and related weight gain (Epel, Lapidus, McEwen, & Brownell, 2001; Fryer, Waller, & Kroese, 1997; Levine & Marcus, 1997). The pattern of more EE among females emerges by late adolescence (Wardle et al., 1992). However, a majority of EE studies have only examined adult women (Pinaquy et al.,

2003; Van Strien et al., 1995). Therefore, further studies are needed on the development of EE in adolescent women and men.

1.2 ERPs and food-cue studies

Event-related potentials (ERPs), with their high temporal resolution, are a useful tool for detecting the early onset of attentional processing of visual food cues (Wolz, Fagundo, Treasure, & Fernandez-Aranda, 2015). The P3 and Late Positive Potential (LPP) are two commonly examined ERP components. The P3 is a positive ERP component occurring ~300 ms post-stimulus onset and is associated with attention allocation and memory (Polich & Kok, 1995). The LPP is a slow wave ERP which emerges as early as 200 ms and lasts 1000 ms or more and is associated with more extended attention allocation driven by the emotional and motivational relevance of visual images (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Hajcak, MacNamara, & Olvet, 2010; Marmolejo-Ramos et al., 2015).

Recent evidence suggests that food cues can preferentially engage attention over nonfood cues (Castellanos et al., 2009; Doolan, Breslin, Hanna, Murphy, & Gallagher, 2014). Enhanced P3 and LPP amplitudes are associated with visual food cues versus neutral stimuli (Asmaro et al., 2012; Gable & Harmon-Jones, 2010; Nijs, Franken, & Muris, 2008, 2010). Additionally, P3 and LPP amplitudes to food cues are sensitive to the motivational salience of the cue. Greater amplitude enhancements to food cues are observed in hungry versus satisfied states (Stockburger, Schmalzle, Flaisch, Bublatzky, & Schupp, 2009) and to high versus low calorie foods (Meule, Kubler, & Blechert, 2013). LPP amplitude is also sensitive to emotion regulation and cognitive control. LPP amplitudes are reduced when adults are instructed to attend to the less arousing parts of emotional images (Hajcak, Dunning, & Foti, 2009) and when children are instructed to reappraise emotional images as neutral situations (Dennis & Hajcak, 2009).

Similarly, suppressing craving towards food cues decreases LPP amplitude to food cues in adult female restrained eaters (Svaldi et al., 2015).

P3 and LPP amplitudes can reflect more stable individual differences as well. For example, enhanced P3 amplitudes to food cues are correlated with higher external eating in adult women (Nijs, Franken, & Muris, 2009). Similarly, women who are restrained eaters exhibit greater LPP amplitudes to images of food that is unavailable versus available (Blechert, Feige, Hajcak, & Tuschen-Caffier, 2010). Regarding EE specifically, researchers have found associations of EE with LPP amplitudes to food cues (Blechert, Goltsche, Herbert, & Wilhelm, 2014; Meule et al., 2013), but not with P3 amplitudes (Nijs, Muris, Euser, & Franken, 2010). Although emotional eaters are particularly vulnerable under emotional situations, some evidence suggests that emotional eaters show different electrophysiological responses to food cues regardless of their current mood state. For example, in electrophysiological studies that measured EE in adults, high emotional eaters have been shown to have heightened LPP responses to food cues across both neutral and negative mood states (Blechert et al., 2014) and in the absence of any mood induction (Meule et al., 2013). These findings suggest that emotional eaters find food cues to be more salient even in the absence of negative emotional states. It is worth mentioning that in both of these studies all stimuli were food cues and there were no nonfood cues as controls. Blechert et al. (2014) suggested that future studies on EE including both food and nonfood cues would be necessary in order to confirm the food-cue specificity of heightened LPP responses in those high in EE.

1.3 Emotional eating in Adolescents

Adolescence is a particularly important developmental period in which to study EE. Adolescents experience heightened emotionality due to changes in hormone levels and their

social environment (Arnett, 1999; Larson, Moneta, Richards, & Wilson, 2002). They also start to develop more independence of food choice (Bassett, Chapman, & Beagan, 2008). In fact, EE increases with age during adolescence and is also more prominent in girls than in boys (Wardle et al., 1992). Unhealthy eating behaviors emerging in childhood contribute to eating disorders and obesity in adults (Stark, Atkins, Wolff, & Douglas, 1981), and EE is significantly more common among obese children compared to their non-obese counterparts (Braet & Van Strien, 1997). However, only a handful of studies have examined the brain-based correlates of the onset of problematic eating behavior during adolescence. P3 amplitude to food cues was found to be positively correlated with restrictive feeding practices in children (Hill, Wu, Crowley, & Fearon, 2013) and restrained eating scores in obese adolescents (Hofmann, Ardelt-Gattinger, Paulmichl, Weghuber, & Blechert, 2015). Additionally, enlarged P3 and LPP amplitudes to food cues were found in adolescents with anorexia nervosa compared to the control group (Novosel et al., 2014).

Despite the importance of emotional eating for understanding the onset of problematic eating behavior in adolescence, no previous studies have examined how EE relates to P3 or LPP amplitudes to food cues in adolescents. The present study fills this research gap. We expect that electrophysiological responses to food cues will reflect individual differences in EE in adolescents.

1.4 The present study

While EE and neural responses to food cues have been examined in adults, no studies have explored this potential link in adolescence. Thus, employing high-density EEG, we measured the neural responses to food versus non-food cues in relation to EE within a sample of adolescents aged 10 to 17 years. Given existing literature, we examined the modulations of two ERP components, the P3 and LPP, in response to food and non-food images and in association

with adolescent self-reported EE. Behaviorally, we hypothesized that greater EE would be observed as age increases, and that girls would report greater EE than boys. In terms of neural response, we hypothesized that adolescents would show a food bias that is comparable to what has been found in adults, with food cues generating an enhanced P3 and LPP, relative to nonfood cues. Moreover, we predicted that adolescents with higher EE would exhibit a larger food bias than those with lower EE, which is consistent with the heightened food-cue sensitivity in the former group.

2. Method

2.1 Participants

The current study included 86 adolescents (10-17 years old, 38 female, Table 1) who were a subset of participants in a larger study (Crowley et al., 2014; Crowley et al., 2013). The participants included in the paper completed the food-nonfood cue viewing task described in the current report. The remaining participants in the larger study completed a different task not reported here. Families were recruited via mass mailings to New Haven, CT, and surrounding towns within a 20-mile radius of the study research offices. Children were fluent in English and had no evidence of serious mental illness (psychosis, autism, bipolar disorder) assessed via a parental telephone screen. Participants were intellectually in the normal range based on results from the Vocabulary and Similarities subscales of the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999). Seven participants did not have a sufficient number of trials for the EEG task and 1 participant did not complete the post-task Food-Cravings Questionnaire-State. Thus the remaining 78 subjects (34 female) were included in the EEG related report. The mean age of the adolescents was 13.86 years (SD = 2.24, min =10.14, max = 17.89). The ethnic background of the participants was 7.0% African American, 5.8% Hispanic, 75.6% Caucasian,

7.0% Asian, 2.3% Native American, and 2.3% other ethnic background. This study was approved by the Human Investigation Committee of the Yale University School of Medicine.

