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Attentional Blink: An Antecedent to Binge Eating Behavior

A Thesis

Submitted to the Graduate Faculty of the
University of New Orleans
in partial fulfillment of the
requirements for the degree of

Master of Science
In
Psychology
Biopsychology

by

Gregory Denke

B.A. University of Southern Maine, 2012

December, 2014

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Abstract

This study examined how attentional sub-processes contribute to binge-eating. Dense-array EEG and a version of the canonical attentional blink task were used to ascertain the neural correlates underlying the attentional sub-processes that comprise the Posner model of attention (alerting, orienting, and executive control) and how attentional activation differs for binge-eaters vs. non-binge eaters. Furthermore, we examined a number of the event-related potentials (ERP), including P2 activation, which has been linked with orientating of attention, and N2 activation which has been linked with attentional conflict. We found decreased P2 activation for binge-eaters, in the negative condition, for incorrect target 2 (T2) detection trials. We also found more N2 activation for binge-eaters than non-binge eaters, in negative trials when T2 was not detected. This pattern of results suggest that binge-eaters showed deficiencies in allocating attention to stimuli that followed negative images; this attention deficiency may be a key factor for binge-eating behavior.

Binge-eating, Event Related Potential, Dense Array EEG, Attentional Blink, Posner Model of Attention

Introduction

1.1 Prevalence and implications of obesity

Obesity makes life more difficult for the individual, causes problems for all of society, and has shown incredible growth and worldwide pervasiveness (Falkner et al., 2001; Finkelstein, Trogon, Cohen, & Dietz, 2009; Flegal, Carroll, Kit, & Ogden, 2012; Reilly et al., 2003). Obese individuals face numerous health and psychosocial consequences and everyone, obese or not, is subjected to the economic consequences of an obesity epidemic (Reilly et al., 2003). Adverse health consequences, such as increased risk for heart disease, type 2 diabetes, cancers, hypertension, elevated cholesterol levels, stroke, liver and gallbladder disease, respiratory problems, osteoarthritis, and reproductive problems are all associated with obesity (Falkner et al., 2001; Initiative, 2004). Furthermore, obesity associated disease reduces the life expectancy of severely obese persons by an estimated 5 to 20 years (Fontaine, Redden, Wang, Westfall, & Allison, 2003). Psychosocial consequences for the obese are abundant and include low self-esteem and impaired social functioning (Kolotkin, Meter, & Williams, 2001). Finkelstein, Fiebelkorn, and Wang (2003) estimated that the medical expenditure attributed to obesity in 1998 was \$78.5 billion, accounting for 9.1 percent of the total US medical expenditure. As of 2008, obesity related medical expenditures had grown to an estimated \$147 billion (Finkelstein et al., 2009). Given the high obesity rate and increasing health care costs, the growth of obesity related medical expenditures is expected to continue (Thorpe, Florence, Howard, & Joski, 2004).

For the United States as well as for numerous other countries, the growth and pervasiveness of obesity is large scaled and alarming. Obesity rates in the U.S. were found to be 35.5 percent among adult men and 35.8 percent among adult women in 2009 to 2010 (Flegal et al., 2012). Over the past 20 years, obesity rates have increased in the U.S. and worldwide (James, Leach,

Kalamara, & Shayeghi, 2001). More than one-third of U.S. adults and 16.9 percent of children aged 2-19 years are obese (Ogden & Carroll, 2010) and overweight parents are more likely to have obese children (Garn et al., 1976). Given the abundant worldwide growth of obesity and the resultant harm incurred by individuals and society, exploring antecedents to this issue is not only valuable, it is critical.

In general, obesity is caused by consuming more calories than will be expended. However, there are numerous factors that cause this unbalanced calorie consumption/calorie use relationship. According to The Centers for Disease Control (CDC, 2012) genes, metabolism, behavior, environment, culture, socioeconomic status, diseases, and drugs are all implicated in the cause of obesity. Among these factors, behavior, binge-eating plays a significant role (CDC, 2012). Given the numerous factors that can lead to obesity, an exhaustive exploration of the antecedents is beyond the scope of this study. Hence, this investigation will focus only on binge-eating behavior. More specifically, the current study will use neurophysiological measures to decompose the factors contributing to binge-eating behavior. However, first we are going to describe binge-eating behavior and discuss contributing factors. Second, we will outline a cognitive process that may contribute to binge-eating behavior. Lastly, we are going to drill down into this cognitive process to investigate what specific neural mechanisms underlie binge-eating behavior.

