**ORIGINAL PAPER** 



# Neural Correlates of Attentional Bias to Food Stimuli in Obese Adolescents

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#### Abstract

Adolescent obesity is an increasingly prevalent problem in several societies. Researchers have begun to focus on neurocognitive processes that may help explain how unhealthy food habits form and are maintained. The present study compared attentional bias to food stimuli in a sample of obese (n = 22) and Normal-weight (n = 18) adolescents utilizing an Attention Blink (AB) paradigm while electroencephalography (EEG) was recorded. We found lower accuracy and Event-Related Potential (ERP) P3 amplitudes during the presentation of food stimuli in AB trials for obese adolescents. These findings suggest an impaired ability of their brains to flexibly relocate attentional resources in the face of food stimuli. The results were corroborated by lower P3s also being associated with higher body mass index (BMI) values and poorer self-reported self-efficacy in controlling food intake. The study is among the few examining neural correlates of attentional control in obese adolescents and suggests automatic attentional bias to food is an important aspect to consider in tackling the obesity crisis.

Keywords Obesity · Adolescence · Attentional blink · ERP

# Introduction

Alarming increases in the rate of adolescents with obesity the past few decades in the United States (Ogden et al. 2014; Wang and Beydoun 2007) have been linked to a host of physical and mental health problems including, but not limited to: cardiovascular disease, pulmonary and related sleep difficulties, impaired glucose homeostasis, and selfesteem and social problems (Griffiths et al. 2010; Must and Strauss 1999; Valerio et al. 2006). The problem is pervasive as obese adolescents are at higher risk to become obese as

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<sup>1</sup> Department of Educational Psychology, Texas A&M University, 718B Harrington Tower, TAMU, College Station, TX 77843-4225, USA adults, greatly increasing their future health risks and creating a large burden for demands on health services (Farhat et al. 2010; Fonseca et al. 2009; Hedley et al. 2004).

A better understanding of the complex underlying mechanisms of adolescent obesity is critical, considering the possibility of early prevention and intervention efforts. Next, to the investigation of metabolic correlates of childhood and adolescent obesity, there is a growing interest in psychological and cognitive neuroscience factors that can help explain how unhealthy food habits are formed and maintained (Chen et al. 2018; Thamotharan et al. 2013). In the present paper, we examined the role of attention, and its neural correlates, in adolescent obesity. We will first briefly review the relevant literature on attention and its neural mechanisms after which we will link this to adolescent obesity.

As human beings, our minds are limited in the amount of information that can be processed consciously in a short span of time. We ignore the vast majority of the information that reaches our senses and appear to do so quite swiftly and automatically. Attention is the process that helps select and process relevant information that can access conscious working memory representations as well as discount irrelevant information. In the context of obesity, once food stimuli are captured by attention they may result in approach behavior which can maintain and exacerbate their condition (e.g., Castellanos et al. 2009; Werthmann et al. 2011; Schag et al. 2013). The cognitive science literature regarding food bias also reported avoidance tendencies, possibly because these food stimuli are being experienced as threatening due to potential weight gain (See, Nijs et al. 2010; and Field et al. 2016, for an excellent review).

A phenomenon that illustrates the limitations of our attention to process information simultaneously is the Attentional Blink (AB). In a classic visual AB-task, a subject is shown a rapid serial visual presentation (RSVP) of stimuli at a fast rate. The subject is then instructed to identify two specified targets, referred to as T1 and T2 (e.g., two letters among numbers), which are part of the stream of stimuli. Subjects often fail to report T2 when it is presented within roughly 200–500 ms after the correct identification of T1, whereas this is usually not the case when the interval is longer than 500 ms (Raymond et al. 1992; Chun and Potter 1995; and see, Martens and Wyble 2010, for review).