Table 1 goes here

2.2 Behavioral measures

Anthropometric measures

Participants' weight and height were measured in the laboratory by a trained researcher using Detecto Weigh Beam (Detecto Inc, Missouri, US). Height was measured to the nearest inch and then converted to meter (m). Weight was measured to the nearest pound and then transformed to kilogram (kg). Body Mass Index (BMI) was calculated as kg/m^2.

Food-Cravings Questionnaire-Trait (FCQ-T)

The Food-Cravings Questionnaire-Trait (FCQ-T) (Cepeda-Benito, Gleaves, Williams, & Erath, 2000) is composed of 39 questions assessing food-related traits including emotional eating, positive and negative reinforcement, with a 1-6 Likert scale: Never/Not applicable, Rarely, Sometimes, Often, Usually, Always. The EE subscale score was calculated as the total scores of 4 items: item 20 (I crave foods when I feel bored, angry, or sad.), item 30 (When I'm stressed out, I crave food), item 34 (My emotions often make me want to eat) and item 39 (I crave foods when I'm upset). The Cronbach's alpha of the EE subscale was .88.

Food-Cravings Questionnaire-State (FCQ-S):

The Food-Cravings Questionnaire-State (FCQ-S) measures hunger and food cravings at the current moment (Cepeda-Benito et al., 2000). We implemented the questionnaire on the

computer so that it first asked the subject to enter his/her favorite food, and then used the food entered by the participant to populate the questions in FCQ-S. The subject was asked to provide ratings using a 1 (strongly disagree) to 5 (strongly agree) Likert scale regarding 15 questions about the craving intensity at the current moment. In 8 of the 15 questions, a favorite food was used in the question, example: "Eating ice cream would make things seem just perfect", with ice cream being the favorite food entered by the subject earlier. The hunger subscale score was calculated as the total response of 3 items, item 13 (I'm hungry), item 14 (If I ate right now, my stomach wouldn't feel as empty) and item 15 (I feel weak because of not eating). The Cronbach's alpha for the hunger subscale was .71.

2.3 Instructed food and nonfood cue-viewing task

The EEG experiment consisted of presenting a mixture of food and nonfood images (45 in each category without repetition). Participants were presented with the following food-cue processing instructions: "You are going to see some food and nonfood images. Try to imagine what the food tastes like if it is a food image." Food images were savory snacks, sweets and high fat food. (Example: pizza, burger, ice cream, chicken, pork, pasta). Nonfood images were neutral objects. (Example: boat, backpack, table, house, chair, cup). Figure 1 shows samples of the food and nonfood stimuli. Appendix 1 has a complete list of all the stimuli in both categories. Roughly half of all stimuli were from the IAPS library and the other half of the stimuli were pulled from the internet (due to the small number of food pictures in the IAPs). Each trial began with a white cross on a black screen for a variable interval of 2300 to 3000 ms. Then a stimulus from either the food category or the nonfood category would appear on the screen for 2000 ms. The stimuli were presented as full-screen images on a 19-inch square LCD screen. Subjects sat 24 inches away from the screen. A pseudo-random list was used to present the images so that no

more than three stimuli from the same category were presented consecutively. The total length of the task was about 10 minutes. Stimuli were presented using E-Prime 2.0 (PST, Sharpsburg, PA, USA).

Figure 1 goes here

2.4 Procedure

Participants made two visits to the lab in order to finish all the requirements. In visit 1, parental permission and youth questionnaire assessments were obtained. FCQ-T was completed in this visit. Within 2 weeks, families attended a second visit to conduct the EEG experiment. Participants' heights and weights were measured. After the EEG net application, the participants completed a 7-minute resting EEG task, which was not included in the present study. Participants then completed the food and nonfood picture viewing task described in the current report. All EEG recording for the task was scheduled between 3:30 and 4:30 pm in order to control the daytime and circadian rhythms and hunger level. After the EEG measurement, participants completed the FCQ-S.

2.5 EEG collection and preprocessing

EEG was collected using NetStation 4.4 and a high-impedance amplifier (Series 300 Amplifier) (Electrical Geodesic Inc., Eugene, OR). Hydrocel-128 nets (Electrical Geodesic Inc., Eugene, OR) with saline electrolyte were used to collect data. Data was recorded at 250 Hz sampling rate with 0.1 to 100 Hz frequency band. Data was referenced to Cz while recording and

re-referenced to the averaged reference offline. All impedances were assessed at or under 40 kOhm before recording.

EEG was first passed through a first-order high pass filter of 0.1 Hz and a low pass filter of 30 Hz. Then continuous EEG was segmented into 100 ms pre-stimulus and 2000 ms post-stimulus segments. Eye blinks (identified as eye blink channel amplitudes exceeding 150 μV) and eye movements (identified as eye movement channel amplitudes exceeding 150 μV) were corrected using the Ocular Artifact Removal Tool in NetStation 4.5 (Electrical Geodesic Inc., Eugene, OR). Bad channels (more than 40% of the segments having any data point higher than 200 μV) were marked; trials with more than 10 bad channels were marked bad and excluded from future steps. In the next step, bad channels were replaced by the surrounding channels using spherical spline interpolation. Then all the channels were re-referenced from Cz to the average reference. Baseline correction was conducted on each segment using the pre-stimulus 100 ms duration. At the end, trials of the same condition were averaged. The number of trials for the food condition was M =27.70, SD = 7.46, Range = 16 to 43. The number of trials for the nonfood condition was M = 26.97, SD = 6.93, Range = 15 to 43. NetStation 4.5 (Electrical Geodesic Inc., Eugene, OR) was used for data preprocessing. SPSS 22.0 (IBM Corp, Armonk, NY) was used for statistical analysis.

2.6 Data analysis

Self-reported EE

Since prior research has shown that sex and age modulate EE in adolescents (Wardle et al., 1992), linear regression models were conducted using EE as the dependent variable and sex, age and the sex-by-age interaction as predictors.

ERP Data Analysis

For the primary analyses, mean amplitudes of the P3 (250 to 500 ms) and LPP (500 to 2000 ms) were calculated at two symmetrical clusters in the left and right parietal regions. These clusters were identified based on previous literature (Blechert et al., 2014; Meule et al., 2013; Nijs et al., 2008, 2009; Nijs, Muris, et al., 2010; Stockburger, Renner, Weike, Hamm, & Schupp, 2009; Svaldi et al., 2015) and on visual examination of the ERP waveforms for food and nonfood cues where the difference appeared maximal (Figure 3). Specifically, the left hemisphere parietal cluster consisted of channels 42, 53, 61, 47, 52, 60, 67, 51, 59, 66, 58, 65, and the right hemisphere parietal cluster consisted of channels 93, 86, 78, 98, 92, 85, 77, 97, 91, 84, 96, 90 of an EGI 128-channel Hydrocel EEG net (Figure 4, panel A). A repeated measures ANOVA was conducted for the P3 and LPP separately with condition (food vs. nonfood) x hemisphere (left vs. right) factors. Then, a repeated measures ANCOVA was conducted with EE as a continuous predictor (a covariate of interest) of P3 and LPP amplitudes, and interactions between EE and condition, hemisphere, and condition x hemisphere were tested.