1.2 Binge-eating

According to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), binge-eating disorder (BED) is defined as the recurrence of binge-eating episodes, or a high frequency of binge-eating. A binge-eating episode is characterized by eating a greater amount than most in a discrete amount of time, feeling a loss of control over the binge-eating,

and the presence of 3 of the following criteria; eating more rapidly than normal, eating until uncomfortably full and feeling self-disgust, eating large quantities of food when not physically hungry and eating alone due to embarrassment, guilt or depression after bingeing (APA, 2013). Spitzer and colleagues (1993) found that 43 percent of participants with BED had a history of obesity compared to 27 percent of participants without BED; the overall prevalence of BED was found to be 28.8 percent among individuals in weight control programs. In a non-clinical sample of 817 women aged 20-45, French and colleagues (1999) found binge-eating to be twice as prevalent among overweight women (21 percent) compared to normal weight women (9 percent). In yet another non-clinical sample, Hill and Williams (1998) found binge-eaters to weigh more, have lower self-esteem, and score higher on the Mental Health Inventory, a questionnaire used to evaluate psychological health (Stewart, Hays, & Ware, 1988). Thus, not surprisingly, a large number of individuals who struggle with obesity also have binge-eating tendencies.

1.3 Etiology of binge-eating tendencies

The study of binge-eating behavior has focused on a few different precursors, such as excessive dietary restraint (at any point in life), a history of childhood sexual abuse, and depression (Agras & Kirkley, 1986; Hall, Tice, Beresford, Wooley, & Hall, 1989; Kuehnel & Wadden, 1994; Marcus et al., 1990; Marcus, Wing, & Hopkins, 1988; Palmer, Oppenheimer, Dignon, Chaloner, & Howells, 1990; Polivy & Herman, 1985; Waller, 1991). Studies have found that a high rate of individuals who engage in binge-eating behavior have either experienced childhood sexual abuse (Hall et al., 1989; Palmer et al., 1990; Waller, 1991) and/or bouts of depression (Kuehnel et al., 1994; Marcus et al., 1990; Marcus et al., 1988). Furthermore, dietary restraint is believed to initiate binge-eating behavior by depriving individuals of food, to an

extent which cannot be maintained, which ultimately leads to overeating (Agras et al., 1986; Polivy et al., 1985). However, Stein and colleagues (2007) found that while factors, such as dietary restraint may contribute to binge-eating behavior, negative mood was more frequently the catalyst for this behavior.

There have been numerous studies that have shown binge-eating episodes to be triggered by emotion (Arnou, Kenardy, & Agras, 1995; Cattanach, Malley, & Rodin, 1988; Chua, Touyz, & Hill, 2004; Elmore & De Castro, 1990; Heatherton & Baumeister, 1991; Grange, Gorin, Catley & Stone, 2001; Greeno, Wing, & Shiffman, 2000; Grilo, Shiffman, & Carter-Campbell, 1994; Johnson & Larson, 1982; Lingswiler, Crowther, & Stephens, 1989; Masheb & Grilo, 2006; Van Strien, Frijters, Bergers, & Defares, 1986; Waters, Hill, & Waller, 2001). Lingswiler and colleagues (1989) found stress, preoccupation with food, and negative moods to be precursors to binge-eating episodes. Grange and colleagues (2001) found negative affect to be an antecedent for binge-eating episodes for obese women with and without BED. Similarly, binge eating non-BED women tended to experience worse mood than non-BED women who did not binge-eat (Greeno et al., 2000). Chua and colleagues (2004) found an effect of mood on amount of food eaten for obese binge-eaters and the desire to eat when experiencing negative moods was found to increase binge-eating levels for individuals with BED (Arnou et al., 1995) as well as for normal weight individuals who regularly binge-eat (Grilo et al., 1994). Lastly, emotional eating is believed to improve an individual's mood, reduce their anxiety, and distract them from negative feelings (Elmore et al., 1990).