The AB-task is currently a well-studied paradigm. Experiments have shown that the length of the blink (e.g., the period in which subjects are unable to report T2 after correctly identifying T1) is subject to individual differences. A stronger attentional focus on T1 has been linked to a larger AB. In other words, T2 accuracy can reflect the amount of attentional resources allocated to T1 related to the relevance that T1 has to the subject (Dale and Arnell 2010; Olivers and Nieuwenhuis 2005; Shapiro et al. 2006). More precisely, the attentional blink appears to be affected by the level of arousal or relevance a stimulus has to the individual regardless of valence (e.g., whether the stimulus is positive or negative) (Anderson 2005; Smith et al. 2006).

There are a number of theories attempting to explain the underlying mechanisms of the AB effect, including the inhibition model and the two-stage model. The inhibition model proposes that the AB is the result of the suppressive mechanism that reduces the interference from post-target stimuli to reduce target and distractor confusion, whereas the two-stage model explains the AB is caused by capacity limitations of the second stage of processing. For a more in depth review of these, and other theories, we refer readers to reviews by Shapiro et al. (1997), McHugo et al. (2013), and Dux and Marois (2009).

Researchers have also examined the AB-task using electroencephalography (EEG). EEG, able to capture rapid shifts in bioelectric fields generated by cortical activation, is particularly well-suited to measure the temporal dynamics of attentional processes with millisecond precision. The amplitudes of Event-Related Potentials (ERPs: averaged EEG activation to time-locked events) have been used to measure neural correlates of the AB.

The N2 ERP component, for example, is a negative deflection which, depending on the task and subjects, typically occurs between 200 and 400 ms on fronto-central sites.

In the context of AB tasks, the N2, measured locked to T2 (when the blink effect occurs), has been interpreted as an index of attentional conflict processing, i.e., the degree to which attentional resources allocated to T1 interfere with T2 (Denke et al. 2018). The P3, a positive deflection typically occurring between 400 and 800 ms on centro-parietal sites, has been studied more extensively than the N2 in AB-tasks. This component, also locked to T2 (e.g., see, Sessa et al. 2007; Vogel et al. 1998), has been associated with the process of item updating and consolidating in working memory (Martens et al. 2006; Vogel et al. 1998).

Few studies have examined eating disorder pathology using the AB-task and even fewer have done so using neural measures. In line with the incentive-sensitization model of addiction (Robinson and Berridge 2001), it has been proposed that individuals with food-related disorders may have a strong attentional bias towards food stimuli which may lead to a vicious cycle in which attentional bias and craving to food reinforce each other (Castellanos et al. 2009; Franken 2003). In the context of the AB task, we can ask whether obese individuals, when compared to their non-obese peers, may exhibit a stronger AB when T1 consists of a food stimulus compared to a more neutral target.

Indirect evidence supporting this notion comes from studies examining the effects of people's motivation to eat on attentional bias to food stimuli using the AB-task. One study examined 30 healthy undergraduate students under conditions having been food-deprived or not. The findings showed that food stimuli decreased T2 detection (e.g., a larger AB) when participants were hungry (Piech et al. 2010).

Studies have been conducted on individuals with emotional eating disorders. Schmitz et al. (2015) used the AB task to assess attentional bias between a group (n=25) of overweight females with Binge Eating Disorder (BED) and a group of overweight women (n=30) without BED, and showed that attentional engagement to food stimuli was more pronounced in the BED group. An EEG study by Denke et al. (2018) examined whether the N2, an index of conflict processing, moderated the relationship between anxiety and emotional eating behavior in undergraduate students using an AB task. The results confirmed their hypothesis and showed that highly anxious individuals who also had increased N2 magnitudes (interpreted as ineffective processing) were more likely to show high levels of emotional eating.

Though the prior review supports the notion that the AB can be sensitive to the degree ones attention can be overly engaged to food stimuli, to the best of our knowledge, no studies have examined AB between obese and normal-weight individuals. The current study aims to examine attentional bias to food stimuli in an AB-task in a sample of obese and normal-weight adolescents using EEG. We investigated adolescents considering this population is increasingly at

risk for following an unhealthy trajectory of lifetime obesity and adolescence is also a period in which neural systems involved in the regulation of emotions and motivations mature (Powers and Casey 2015; Woltering and Shi 2016).