Following these primary analyses, a more exploratory secondary analysis was conducted of the associations between EE and the food-nonfood difference wave. Difference waves for food versus nonfood cues were calculated at each channel, then correlation of EE and the difference wave was calculated at each 100-ms bin window across the whole head. Topographic maps of the correlation coefficients over time were then visually examined for clusters of channels exhibiting persistent correlations with EE. Then analyses were conducted on the identified clusters testing the significance of the EE associations with mean voltages across these channel clusters in the relevant time windows following the ANCOVA modeling procedures described above.

Several other factors and covariates are also worth considering. Sex and age modulate ERPs in adolescents (Crowley et al., 2013; MacNamara et al., 2016; Speed et al., 2015). Hunger modulates attentional allocation towards food (Loeber, Grosshans, Herpertz, Kiefer, & Herpertz, 2013; Mogg, Bradley, Hyare, & Lee, 1998), and has been found to modify food-cue induced ERPs (Stockburger, Schmalzle, et al., 2009). In this report, hunger was positive correlated to EE, $r = .26$, $p < .05$, (Table 2). Body Mass Index (BMI) has been found to not be related with EE (Nguyen-Rodriguez, Chou, Unger, & Spruijt-Metz, 2008; Snoek, Engels, van Strien, & Otten, 2013), and it was not correlated with any ERPs in the current study (Table 3), thus it was not included in the model. We examined the EE-related effects with the presence of sex, age and hunger, by adding them as covariates to the original ANCOVA model. Linear regressions were used for post-hoc tests upon significant EE-related interactions. All the covariates were standardized prior to being entered in the ANCOVA models.

Table 2 goes here

Finally, previous work reported that EE is closely related to the positive and negative reinforcement aspects of eating (Hayaki, 2009). The positive reinforcement subscale in FCQ-T has 5 items that ask about the positive feelings that may result from eating, and the negative reinforcement subscale in the FCQ-T has 3 items that ask about anticipation of relief from negative states as a result of eating (Cepeda-Benito et al., 2000). In the current study, EE was found to be highly correlated with both the positive reinforcement subscale, $r = .72$, $p < .001$, and the negative reinforcement subscale, $r = .59$, $p < .001$. Therefore, in order to examine the

divergent validity of the EE effects, additional ANCOVAs were conducted using FCQ-T positive reinforcement and negative reinforcement subscale scores as predictors in place of EE. Additionally, a model was conducted in which the FCQ-T positive and negative reinforcement subscales were included as predictors in addition to EE in order to establish whether EE is uniquely correlated with P3 and LPP amplitudes after controlling for these closely-related constructs.

3. Results

3.1 Self-report measures of EE

A General Linear Model using EE as the dependent measure, and using sex (male coded 0 and female coded 1), age, and the sex by age interaction as predictors, showed a significant model effect, $F(3, 82) = 3.29$, $p = .025$. There were no significant main effects of sex, $b = -9.27$, $t(82) = -1.75$, $p = .084$, or of age, $b = -0.07$, $t(82) = -0.27$, $p = .789$. However, there was a significant sex by age interaction predicting EE, $b = 0.77$, $t(82) = 2.05$, $p = .044$. To probe this significant interaction, correlations between EE and age were calculated separately for males and females. EE was positively correlated with age in the female sample, $r = .36$, $p = .025$, $n = 38$, transformed to $z = 0.38$, $SE = 0.17$, but not in the male sample, $r = -.04$, $p = .775$, $n = 48$, transformed to $z = -0.04$, $SE = 0.15$ (Figure 2). The difference of the two correlations was significant using a one-tailed t-test, $z = 1.88$, $p = .030$, indicating that the correlation of age and EE was stronger in girls than boys.

Figure 2 goes here

3.2 EEG

3.2.1 Parietal P3 and LPP amplitudes to food vs. nonfood cues

To test for the main effects of food vs. nonfood cues, repeated-measures ANOVAs were conducted for the parietal P3 (250 – 500 ms) and LPP (500 – 2000 ms) separately with condition (food vs. nonfood) x hemisphere (left parietal vs. right parietal) factors. Significant condition effects were found for both P3 and LPP, $F(1, 77) = 22.12$, $p < .001$, $\eta_p^2 = .22$, and $F(1, 77) = 21.01$, $p < .001$, $\eta_p^2 = .21$, respectively, reflecting more positive P3 and LPP amplitudes for the food cue than the nonfood cue, P3, $M_{food} = 7.13$ μV, SD = 3.75 μV, $M_{nonfood} = 6.19$ μV, SD = 3.40 μV, $t(77) = 4.70$, $p < .001$, LPP, $M_{food} = 1.40$ μV, SD = 2.16 μV, $M_{nonfood} = 0.49$ μV, SD = 1.88 μV, $t(77) = 4.58$, $p < .001$.

Figure 3 goes here

There was also a significant main effect of hemisphere for the P3, $F(1, 77) = 20.57$, $p < .001$, $\eta_p^2 = .21$, indicating the left hemisphere had a less positive P3 than the right hemisphere, $M_{left} = 5.87$ μV, SD = 3.68 μV, $M_{right} = 7.46$ μV, SD = 3.92 μV, $t(77) = -4.54$, $p < .001$. The hemisphere effect was not significant for the LPP, $F(1, 77) = 0.03$, $p = .859$, $\eta_p^2 < .01$. There was no significant condition x hemisphere interaction for the P3, $F(1, 77) = 0.11$, $p = .743$, $\eta_p^2 < .01$, or for the LPP, $F(1, 77) = 1.52$, $p = .222$, $\eta_p^2 = .02$, indicating that the main effect of condition did not differ across hemispheres.

3.2.2 Association of EE with parietal P3 and LPP amplitudes

After adding EE to the models, we found no significant EE-related effects predicting parietal P3 or LPP amplitudes. Specifically, for the P3, the EE-related effects were: EE main effect, $F(1,76) = 1.09$, $p = .301$, $\eta_p^2 = .01$, EE x hemisphere, $F(1,76) = 0.03$, $p = .858$, $\eta_p^2 < .01$, EE x condition, $F(1,76) = 0.41$, $p = .521$, $\eta_p^2 = .01$, and EE x hemisphere x condition, $F(1,76) = 0.37$, $p = .547$, $\eta_p^2 = .01$. For the LPP, the EE-related effects were: EE main effect, $F(1,75) = 3.03$, $p = .086$, $\eta_p^2 = .04$, EE x hemisphere, $F(1,76) = 0.26$, $p = .609$, $\eta_p^2 < .01$, EE x condition, $F(1,76) = 0.29$, $p = .595$, $\eta_p^2 < .01$, and EE x hemisphere x condition, $F(1,76) = 0.27$, $p = .602$, $\eta_p^2 = .01$.