Given that a negative emotional state has been implicated as a primary trigger for binge-eating episodes (Waters et al., 2001), often interfering with cognitive control over eating behavior (Engelberg, Steiger, Gauvin, & Wonderlich, 2007), it may be that binge-eating results

in part from an attempt to avoid or dampen negative feelings. Heatherton and colleagues (1991) explored the phenomena, cognitive narrowing, which is used by binge-eaters as an attentional escape from emotional distress. Employed to eliminate upsetting thoughts from consciousness, cognitive narrowing refers to a narrowed attention span that focuses only on the immediate present. Narrowing of attention disengages normal inhibitions against eating by focusing on an exclusive small range of stimuli (Heatherton et al., 1991). Thus, in the context of negative emotion, binge eaters may focus solely on food (cognitive narrowing) to avoid thinking about negative events, but this cognitive narrowing also prevents them from thinking about ongoing dietary restraints thereby facilitating binge-eating episodes.

However, the concept of cognitive narrowing may also be applied in a second attentional process. It may be that binge eaters narrow their attention on current negative events, i.e., ruminate, and that this ruminative behavior prevents thoughts about ongoing dietary restraints and contributes to binge-eating episodes. This theory is supported by the fact that a number of studies have found a strong link between binge-eating behavior and rumination (Fairburn et al., 1995; Kubiak, Vögele, Siering, Schiel, & Weber, 2008; Nolen-Hoeksema, Stice, Wade, & Bohon, 2007). Consequently, emotion may trigger binge-eating through two attentional processes: 1) emotion-induced cognitive narrowing on food to avoid negative thoughts and thus leading to unrestrained eating behavior, and 2) emotion-induced cognitive narrowing or ruminating on negative thoughts, also causing unrestrained eating behavior.

1.4 Evolutionary role of attentional narrowing and binge-eating behavior

Perhaps at the core of binge-eating behavior rests once vital survival behaviors not needed as much in the modern environment, i.e. the ability to quickly locate and focus attention on important stimuli. Along with water and shelter, food and danger recognition are among the

most important stimuli in terms of survival. Moreover, excessive attentional focusing has been found to be triggered by both threatening (Pratto & John, 1991) and rewarding stimuli (Treue, 2003), and these survival relevant stimuli have been shown to attract attention away from other stimuli. In modern times, food cues are all around us in everyday life. We live in an environment in which we are bombarded with food images, which entice us to eat (as discussed in Hill et al., 1998). In an environment where food is limited, this food attention narrowing or bias is beneficial in terms of survival (Dachner & Tarasuk, 2002). However, in the food enriched environment in which we live, a food attention bias may not be beneficial and may actually disrupt long term dietary plans (Drewnowski & Darmon, 2005). Similarly, in a hostile environment a negative attention bias keeps attentional focus on any present danger which promotes safety (Öhman & Mineka, 2001). However, in the relatively safe environment in which we live, a negativity bias may be turned inward leading to rumination or dwelling on negativity that exists within one's life (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). As outlined above, both of these attentional biases or attentional narrowing processes (i.e., focusing on food and focusing on negative events) have been linked with binge-eating behavior (Drewnowski & Darmon, 2005; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

1.5 Attentional mechanisms contributing to binge-eating behavior

Understanding how food and negative images evoke attentional biases (Braet et al., 1997; Ferriday et al., 2008, 2010; Harris et al., 2009; Hill et al., 1998; Jansen et al., 2003; Lavie et al., 1995, 1997; Lutz et al., 2008; Oakes et al., 2000; Piech et al., 2010), cause attentional narrowing (Heatherton et al., 1991) and thereby avoidance of dietary restraint measures, may be key to understanding binge-eating behavior. However, since overall attentional processing is comprised of numerous attentional sub-processes—for example, the Posner Model of attention decomposes

attentional processes into three sub-processes (Posner & Boies, 1971)—it may be that only some attentional sub-processes are dysfunctional in the context of binge-eating behavior. While it is well known that overarching attentional processes, such as cognitive narrowing, contribute to binge-eating behavior (e.g., Heatherton et al., 1991), to the best of our knowledge, the specific attentional sub-processes underlying these attentional biases have not been explored. Therefore the current study will use event-related potentials (ERPs) to characterize the sub-processes of attention, as outlined by the Posner Model of Attention, to decompose the mechanistic chronology underlying attentional deficits contributing to binge-eating behavior.