We hypothesize that obese adolescents, when compared to their Normal-weight peers, show a stronger AB (lower T2 accuracy) to food compared to neutral stimuli, suggesting their attentional resources are captured by food stimuli to a greater degree. Furthermore, in line with Denke et al. (2018), we expect larger N2 amplitudes for the Obese group, suggesting ineffective conflict processing, to incorrect T2 (when a blink occurred). In accordance with the extant literature on P3 in the context of AB tasks, we would expect a smaller P3 in the Obese, compared to the Normal-weight group, suggesting a lack of working memory updating.

# Methods

### **Participants**

Participants were recruited through campus advertisements in the Bryan, TX, and College Station, TX area. The inclusion criteria included: (a) Normal or corrected-to-normal vision; (b) Aged 13–19 years; and c. Free of current psychiatric diagnoses. No participant refused to participate before or during the experiment. All 40 participants (16 males, 24 females; M (age) = 16.90, *SD* (age) = 1.79) were included for data analysis.

Body mass index (BMI; in kg/m<sup>2</sup>) offered a feasible measure to assess weight status in children and adolescents (Ashwell 2011; Pietrobelli et al. 1998). BMI percentile scores have been suggested to strongly correlate with direct measures of total body fat such as dual-energy x-ray absorptiometry in adolescent samples (Pietrobelli et al. 1998; Mei et al. 2002). Participant's height was measured to the nearest millimeter using a stadiometer, and weight was assessed to the nearest 0.1 kg using a digital scale. Body mass index (BMI) was calculated by dividing weight (kg) by height squared (m<sup>2</sup>).

Participants were classified into either the Obese or Normal-weight group based on their BMI percentile scores. The Obese group (n=22) included participants with a BMI equal or larger than the 95th percentile, and the Normal-weight group (n=18) included participants with a BMI less than the 95th percentile based on the BMI-for-age growth chart (Kuczmarski 2002). The BMI cut offs for the Obese and Normal-weight group by age and gender can be found in supplement A. The present study was approved by the Institutional Research Ethics Board (IRB) at Texas A&M University (protocol reference: IRB2010-0877D). As per IRB guidelines, one of the parents/guardians of all participants regardless of the age range of the participants (13–19 years old), was required to sign the consent form. All participants were required to sign the assent form. For participants aged 18–19 years, parents were not required to be present during the laboratory visit. Participants received a \$30 Amazon gift card at the end of the experiment.

# **Electrophysiological Recordings and Procedure**

Upon arrival, research assistants gave the participant and the parent a tour of the laboratory and provided a brief explanation of the experiment. Once all written consent and assent forms were obtained, participants were instructed to complete questionnaires on an iPad. On average, participants completed the questionnaire part within 30 min. Research assistants then applied the sensor net to the participant's head. EEG was recorded from 129 electrodes using an Electrical Geodesics™ (EGI-PHILIPS) high-density EEG system and digitized at a rate of 250 Hz, using Cz as the recording reference. As recommended for the Electrical Geodesics high input-impedance amplifiers, impedances were checked to be below 50 k $\Omega$  before and after the experiment. All channels were preprocessed online using a 0.1-Hz high-pass and 100-Hz low-pass filtering. EEG was recorded in a soundproof testing room that was held at a constant but cool temperature (around 70 °F) (as recommended by, Kappenman and Luck 2010).

After applying the EEG net, participants were presented with one practice block of the attentional blink (AB) task. Participants were instructed to maintain gaze on the center of the screen and to avoid eye and other movements. During the practice block containing ten trials, research assistants visually inspected the participant's eye blinks in response to stimuli and provided direct feedback. The research assistants then left the testing room, and the experiment began. Participants were allowed to take short breaks (1–2 min) between blocks. On average, participants finished the ABtask within 35 min. After this, the participants completed a short Go-Nogo task to assess neural and behavioral correlates of response inhibition (results published elsewhere: Chen et al. 2018).