Figure 4 goes here

3.2.3 Exploratory analysis of EE correlations with the food vs. nonfood difference wave

Since individual differences are not always observed at the same locations where condition effects are observed (Asmaro et al., 2012; Blechert et al., 2010; Blechert et al., 2014), topographic plots of the correlation between EE and the food-nonfood difference wave at each channel were examined to take a more exploratory approach to identifying clusters of channels where EE may be associated with the response to food cues (Figure 5).

Figure 5 goes here

Visual examination of these topographic correlation maps revealed broad clusters of

channels in both fronto-central and occipital regions where individual differences in EE were correlated with the food-nonfood difference wave beginning around 500 ms and strengthening through 2000 ms. EE was positively correlated with the difference wave in the fronto-central region and negatively correlated with the difference wave in the occipital region. Since the ERP waveforms showed inverted voltage fluctuations between fronto-central and occipital regions (Figure 3), we reasoned that the inverse correlation of the food-nonfood difference wave with EE across these two regions may reflect opposite projections of a single dipole source. Therefore we conducted source localization using GeoSource 2.0 (Electrical Geodesic Inc., Eugene, OR) on the responses to food cues, following procedures described in a previous paper (Crowley et al., 2013). The sLORETA representations of the responses to food cues indicated that the maximum activation started in the occipital lobe, with a maximum around 350 ms, and transitioned to the frontal lobe starting around 500 ms and strengthening throughout the LPP time window (Figure 6). The maximum activation during the LPP time window was localized to Brodmann's area 11, which is part of the orbitofrontal cortex. Therefore, we decided to focus our further analyses of the LPP on the fronto-central region only. Guided by the correlation maps, we selected two symmetrical clusters of channels in the fronto-central region for statistical analysis. The left fronto-central cluster consisted of channels 20, 13, 28, 29, 30, 31, 34, 35, 36, 37, 39, and 40, and the right fronto-central cluster consisted of channels 118, 112, 117, 111, 105, 80, 116, 110, 104, 87, 115, and 109 (Figure 7 panel A). We then conducted a repeated-measures ANCOVA on the average voltage in these fronto-central channel clusters in the LPP time window (500 – 2000 ms) with condition (food vs. nonfood) x hemisphere (left frontal vs. right frontal) factors and with EE as the covariate of interest.

Figure 6 goes here

As expected, the ANCOVA revealed a significant condition x EE interaction, $F(1, 76) = 7.83$, $p = .007$, $\eta_p^2 = .09$. Since the EE x condition x hemisphere interaction was not significant, $F = 0.24$, $p = .627$, the LPP amplitudes from the left and right fronto-central regions were averaged for post hoc analyses. Pearson's correlation indicated that EE positively correlated with the fronto-central LPP difference wave (food minus nonfood cue), $r = .31$, $p = .007$ (Figure 7, panel D). The ANCOVA revealed no significant main effect of EE, $F(1, 76) = 0.92$, $p = .342$, partial $\eta^2 = .01$, or other interactions, Fs <= 1.11, ps >= .295.

Figure 7 goes here

When sex, age, and hunger were added to the model as covariates, the condition by EE interaction predicting fronto-central LPP amplitudes remained significant, $F(1, 73) = 6.22$, $p = .015$, partial $\eta^2 = .08$. Linear regressions using the LPP food-nonfood difference wave as the dependent variable with EE, age, sex, and hunger as the predictors indicated that the positive association of EE with the LPP difference wave in fronto-central regions also remained significant after controlling for these covariates, $b = 0.45$, $t(73) = 2.50$, $p = .015$.

To test for divergent validity of the EE effects on LPP amplitude in fronto-central regions, the repeated measures ANCOVA was conducted separately with the positive and

negative reinforcement subscale scores from the FCQ-T as covariates of interest in place of the EE score. The positive reinforcement effect was significant at the trend level, condition x positive reinforcement, $F(1, 76) = 3.40$, $p = .070$, $\eta_p^2 = .04$. The negative reinforcement effect was not significant, condition x negative reinforcement, $F(1, 76) = 2.16$, $p = .145$, $\eta_p^2 = .03$. When both the positive and negative reinforcement scales were included in the ANCOVA model with EE, the EE effect remained significant, condition x EE, $F(1, 76) = 4.11$, $p = .046$, $\eta_p^2 = .05$.

3.3 Outlier sensitivity tests

One female participant had an EE score that was greater than 3 standard deviations from the sample mean (EE = 24, Z-score = 3.90). To test for the sensitivity of the results to this outlier value, we re-ran all analyses that had yielded significant EE findings excluding this participant's data.

3.3.1 The association of EE with age in girls after outlier removal

We conducted the same regression model as in Section 3.1 (EE regressed on sex and age) after removing the outlier, and found comparable results. The sex by age interaction predicting EE was significant, $b = 0.71$, $t(81) = 2.04$, $p = .045$. The correlation of age with EE in girls was significant, $r = .40$, $p = .015$, $n = 37$, transformed to $z = 0.42$, $SE = 0.17$. The correlation of age with EE in boys stayed the same, and the difference between the two correlations remained significant, $z = 2.02$, $p = .022$.

3.3.2 The EE effect on the fronto-central LPP after outlier removal

We conducted the same ANCOVAs as in Section 3.2.3 (fronto-central LPP predicted by condition, hemisphere, and EE) after removing the outlier and found comparable results. With only EE as the covariate, the ANCOVA revealed a significant condition x EE interaction predicting fronto-central LPP amplitudes, $F(1, 75) = 4.78$, $p = .032$, $\eta_p^2 = .06$. Pearson's

correlation indicated that EE positively correlated with the LPP food-nonfood difference wave, r = .25, p = .032.

After adding sex, age and hunger to the model, the condition by EE interaction predicting fronto-central LPP amplitudes remained significant, $F(1, 72) = 4.14$, $p = .046$, partial $\eta^2 = .05$. Again, the post hoc linear regression showed a significant positive association of EE with the LPP difference wave after controlling for these covariates, $b = 0.40$, $t(72) = 2.04$, $p = .046$.

When either positive or negative reinforcement was used in place of EE with this outlier removed, neither of the ANCOVAs produced any significant results related to the covariate of interest. The condition x positive reinforcement interaction term was, $F(1, 75) = 1.86$, $p = .177$, $\eta_p^2 = .02$ and the condition x negative reinforcement interaction term was, $F(1, 75) = 1.20$, $p = .278$, $\eta_p^2 = .02$.

4. Discussion

This study examined whether adolescents with a spectrum of EE traits would have different sensitivity to food cues. We assessed EE via self-report and measured EEG while participants viewed food and nonfood cues, with the instruction to imagine what the pictured foods taste like. Behaviorally, we found a significant sex by age interaction in predicting EE. Among girls only age was positively correlated with the EE score, and there was no such correlation in boys. This finding is consistent with a previous study showing older girls reporting more EE issues (Wardle et al., 1992).