In this study, we examine the sub-processes of attention that contribute to binge-eating behavior in the context of task that allowed for the assessment of neural activation both when salient cues were encoded and missed. It has been found that when an individual focuses a large amount of their attentional resources on a particular item, there is an increased likelihood that a subsequent item will not be recognized (Raymond, Shapiro, & Arnell, 1992). This phenomenon, referred to as an attentional blink, is believed to be the result of using a large amount of attentional resources for the processing of one stimulus, resulting in an inadequate amount of resources left to process a subsequent stimulus. An attentional blink can be reliably measured using a standard paradigm frequently used for this phenomenon (Di Lollo, Kawahara, Ghorashi, & Enns, 2005; Raymond et al., 1992; Shapiro, Raymond, & Arnell, 1997; Vogel, Luck, & Shapiro, 1998). Thus, in the following sections, we will depict how the Posner Model of attention and corresponding ERPs contribute to binge-eating behavior in the context of the attentional blink task. In short, it may be that binge eating behavior is in part caused by attentional deficits brought about by emotion-induced attentional blinks. In the current study, we use a laboratory environment to emulate real world emotion-induced attentional blinks. While

this is a useful approach to decomposing the attentional mechanisms contributing to binge eating behavior, it is likely not as salient as real world emotions. Thus, the current project likely has a much smaller temporal scale than might be found for real world emotion-induced binge eating. Before we further discuss the attentional mechanisms that contribute to binge eating behavior, we will first outline why ERPs are ideal for decomposing such a process model.

ERPs are computed by averaging together EEG data from similar trials. The averaged waveform that is generated is comprised of various positivities and negativities in amplitude, and each of these inflections or deflections are considered an ERP. ERPs are generally labeled in relation to the time-locking stimulus, e.g., the P2 is the second positive inflection after stimulus onset. While scalp activation patterns comprising ERPs are not able to identify specific neural generators, they do allow for very precise decomposition of the chronology underlying cognitive processes, such as attentional deficits. Other non-electromagnetic neuroimaging techniques are not able to provide this temporally specific decomposition of neural processes.

1.6 ERP characterization of the Posner Model of attention

The Posner Model of attention (Posner et al., 1971) proposes that attention can be broken down into three sub-processes: alerting, orientating, and executive control (Posner et al., 1971). Additionally, these three sub-processes of the Posner model of attention line up well with three specific ERPs: The contingent negative variation (CNV), the P2, and the N2. We used ERPs to measure the neuromechanistic activation underlying these sub-processes of attention (alerting [CNV], orienting [P2], and executive control [N2]) in the context of the attentional blink task, and how their attentional activation differs for binge eaters vs. none-binge eaters. Additionally, to ascertain if attentional biases contributed to later cognitive processing deficits, we also measured P3 activation. In the following section, we will decompose attentional deficits

triggered by negative emotion contributing to binge-eating behavior using the Posner model of attention and ERPs.

The first attentional mechanism discussed within the Posner model is the process of being in an alert state. This state refers to occurrences in which an individual is expecting a stimulus (previously cued) and thus subsequent responding is more rapid than if the stimulus was unexpected (Posner & Peterson, 1990). Cues to negative stimuli (e.g. the death of a loved one) have been shown to lead to rumination by inhibiting the attentional processing of concurrent information (Joormann, 2006; Linville, 1996). This process of cueing and subsequent rumination is believed to keep attention focused on negative information (Linville, 1996). The CNV is an ERP that has been associated with cue-induced response anticipation/preparation (e.g., van Wouwe, Band, & Ridderinkhof, 2009; Woodman, 2010), and may thus be a neural marker of alerting. The CNV, which primarily involves activation in the frontal cortex, is an ERP component that is slowly generated over several seconds following a cueing (Rebert & Knott, 1970; Walter et al., 1964). Moreover, Walter and colleagues (1964) found increased CNV activation was associated with increased trial-to-trial accuracy. Furthermore, since negatively cued information may lead to alerting towards the negative information or rumination about the negative information, the CNV may be a neural marker underlying this process. . In other words, it may be that the cueing of negative information may lead to rumination and thus drawing attentional resources away from effective preparation/alerting for the next event. This attentional alerting deficit may then lead to unrestrained eating because attention is not effectively drawn towards the eating behavior.