## Measures

# Surveys

In addition to the AB-task, we also assessed a number of surveys on elements of the eating behavior of the participants to better describe the sample and for the purpose of examining convergent validity.

**Self-efficacy of Eating Behaviors** Self-efficacy of eating behaviors was assessed using the 8-item Weight Efficacy Lifestyle Questionnaire-Short Form (WEL-SF) (Ames et al.

2012). Previous studies showed the WEL-SF is a well-validated measure that can be used in clinical practice and different research settings. The Cronbach's alpha for reliability ranged from good to excellent: 0.86 to 0.92 (see, Ames et al. 2012, 2015). The measure yields a total score of 80 with lower scores revealing less confidence in ones' ability to control eating behavior in different challenging situations (specific items in the WEL-SF measure can be found in Supplement B).

**Restriction of Food Intake** Parental restriction feeding behavior of food intake was assessed using the Restriction subscale from the Child Feeding Questionnaire (CFQ) (Birch et al. 2001). The construct validity of the CFQ among parents of adolescents was confirmed by previous studies (Kaur et al. 2006). The restriction subscale assesses the extent to which parents restrict their child's access to food. Parents' degree of agreement was assessed on a five-point Likert scale (disagree to agree). An average score of all eight items was used as an indicator of a parent's restrictive behavior for food with a higher score meaning more restricted parental feeding behavior (specific items in the CFQ-Restrict measure can be found in Supplement C).

#### **Attentional Blink Task**

The AB task we used was adapted from Denke et al. (2018) and we refer to that paper for technical details on the stimuli and presentation. Our AB task contained 4 blocks with 123 trials each. Each trial consisted of an RSVP stream of 17 black and white images, randomly presented for 75-120 ms, and jittered trial-by-trial to aid in ERP processing. At the end of each trial, participants were asked to identify Target 2's (T2's) direction (e.g. tilted house left, right, or house not seen) and T1's content. Accuracy was determined by the proportion of trials that the participant correctly reported T2 (e.g. tilted house left, right, or house not seen). T1 was either a neutral (e.g., chair; n = 164 trials), negative emotion (e.g., knife; n = 164 trials) or high-calorie food (e.g., burger; n = 164 trials) stimulus and marked by a yellow frame (see Fig. 1). The distance between T1 and T2 was categorized into two types of lags balanced across conditions (Negative Emotion, Food or Neutral images): Lag 2 (324; 66% of total trials) or Lag 8. In the Lag 2 trials, T2 appeared two images after T1, while in Lag 8 trials, T2 was presented eight images after T1. Since the AB would occur within 500 ms after T1, the Lag 2 trials would cover the time period that AB's are more likely to occur (150-240 ms) and Lag 8



#### **Rapid Serial Visual Presentation**

Fig. 1 Depiction of attentional blink task

trials would cover the period that an AB was less likely to occur (600–960 ms).

To ensure participants paid attention to both T2 and T1 and also limit the duration of the AB task, each block contained 10 unprompted trials in which we asked about the T1 stimulus (total 40 trials for the entire task) counterbalanced for condition at random time points. Accuracy on T1 was assessed by a multiple choice question with 3 options on the content on the T1 stimulus. The Normal-weight group showed, on average, 69.3% correct T1 trials (n=27.7, SD=6.5), while the Obese group showed 71.8% correct T1 trials (n=28.7, SD=4.1) on average. There was no significant difference between groups on T1 accuracy, t(38)=0.57, p=0.573.