In terms of electrophysiological results, we found enhanced P3 (250 – 500 ms) and LPP (500 – 2000 ms) amplitudes for food-cue stimuli versus the nonfood-cue stimuli in the parietal regions, suggesting that food cues were preferentially processed over nonfood cues (food-cue bias) during both the P3 and LPP time periods. Previous studies in adults have shown similar

food-cue biases for the P3 and LPP (Meule et al., 2013; Nijs et al., 2008; Nijs, Muris, et al., 2010). In adolescents, the only relevant study we found was that of Hofmann and colleagues (Hofmann et al., 2015), who found an enhanced P3 for food versus object cues, with participants being a combination of healthy controls and obese counterparts. The current study confirmed that, similar to adults, adolescents exhibit enhanced P3 and LPP amplitudes towards food cues versus nonfood cues, reflecting preferential processing of food cues. However, this effect must be interpreted in light of our paradigm instructions. Participants were instructed to imagine the taste of foods pictured in the food cues, whereas no special instructions were provided for the nonfood cues. The enhanced P3 and LPP amplitudes to food cues in this study may thus be due at least in part to the instruction to engage in more elaborate cognitive processing of the food cues.

To our knowledge, the current report is the first to examine the effects of EE on electrophysiological responses to food cues in adolescents. We found no effects of EE on the food cue bias (food-nonfood difference) in the P3 or LPP amplitude when measured at parietal regions where the main effect of the food vs. nonfood cues appeared maximal. However, a subsequent exploratory analysis revealed that higher EE was associated with a more positive amplitude to food vs. nonfood cues specifically in a fronto-central region in the LPP time period. This finding is in line with previous research, in which EE was found to correlate with the LPP to food cues (Meule et al., 2013). The LPP reflects sustained perceptual and cognitive-elaborative processing of motivationally relevant stimuli (Hajcak et al., 2010). Thus, the present results suggest that individual differences in EE are associated with food cue processing during cognitive-elaborative stages of information-processing. Specifically, we found that adolescents with *higher* EE (HEE) exhibited higher food-cue bias (more positive LPP amplitudes to food vs.

nonfood cues) in fronto-central regions. Since participants were instructed to imagine what the pictured foods tasted like, these differences in the food-cue bias may reflect individual differences in both the intrinsic salience of food cues and in the neural resources recruited during in-depth cognitive-elaborative processing of the food cues.

Previous food-related studies have found associations of frontal LPPs with emotionally salient food-cue stimuli. For example, more positive frontal LPPs have been observed for appetitive food cues versus neutral stimuli (Gable & Harmon-Jones, 2010) and for unavailable food cues compared to available food cues in restrained eaters (Blechert et al., 2010). In the present study, the maximum source of the LPP to food cues was localized to the frontal lobe, in particular Brodmann's Area 11, which is part of the orbitofrontal cortex. Our finding is consistent with a previous study in which source localization of the LPP implicated a prefrontal-occipitoparietal attention network that underlies sustained attention during the late stages of stimulus processing (Moratti, Saugar, & Strange, 2011). Sustained attention facilitates affective processing by enhancing activity in visual processing areas and prefrontal cortex (Wessing, Rehbein, Postert, Furniss, & Junghofer, 2013). Compared to the parietal regions, LPPs from the frontal region have been found to relate to top-down cognitive control of emotion (Moratti et al., 2011). For example, cognitive up- and down-regulation of emotion increases magnetoencephalographic activation to emotional pictures in the LPP time window in the dorsal prefrontal cortex (Wessing et al., 2015). Thus we speculate that in the current study, HEE participants may have more strongly engaged the prefrontal attention network to regulate their automatic emotional responses to the food images.

The prefrontal attention network is also involved in reward processing. fMRI studies find that food cues activate brain reward regions including prefrontal cortex, orbitofrontal cortex,

anterior cingulate, insula and amygdala (Garcia-Garcia et al., 2013; van der Laan, de Ridder, Viergever, & Smeets, 2011). Implicating prefrontal activation in reward-related processing, greater prefrontal circuit activation was observed for high-calorie food cues versus low-calorie ones (Killgore et al., 2003). Enhanced prefrontal cortex activations to food images were found in individuals with eating disorders compared to controls (Uher et al., 2004). With regard to EE, greater EE has been associated with greater activation in the left dorsolateral prefrontal cortex in response to high versus low calorie food cues in adults (Wood et al., 2016) and with greater insula activation to food cues in both healthy controls and obese participants (van Bloemendaal et al., 2015). Additionally, only in high emotional eaters, reward regions including anterior cingulate cortex were more activated for food cues in an induced negative mood (Bohon, Stice, & Spoor, 2009). Thus another plausible explanation of the heightened activation over the fronto-central cluster would be heightened reward sensitivity towards food cues in HEE through the prefrontal attention network.

To our knowledge, the only previous research linking EE with brain activities in adolescents was conducted by Bohon (2014). In this study, EE was found to negatively correlate with activations in palatable food regions and reward circuitry upon receipt of milkshake. The reduced reward circuitry activation in relation to greater EE seems to contradict the results from the current study. However, Bohon and colleagues used real food consumption as the stimuli whereas the current paper used visual food cues. It is possible that high EE in adolescents is related to enhanced sensitivity to reward cues, which activate reward anticipation processes, in combination with reduced sensitivity to actual reward consumption. For example, obese adolescents have been shown to exhibit reduced activation in reward circuitry during food consumption but not during food anticipation (Stice, Spoor, Bohon, Veldhuizen, & Small, 2008).

With regard to other subscales of the FCQ-T, the present study showed that neither the positive nor the negative reinforcement subscales significantly contributed to the heightened LPP food bias. Importantly, the association between EE and the heightened LPP food bias remained significant when both the positive and negative reinforcement subscales were included in the model, suggesting that EE contributes uniquely to the LPP food bias, even when controlling for the highly correlated tendency to find food positively and negatively reinforcing.

Several limitations of the current study and future directions should be considered. First, participants in the current study were healthy adolescents, thus there were not many extreme emotional eaters in our sample. Pre-selecting extreme groups of low and high emotional-eating participants would provide more power to examine potential interactions with age and sex (van Strien, Herman, Anschutz, Engels, & de Weerth, 2012). Secondly, the current study was designed to understand the neural correlates of emotional eating in the absence of induced emotional states. Future work could include a scenario to evoke emotion and stress, and thus to study the emotional eating effect in a more emotionally challenging context. Third, participants' dietary preferences (e.g., vegetarian or not) were not collected. Vegetarians may show differential neural responses to meat (Stockburger, Renner, et al., 2009) which could contribute to unexplained variance in the results for EE. Fourth, we did not provide instructions regarding eating prior to the visit, thus participants could have different hunger levels during the experiment. We measured participants' hunger levels at the end of the EEG experiment and tested post-task hunger as a covariate in the analysis model. Using this approach, we accounted for the variance of the brain signal differences due to different hunger levels at the end of the task. However, a mean hunger level (average of pre and post task) could be a better covariate. Fifth, subjective ratings of the stimulus images were not measured, which would otherwise add

more insights to the brain differences for the current report. Sixth, we did not use one of the published standardized food image stimulus sets. Thus the findings should be replicated using standardized food images that are controlled for contrast and recognizability. Lastly, we gave explicit instructions for processing food-cue stimuli, instructing participants to imagine the taste of the food images. Thus the food versus nonfood ERP condition effect should be considered as reflecting individual differences in attention and cognitive control in the context of imagining the taste of food images. Future research is needed to illuminate whether a similar pattern of findings for EE emerges when food cues are presented without the explicit instruction to imagine the taste of the pictured foods.