The second attentional mechanism depicted in the Posner model is orienting (Posner et al., 1971). Orientating is the act of directing attention toward a particular stimulus (Posner et al.,

1971) and it has been found to correspond to activation in the intraparietal cortex and superior frontal cortex (Corbetta & Shulman, 2002). The P2, an ERP evoked around 200 ms after stimulus onset, has been linked with attentional orienting (e.g., Huang & Luo, 2006), with increased P2 activation representing increased attentional orientating (Kanske, Plitschka, & Kotz, 2011). More specifically, studies have found that orientating to emotional stimuli enhances P2 activation (Carretié, Mercado, Tapia, & Hinojosa, 2001; Delplanque, Lavoie, Hot, Silvert, & Sequeira, 2004; Kanske et al., 2011). Kranczioch, Debener, and Engel (2003) found P2 activation at frontal and central midline areas with the greatest activation found at the frontal midline area. Interestingly, this is the same general area of activation found by Corbetta and colleagues (2002) to underlie attentional orienting within the Posner Model of attention. Additionally, negative stimuli have been found to draw an individual's attention even when the person attempts to ignore them (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001; Piech et al., 2010) and studies have found that binge-eaters orient their attention to negative stimuli to a greater extent than non-binge eaters (Harrison, Sullivan, Tchanturia, & Treasure, 2010). Hilbert and Tuschen-Caffier (2007) found that attentional orientating to negative events helped to maintain binge-eating behavior among binge-eating group. Thus, deviant orienting of attention to negative stimuli may be predictive of risk for binge-eating behavior (Passamonti et al., 2009). More specifically, binge eaters may have difficulty orienting their attention away from a negative stimulus and towards a subsequent stimulus, e.g., a second event, as is the case within the attentional blink task or food to be consumed. Thus, because of this attentional orienting deficit, binge eaters may not be aware of their unrestrained eating behavior.

The final sub-process of Posner's attentional model is executive control of attention. This sub-process is thought to be an error detection and correction mechanism triggered in the context

of cognitive conflict and has been associated with anterior cingulate cortex activation (Bush, Luu, & Posner, 2000; Fan et al., 2005), a brain region routinely associated with conflict monitoring (Kerns et al., 2004). This attentional sub-process may be measured by examining N2 activation, a mediofrontal ERP component peaking roughly 200-400 ms after stimulus onset, since this ERP component has been shown to reflect conflict monitoring (Donkers & van Boxtel, 2004). Furthermore, the N2 has been found to be evoked by the processing of emotional information (Lamm, Pine, & Fox, 2013; Lamm, White, McDermott, & Fox, 2012; Lewis et al., 2006). Lewis and colleagues (2006) found emotion-related changes in N2 activation within a go/no-go task designed to induce negative emotion. Additionally, Lamm, White, McDermott, and Fox (2012) found greater N2 activation in response to emotional stimuli in comparison to neutral stimuli. Thus, N2 activation is increased in the context of negative emotion/negative events. Because N2 activation represents neural resources recruited to modulate cognitive conflict and is influenced by emotional stimuli, it may be that binge eaters focus excessive attentional neural resources on one event (e.g., negative event) and thus are not able to recruit enough N2 activation to facilitate cognitive awareness of subsequent events (e.g., a second event, as is the case for the attentional blink task, or food stimuli to be consumed). Thus, insufficient N2 activation may contribute to binge-eating behavior.