## **EEG Data Processing and Analysis**

Using Net Station software (Electrical Geodesics Inc, EGI), data was first filtered using a 0.1-50 Hz finite impulse response (FIR) bandpass filter. Then, the correct Food, incorrect Food, correct Neutral and incorrect Neutral trials were segmented into 1000 ms epochs from 200 ms before to 800 ms after T2 stimulus onset. Segmented files were then scanned from artifacts with automatic algorithms for the detection for eye blinks, eye movement, as well as large drifts, and spikes in the data. Eye blinks were detected when the vertical eye channels exceeded a threshold of 120 µV (max-min) within a 160 ms (moving) time window for each trial after running a 20 ms moving-average smoothing algorithm across the entire trial period. Eye movements were detected when horizontal eye channels exceeded a threshold of 120 µV (max-min) over a 200 ms time window. Channels were automatically marked as bad when they exceeded a transition threshold of 200 µV (max-min) over the entire segment. Segments that contained more than 30 bad channels were automatically removed. Bad channels were replaced with a statistically weighted spherical spline interpolation from the full channel set. Finally, EEG data were visually inspected by a trained research assistant.

EEG Data were then transferred to Matlab R2018b (Mathworks, Natick, MA, USA) after they were re-referenced to an Average Reference montage for further processing. In terms of trial counts, participants in Obese and Normal-weight groups did not significantly different in trail count for either the Food trials (Obese: mean (SD)=106.9 (7.3); Normal-weight: mean (SD)=101.3 (16.6); p=0.20) or Neutral trials (Obese: mean (SD)=108.1 (5.6); Normal-weight: mean (SD)=101.7 (15.5); p=0.11). Data were then detrended and baseline corrected using 200 ms preceding the stimulus onset and averaged across all trials based on different conditions (correct Food, incorrect Food, correct Neutral and incorrect Neutral).

Sites and time window for hypothesized components (N2, P3) were determined a priori based on published results from study using a similar task (Denke et al. 2018) and subsequently confirmed by visual inspection using the grand averaged waveform that combined both groups. The sites of the components were determined by visual inspection using the grand averaged topo plot in the windows of interest. The N2 was the largest peak negative deflection with a fronto-central topography (averaged electrodes recorded around FCz, Fz, and Cz) between 350 and 450 ms after the T2 onset. The P3, a longer-duration waveform, was determined by taking the mean activation across central sites (averaged electrodes recorded around FCz, Fz, and Cz) between 400 and 550 ms.

#### **Analysis Plan**

Since the purpose of this paper was to identify the difference between Obese and Normal weight group related to food, we decided to focus on Food and Neutral conditions of the task in the present analysis. For behavioral data, accuracy was calculated for both Conditions (Food, Neutral) and Lags (Lag 2 and Lag 8) for each participant. First, a task validation testing whether an AB was elicited was conducted using a 2×2 repeated-measures ANCOVA with Lags (Lag 2, Lag 8) and Conditions (Food, Neutral) as independent variables and accuracy as the dependent variable. If the AB task successfully captured the AB phenomenon, we aimed to analyze Lag 2 accuracy trials using  $2 \times 2$  repeated measures analysis of covariance (ANCOVA) with Conditions (Food, Neutral) as independent variables, Groups (Obese, Normal-weight) as a fixed factor, and age as covariates. For the EEG data, N2 and P3 mean amplitudes were analyzed separately using a  $2 \times 2 \times 2$ repeated-measures ANCOVA with Conditions (Food, Neutral) and AB-presence (Correct, Incorrect) as within-subject factors, Groups (Obese, Normal-weight) as between subject factor with age and gender as covariates.

Post-hoc *t*-test was conducted using Bonferroni adjustment for multiple comparisons. Partial  $\eta^2$  values were computed as effect sizes, where  $\eta^2 = 0.01$ , 0.10 and 0.25 corresponds to small effect size, medium effect size and large effect size respectively (Vacha-Haase and Thompson 2004). Data points of ERP as well as behavioral variables that are larger than three times of its standard deviation were categorized as outliers. No outliers were detected and removed from the data analysis. All the analyses were conducted using Jamovi 0.9 (Jamovi Project, Amsterdam, The Netherlands), an R based statistical platform (R Core Team 2013).