In summary, the current study was the first to examine emotional eating in relation to EEG correlates of visual food cue processing in adolescents. We first confirmed that emotional eating positively correlates with age but only in girls. Secondly, we confirmed that posterior P3s and LPPs exhibited food-cue biases in adolescents consistent with studies in adults. Thirdly, we found that EE contributed significantly to heightened food-cue biases in LPP amplitudes in the fronto-central region. This heightened fronto-central LPP may reflect greater cognitive regulation of the emotional response to food cues and/or heightened reward network activation to food cues among high emotional eaters.

Tables and Figures

Table 1. Mean and SD of age, emotional eating (EE), body mass index (BMI), hunger and age separated by sex groups

	age	EE	BMI	hunger
Male, n = 48	13.71 (2.21)	7.56 (3.57)	22.36 (5.99)	9.04 (2.06)
Female, n = 38	14.06 (2.31)	9.11 (4.45)	22.39 (4.30)	9.34 (2.70)
Total, n = 86	13.86 (2.24)	8.24 (4.04)	22.37 (5.28)	9.18 (2.56)

Table 2. Correlations (Pearson's r values) between sex (male and female coded as 0 and 1 respectively), age, emotional eating (EE), hunger, and body mass index (BMI).(for .05 significance and ** for .01 significance.)*

	sex	EE	hunger	BMI
age	.061	.079	.003	.406**
sex		.153	.048	.028
EE			.261*	-.097
hunger				-.117

Table 3. Correlations (Pearson's r values) of P3 and LPP amplitudes in parietal and fronto-central regions with age, sex (male and female coded as 0 and 1 respectively), emotional eating (EE), hunger, and body mass index (BMI) (for .05 significance and ** for .01 significance).*

	age	sex	EE	hunger	BMI
parietal P3 food	-.38**	-.38**	-.13	.24*	-.09
parietal P3 nonfood	-.25*	-.42**	-.10	.26*	-.07
parietal P3 difference	-.35**	.003	-.07	.02	-.06
parietal LPP food	-.31**	-.27*	-.19	.07	.06
parietal LPP nonfood	-.12	-.29*	-.16	.07	.10
parietal LPP difference	-.26*	-.02	-.06	.01	-.18
fronto-central LPP food	.29*	.10	.25*	.08	.11
fronto-central LPP nonfood	.15	.05	-.09	-.04	.09
fronto-central LPP difference	.14	.05	.31**	.11	.03

Figure 1:
Figure 1 Caption: Examples of food stimuli (left panel) and nonfood stimuli (right panel).

Figure 2.

Figure 2 Caption:

Scatter plots of emotional eating and age in boys and girls. Emotional eating is not correlated with age in boys (left panel), but positively correlates with age in girls (right panel).

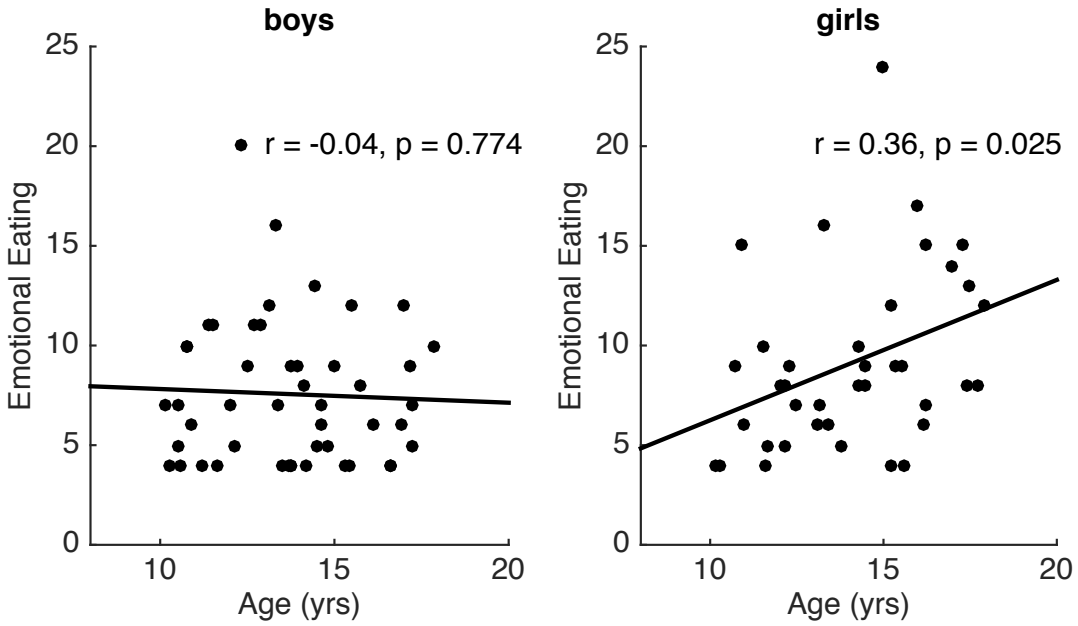


Figure 3
Figure 3 Caption:
ERP waveforms at eight representative locations: F3, F4, C3, C4, P3, P4, O1, O2 with two time windows of interest P3 (250 to 500 ms) and LPP (500 to 2000 ms).