The fourth ERP component we examined was the P3. Even though it is not part of the Posner Model of attention, it has been associated with deeper processing of information (e.g., Kranczioch, Debener & Engel, 2003), and thus may be a neural marker of affective attentional processing. The P3 is the largest cognitive ERP, peaks at roughly 300 ms after stimulus onset, reaches maximum amplitude at central-parietal midline sites, and is generally evoked by infrequent occurring stimuli (Pritchard, 1981). This component has been linked with processing

of information in working memory, and reduced P3 activation may be a marker of information not making it to working memory (Kranzloch, Debener & Engel, 2003). Detected targets have been found to show an enhanced P3 while missed targets show little or no P3 (Kranzloch et al., 2003; Ritter & Vaughn, 1969). Hillyard, Squires, Bauer, and Lindsay (1971) found a relationship between P3 amplitude and stimuli detection accuracy. More specifically, they found that larger P3 amplitudes corresponded with increased likelihood that stimuli would be accurately detected. Furthermore, Hillyard, Hink, Schwent, and Picton (1973) found that when attention is highly focused on a particular task, additional stimuli that would usually elicit a P3 do not. For binge eaters, a small P3 may indicate ineffective stimulus processing in working memory. Furthermore, larger P3s have also been linked to negative emotionally valiant stimuli (Keil et al., 2002). Bradley, Codispoti, & Lang (2006) found increased attention to emotional stimuli to result in reduced attentional reaction to subsequent stimuli. Therefore, in the context of negative emotion (i.e., target 1), binge eaters may not process additional stimulus information affectively in working memory, and thus leading to attentional unawareness (i.e., missing target two or missing food eating behavior and thus contributing to unrestrained eating). In the next section, we will depict specific hypotheses for each attentional sub-process and the corresponding ERP component.

1.17 Hypotheses

Given that we have three overarching questions, we have broken the hypotheses section into three subsections. The three overarching questions are: 1) how does each of the attentional sub-process ERP activation contribute to increased accuracy/better performance, 2) how does emotional cueing contribute to the neural resources required for specific attentional sub-processes, and 3) how do binge eaters differ from non-binge eaters (group differences) in the

amount of neural resources required for effective attentional processing. Therefore, the three subsections are: Accuracy, Emotion, and Group differences. Furthermore, within each subsection, we will outline specific hypotheses for each ERP component.

1.17.1 Accuracy

1.17.1.a Accuracy (CNV). CNV activation increases with the presence of a cueing/alerting stimulus and increases further with repeated trial accuracy (Walter et al., 1964). In the context of the attentional blink paradigm, CNV activation represents the cueing of a stimulus by a preceding stimulus, i.e., T1 cues T2. CNV activation is generally time-locked to the second stimulus (Smith, Johnstone, & Barry, 2007; van Wouwe, Band, & Ridderinkhof, 2011; Verleger et al., 2006), in this case T2. Thus, we are able to ascertain the amounting of cortical alerting occurring immediate prior to the second event (i.e., T2). Based on this argument, we expected more (greater negativity) CNV activation for accurate trials than inaccurate trials (blinked trials).

1.17.1.b Accuracy (P2). An increase in P2 activation has been linked with effective orientating (Delplanque et al., 2004). In the context of the attentional blink paradigm, P2 activation represents the neural activation underlying attentional orientation or lack of attentional orientation toward stimuli. In other words, are there sufficient attentional resources available to orient attention away from T1 and towards T2 or have attentional resources been maxed out by T1? Based on this argument, we expected increased T2 P2 activation for accurate trials than inaccurate trials (blinked trials).

1.17.1.c Accuracy (N2). N2 activation has been shown to increase as more cognitive resources are recruited (Van Veen et al., 2002). Therefore, in general, we expected increased activation (greater negativity) for more challenging trials. In the context of the attentional blink

paradigm, N2 activation represents the attentional conflict between two competing stimuli. Namely, do attentional resources allocated to T1 override T2 or are T2 attentional resources sufficient to allow for T2 to be noticed? Based on this argument, we predicted increased T2 N2 activation for accurate trials than inaccurate trials (blinked trials).

1.17.1.d Accuracy (P3). P3 activation is linked with the processing of information in working memory, with increased activation for detected targets and little or no P3 for missed targets (Ritter & Vaughn, 1969). In the context of the attentional blink paradigm, P3 activation represents if T2 information makes it into working memory and thus allows for it to be noticed, or does the attention allotted toward T1 cause T2 to be missed? Based on this argument, we expected increased T2 P3 activation for accurate trials when compared with inaccurate trials (blinked trials).