## Results

## **Task Validation**

For task validation purposes, a  $2 \times 2$  repeated-measures ANCOVA was run to investigate the impact of Lags (Lag 2, Lag 8) and Conditions (Food, Neutral) on task performance. If the AB task successfully captured the AB phenomenon, T2 in Lag 2 trials should be less likely to be detected compared to Lag 8 trials. In other words, we would expect lower accuracy in Lag 2 trials compared to Lag 8 trials. As we expected, the result of accuracy showed a significant two-way interaction between Lags and Conditions, F(1, 39)=11.70, p=0.001, partial  $\eta^2 = 0.23$ . Bonferroni-adjusted post-hoc *t*-test on simple main effects suggested the accuracy of Lag 2 trials was significantly lower than Lag 8 trials for both the Food, t(39) = -8.15, p < 0.001, and Neutral condition, t(39) = -3.60, p = 0.003. Thus, the AB task successfully captured the AB phenomenon.

#### **Behavioral and Survey Outcomes**

Table 1 shows the demographic, behavioral performance, and survey data for the participants broken down by Group to characterize our sample. Participants in the Obese group had a significantly higher BMI (p < 0.001), was older (p = 0.002) and showed lower accuracy on the Neutral Lag 8 (p = 0.005) and Food Lag 2 trials (p = 0.005). Obese participants showed significant lower self-reported self-efficacy of eating behaviors in challenging situations (p < 0.001). No group differences were found on the parents' restrictive feeding behavior measure. Due to the significant difference of age between groups, participant age was controlled in further repeated-measures ANCOVA analyses.

We further examined the relationship between task performance measures and survey measures. Results suggested significant associations between the accuracy of Food trials, BMI, self-efficacy of eating behavior, and parental restrictive feeding behavior. The correlation matrix and scatterplots are displayed in supplementary figure D. Specifically, the results showed that the accuracy of Food trials was negatively correlated with an individual's BMI, r(39) = -4.02, p = 0.010. Second, the accuracy of Food trials was positively associated with an individual's rating on their self-efficacy of eating behaviors, r(39) = 0.39, p = 0.013. There was a significant positive correlation between parents' restrictive feeding behavior and individuals' BMI, r(39) = 0.33, p = 0.038. No significant association between parental restrictive feeding behavior and the accuracy of Food trials was found, r(39) = 0.10, p = 0.544.

#### **ERP Outcomes**

Figure 2 shows the grand averaged ERP waveforms comparing Food and Neutral conditions for both Obese and Normal-weight group. The repeated-measures ANCOVA for N2 amplitudes revealed no significant main effects and interactions. The repeated-measures ANCOVA for the P3 amplitudes revealed a significant three-way interaction (Condition × AB-presence × Group), F(1,35) = 4.63, p = 0.038,  $\eta^2 = 0.117$ . Bonferroni-adjusted post-hoc *t*-tests suggested that when an AB was present in the Food trial (incorrect Food trial), the Normal-weight group showed significantly larger P3's compared to the Obese group, t(35) = 3.42, p = 0.020. Figure 3 illustrates the Estimated Marginal Means of P3 amplitudes by Condition, AB-presence, and Group with the error bars indicating the standard error of the marginal means (Table 2).

 Table 1
 Mean and standard

 deviations for demographic,
 behavioral performance, and

 survey measures of the obese
 and the normal-weight group

Variables	Obese $(n = 22)$	Normal-weight $(n = 18)$	<i>p</i> -Value
Demographic			
Age	17.7 (1.21)	15.9 (1.98)	0.002
Gender (% of female)	50%	72%	0.154
BMI	41.8 (8.44)	22.4 (2.19)	< 0.001
Behavioral performance (acc	euracy)		
Neutral Lag 8 trials	0.62 (0.10)	0.72 (0.11)	0.005
Neutral Lag 2 trials	0.61 (0.07)	0.62 (0.07)	0.785
Food Lag 8 trials	0.62 (0.09)	0.65 (0.10)	0.362
Food Lag 2 trials	0.48 (0.07)	0.55 (0.07)	0.005
Survey measures			
WEL-SF	51.94 (9.92)	67.29 (6.28)	< 0.001
CFQ-restrict	3.06 (0.86)	2.83 (0.95)	0.415