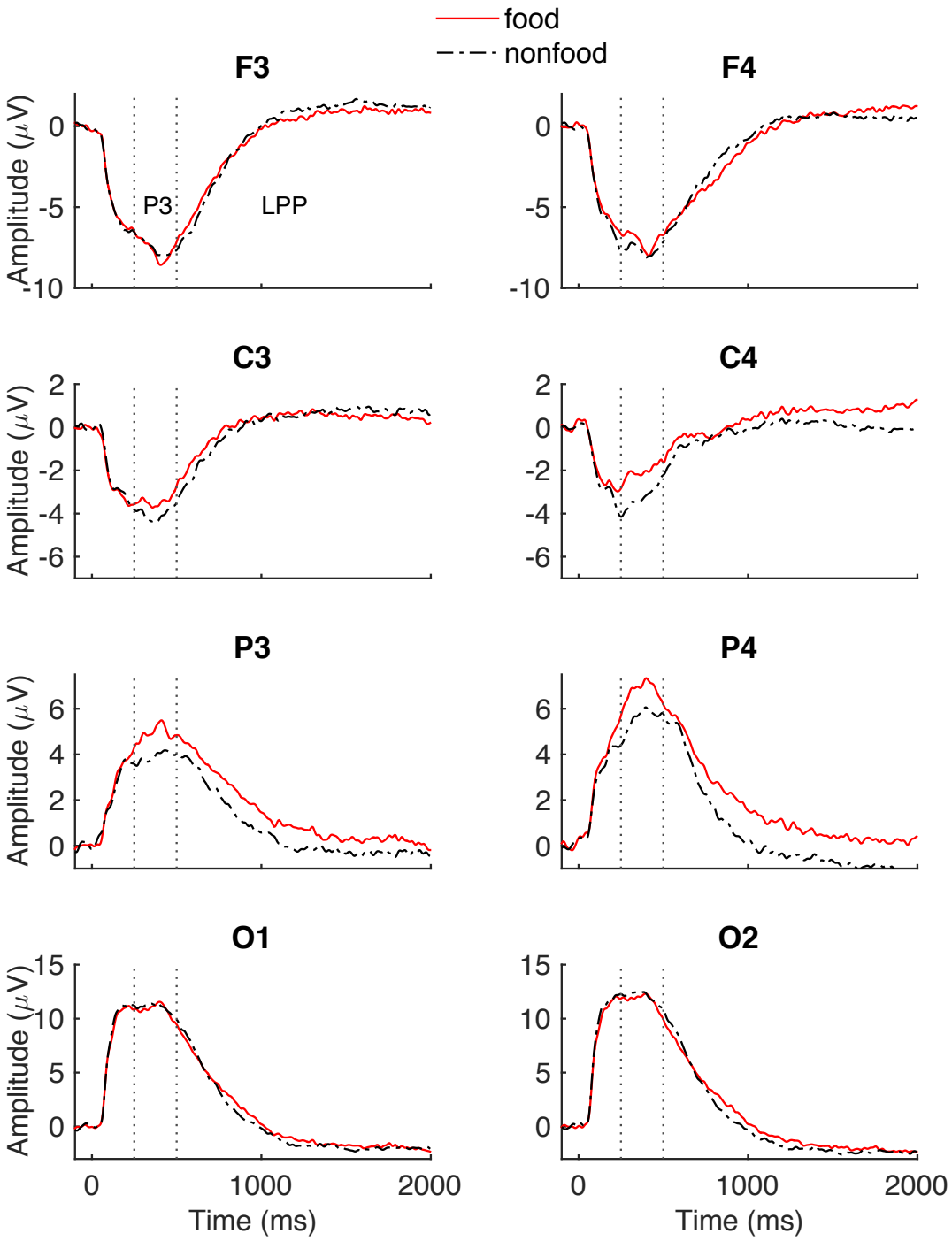


Figure 4.
Figure 4 Caption:
This figure illustrates the waveforms of P3 (250 to 500 ms) and LPP (500 to 2000 ms) at the parietal region (averaged across hemispheres). For illustration purpose only, the sample was split into high emotional eating (HEE, emotional eating score > median score 7) and low emotional eating (LEE, emotional eating score <= median score 7). Both HEE and LEE exhibited a more enhanced P3 and LPP for food cues than nonfood cues, but the effect did not interact with EE.

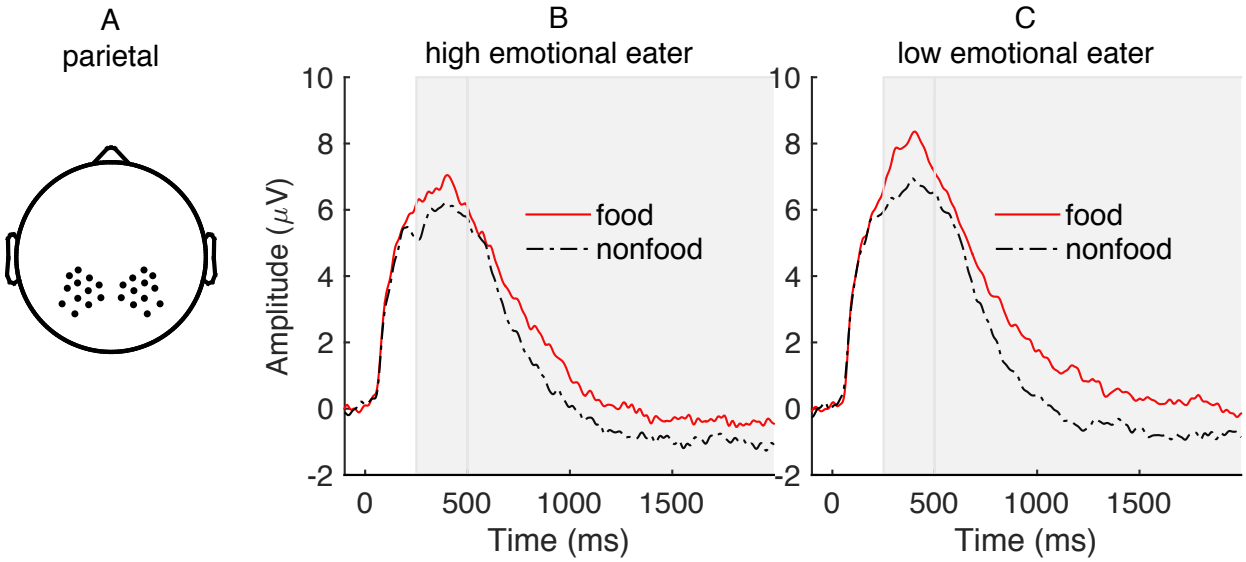


Figure 5

Figure 5 Caption:

This figure illustrates the dynamic changes of the correlation coefficient (Pearson's r) of emotional eating with the food versus nonfood cue difference wave. Starting around 500 ms and strengthening through 2000 ms, a positive correlation emerged in the fronto-central region and a corresponding negative correlation emerged in the occipital region.