WEL-SF Weight Efficacy Lifestyle Questionnaire-Short Form, *CFQ-Restrict* restriction subscale from the Child Feeding Questionnaire (CFQ)



Fig. 2 Averaged ERP waveforms between groups

# Relationship Between ERP Components and Survey Measures

To further investigate the relationship between ERP components and behavioral measures at an individual level, we examined the correlations between the individual's ERP components and their response on the self-efficacy of eating scale, parental restrictive feeding scale, and BMI.

The P3 amplitudes at AB-present food trials (incorrect food trials) were negatively correlated with individual's BMI, r(39) = -0.33, p = 0.037. The results suggested that individuals with a higher BMI were more likely to show lower P3 amplitudes in food trials when an AB was

present. The P3 amplitudes also positively correlated with individual's rating on their self-efficacy of eating behaviors, r(39) = 0.33, p = 0.037. The results revealed an association between better self-efficacy and larger P3 amplitudes during the food trials.

No significant correlation was observed between N2 amplitudes and behavioral measures for AB present food trials and AB present neutral trials. No significant correlation was observed between parental restrictive feeding behaviors and ERP component amplitudes. The correlation matrix and scatterplots are displayed in supplementary figure E.



Fig. 3 Estimated marginal means of P3 amplitudes by condition and group

 Table 2
 Mean and standard deviations for ERP amplitudes for Food and Neutral trials

	ERP amplitudes (µV)		
	Obese	Normal-weight	
AB present (incorrect)			
N2			
Neutral	- 0.68 (0.82)	- 0.80 (0.93)	
Food	- 0.81 (0.88)	- 1.00 (0.87)	
P3			
Neutral	1.38 (0.97)	1.70 (0.71)	
Food	1.23 (0.56)	2.22 (1.26)	
AB absent (correct)			
N2			
Neutral	- 1.21 (1.27)	- 0.84 (1.16)	
Food	- 1.00 (1.33)	- 0.62 (0.93)	
Р3			
Neutral	1.48 (0.82)	2.16 (0.83)	
Food	1.74 (1.27)	2.17 (1.27)	

# Discussion

Our study set out to explore behavioral and ERP correlates of attentional bias to food in adolescents with obesity. Behaviorally, the Obese group showed lower accuracy in AB trials related to food compared to the Normal-weight group. We found that the P3 during an AB was smaller in obese participants compared to their normal-weight peers suggesting their brains had a lower ability of updating working memory after their attention was captured by food stimuli. Further converging evidence was found in our survey measures for the notion that the P3 may indicate attentional resources being overly dedicated to food stimuli: lower P3 amplitudes during an AB in Food trials were associated with larger BMI as well as poorer self-efficacy of eating behaviors.

Behaviorally, our findings supported our hypothesis that obese adolescents had a stronger AB than their peers to food stimuli, and *not* the neutral trials, during the Lag 2 trials in which the AB was most likely to be present. These findings were in line with literature suggesting that the AB is subject to individual differences in personal motivation (Dale and Arnell 2010; Olivers and Nieuwenhuis 2005; Shapiro and 2006) as well as other literature examining attentional bias using different paradigms (Brignell et al. 2009) but can now be extended to obese adolescents relation to food.

Our hypothesis expecting larger N2 amplitudes for the obese group, which was loosely based on the study by Denke et al. (2018), did not bore out. There are, however, some notable differences between our studies that could explain discrepancies. Our study used a different sample (adolescents versus undergraduate students) as well as a different design (directly comparing obese adolescents with their peers instead of a meditation analysis within a normative sample). More importantly, Denke et al. (2018) did not use food stimuli but used highly fear-based images taken from the International Affective Picture System (IAPS; Lang et al. 2008) which, for example, portrayed scenes of explicit violence. Furthermore, their analysis also directly tested for whether the relation between degrees of anxiety and emotional eating were mediated by the N2. It is therefore possible that our stimuli were simply not intense enough to elicit an N2 effect and/or that N2 activation would generally be stronger when directly related to conflict processing in the context of fear processing and emotional problems (Hum et al. 2013; Sehlmeyer et al. 2010; Woltering et al. 2011).

Finally, as we hypothesized, we found smaller P3s in the Obese group compared to the Normal-weight group suggesting a lack of working memory updating. These findings were consistent with studies that associated a low, or absent, P3 with a lack of working memory updating in AB tasks (Martens et al. 2006; Vogel et al. 1998). No studies were available, however, examining ERPs in an AB task in populations with food-related disorders. Our study suggest that in obese participants, T1 food stimuli capture attentional resources to such an extent that working memory has difficulty updating for T2. Our findings are in line with another fMRI neuroimaging study in 35 female adolescents using the food attention network test (ANT) which found greater attentional bias to food, and activity related brain regions, to predict future increases in BMI (Yokum et al. 2011).

A number of other, non-hypothesized, findings stood out to us. First, our measure of self-efficacy, or one's ability to control their eating behavior in challenging situations, was lower for the Obese compared to the Normal-weight group and was remarkably sensitive to neural and behavioral indices of the AB. Not only was higher self-efficacy related to better performance on food trials (suggesting less ABs) but a higher self-efficacy also had a positive linear relationship with P3 amplitudes. These findings may suggest the relation of attentional resource allocation and updating to self-regulatory processes in the brain (Chen et al. 2018; Inzlicht and Schmeichel 2012; Mann and Ward 2007). Second, the parental food restriction subscale (Birch et al. 2001) was not different between our Obese and Normal-weight sample yet we did find a positive correlation between BMI and parental food restriction. These findings suggest that parental influence on food intake in adolescence may be effective on controlling BMI but is not specific to children being obese or not. This measure did not correlate with any of the ERP amplitude or behavioral measures related to the AB task.

The present study has a number of strengths and limitations. A strength of the study was the analysis employed. The multifactorial repeated-measures ANCOVA allows for the testing of several contrasts within one model. More specifically, next to whether the Obese and Normal-weight participants were different for a dependent variable, we could simultaneously assess the veracity of the findings across Lags and Conditions while controlling for extraneous effects. Another strength was the degree of convergent validity in our P3 outcomes which directly correlated with BMI, behavioral, and psychological self-report measures related to food behavior. A weakness was our relatively low sample size and reliance on temporal instead of more neuroanatomical methods of analyzing and measuring neural activation. Further, our study was not designed to test downstream behavioral effects, such as resulting approach-avoidance tendencies, to being exposed to food stimuli nor does the cross-sectional nature of our design allow for a direct causal inference of what our AB effects may mean for people's choices and behaviors related to food intake in real life.

The implications of our findings are still more fundamental in nature. We found that food stimuli get prioritized by attention over neutral stimuli in obese adolescents which helps explain why obese individuals often fail to restrict their food intake (Blundell and Gillett 2001; Jeffery et al. 2000). Though these rapid attentional processes are often considered to be bottom-up, it is likely they are influenced by top-down processes (Folk et al. 2002; Hommel et al. 2006). Our findings, relating processes of self-regulation to our neural and behavioral AB, suggest that techniques of bolstering self-regulation may be a helpful component for some when facing this public health crisis. The present study has the potential to influence treatment plans by addressing cognitive strategies aimed at reducing automatic biases.

This is one of the first studies examining neural correlates of attentional processing in adolescents obesity. Our findings suggest that obese adolescents may have difficulty disengaging from food stimuli, which may explain their focus on food over more neutral stimuli compared to their peers. Our findings contribute to a growing literature examining neuro-cognitive components of obesity.

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