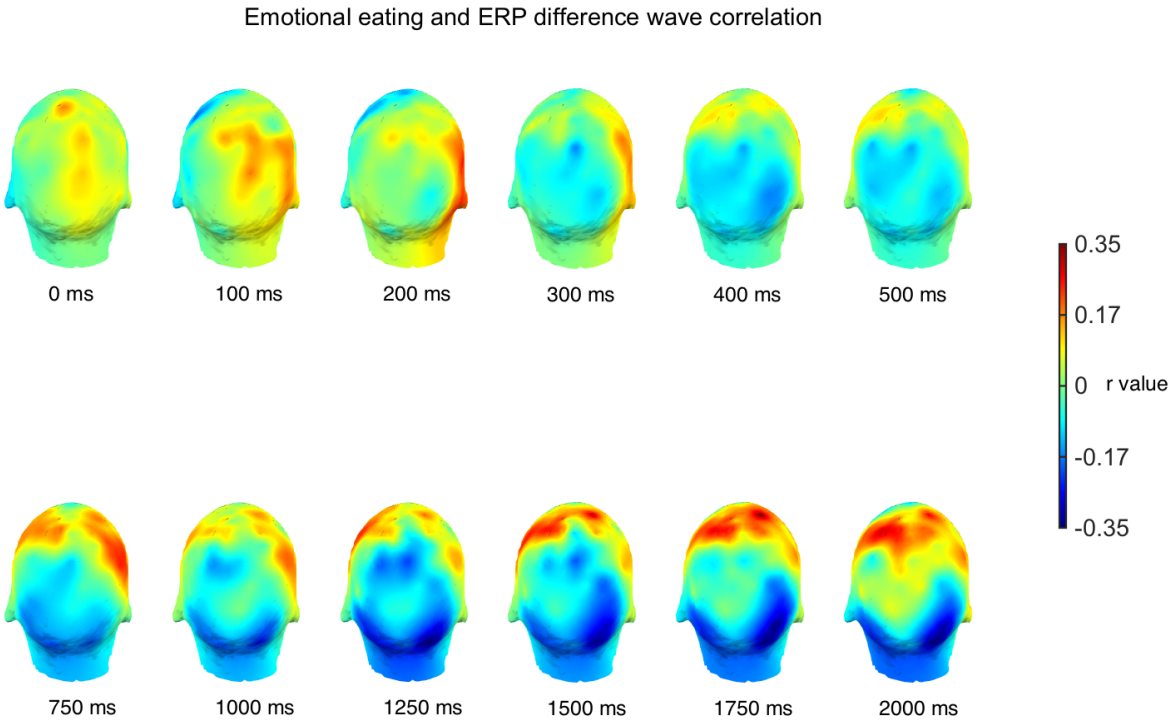


Figure 6

Figure 6 Caption:

This figure illustrates the dynamic location of the maximum source of the ERPs in response to the food cues. The maximum source was located in the occipital lobe during early processing (P3, 200 to 500 ms) and it switched to the frontal lobe during later processing (LPP, 500 to 2000 ms).

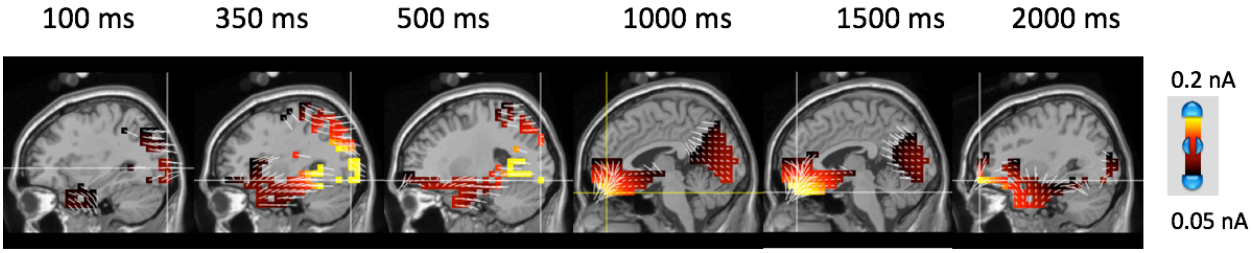
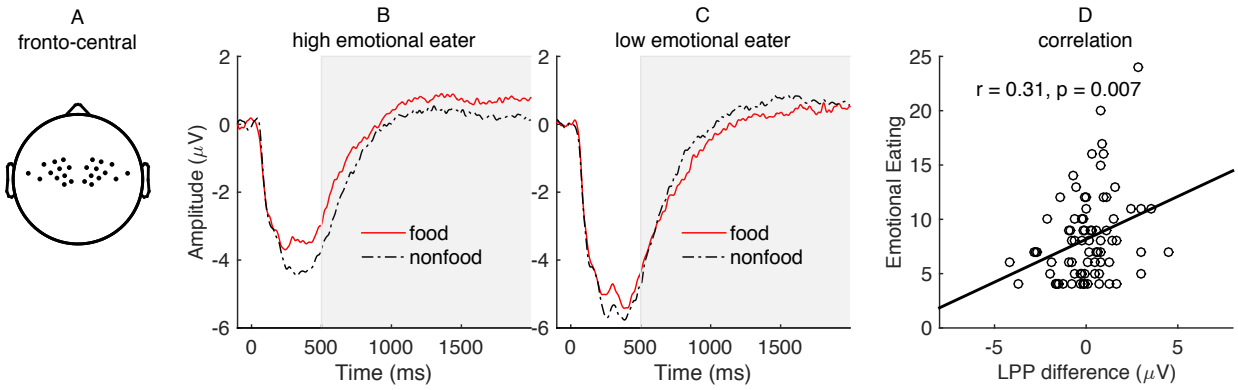


Figure 7

Figure 7 Caption:

This figure illustrates the waveforms of the LPP (500 to 2000 ms) at the fronto-central region (averaged across hemispheres). For illustration purpose only, the sample was split into high emotional eating (HEE, emotional eating score > 7) and low emotional eating (LEE, emotional eating score <=7). HEE individuals exhibited a more positive food-nonfood difference in fronto-central LPP amplitude than did LEE individuals.



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Appendix 1

List of stimulus images from the IAPs library

- food from IAPs (n=24/45)
  7200 (brownie), 7220 (pastry), 7230 (fruit pie), 7260 (ice cream), 7270 (ice cream), 7282 (ice cream), 7283 (fruit), 7284 (fruit), 7286 (bread and cheese), 7289 (meat), 7291 (chicken), 7330 (ice cream), 7340 (ice cream), 7350 (pizza), 7351 (pizza), 7402 (pastry), 7430 (cake), 7450 (burger), 7470 (pancake), 7475 (shrimp), 7480 (pasta), 7481 (shrimp), 7482 (meat), 7484 (fish)
- food stimuli not from IAPs (n=21/45):
  beef dishes n=3, bread n = 1, cake n = 1, chichen dishes n = 2, fries n = 1, fruit n = 1, hotdog n = 1, ice cream n = 2, pasta n = 2, pizza n = 1, pork dishes n = 2, potato dishes n = 1, strawberry pie n = 1, sushi n = 1, tacos n = 1
- nonFood from IAPs (n = 18/45)
  5390 (boat), 5740 (leave), 7000 (roller), 7009 (cup), 7010 (basket), 7025 (chair), 7030 (iron), 7040 (dust pan), 7052 (clippers), 7080 (fork), 7090 (book), 7100 (fire hydrant), 7130 (truck), 7140 (bus), 7150 (umbrella), 7175 (lamp), 7211 (clock), 7950 (box of tissue)
- nonfood stimuli not from IAPs (n = 27/45)
  backpacks n = 3, bed n = 3, bike n = 1, boat n = 2, building n = 1, chair n = 1, coffee table n = 2, couch n = 1, desk n =3, door n = 1, house n = 1, mailbox n = 1, playground n = 1, racket n = 1, schoolbus n = 1, street n = 1, tree n = 1, washer n = 1, window n = 1.