1.17.2 Emotion

1.17.2.a Emotion (CNV). In the context of the attentional blink task, the CNV functions as a cuing or alerting in preparation for a second event. Furthermore, neural activation is often greater in the context of negative emotion (Davidson & Irwin, 1999; Ochsner et al., 2004; Schaefer et al., 2002), Therefore, it may be that in the context of negative T1s, our participants had to recruit additional resources to prepare for T2. Therefore, we predicted increased CNV activation for negative trials compared to neutral trials.

1.17.2.b Emotion (P2). Negative stimuli have been shown to attract more attention than neutral stimuli i.e. negative attention bias (Carretié et al., 2001; Smith et al., 2003, 2006). Therefore, it may be that our participants had difficulty orienting their attention away from T1

stimuli and towards T2 stimuli. Therefore, we predicted a decrease in T2 P2 activation for trials with negative T1s compared to trials with neutral T1s.

1.17.2.c Emotion (N2). Prefrontal cognitive-control-related neural activation is consistently greater in the context of negative emotion compared to relatively neutral contexts (Lamm et al., 2013). Therefore, we expected greater T2 N2 activation for negative trials compared to neutral trials.

1.17.2.d Emotion (P3). Because of previous emotion-induced attentional deficits brought about by difficulties in alerting, orienting, and executive attention, it may be that in the context of negative emotion, events are not fully processed in working memory. Therefore, we predicted decreased T2 P3 activation for negative trials compared to neutral trials.

1.17.3 Group

1.17.3.a Group (CNV). Since binge-eaters have been shown to restrict attentional focus to one stimulus at a time (Heatherton et al., 1991), binge-eaters may maintain more attention on T1 than non-binge eaters (resulting in less or no alerting behavior). Therefore, we expected binge-eaters to show decreased (less negative) CNV activation compared to non-binge eaters.

1.17.3.b Group (P2). As outlined by Heatherton et al. (1991), binge eaters show attentional narrowing in the context of negative events. Additionally, Binge eating behavior has been associated with cognitive rumination on negative events (Fairburn et al., 1995; Kubiak et al., 2008; Nolen-Hoeksema et al., 2007). Thus, it may be that binge-eaters have difficulty orienting away from negative events and towards subsequent events. Therefore, we expected binge-eaters to show decreased T2 P2 activation compared to non-binge eaters for trials with negative stimuli.

1.17.3.c Group (N2). If binge eaters have difficulty orienting away from negative events (see P2 hypothesis), they may therefore have decreased conflict in negative trials between T1 and T2 events (less attentional resources to T2) leading to inaccurate responding. Therefore, we predict decreased (less negative) N2 activation for binge-eaters compared to non-binge eaters for blinked negative trials.

1.17.3.d Group (P3). Because binge-eaters may be affected more by negative stimuli than non-binge eaters (Heatherton et al., 1991; Kubiak et al., 2008) preventing them from orienting away from negative events (see P2 hypothesis) and preventing them from recruiting enough attentional conflict resources (see N2 hypothesis), it may be that binge eaters have difficulty processing T2 information in working memory leading to inaccurate responding. Therefore, we expected binge-eaters to show decreased T2 P3 activation compared to non-binge eaters for negative trials that were blinked.

Method

2.1 Participants

Participants were undergraduate students ($N = 71$) who attended the University of New Orleans. All participants had normal or corrected-to-normal vision, had a hair style that was conducive to EEG collection procedures, and were free of current psychiatric diagnoses. One participant was excluded from the analysis due to missing questionnaire data. Twenty-five participants who did not have a sufficient amount of trials to make an ERP, due to artifacts or poor performance, were also excluded from the analysis. Excluded participants did not significantly differ from included participants in age $t(68) = .76, p = .45$, sex $t(68) = -1.52, p = .13$, or BMI $t(68) = .24, p = .81$. Participants were recruited through undergraduate classes and earned course credit for their participation. This study received IRB approval from the University of New Orleans.

2.2 Measures

2.2.1 Binge-eating behavior. A binge-eating behavior composite score was generated for each participant by averaging the standardized scores for the Dutch Eating Behavior Questionnaire (DEBQ) and Emotional Eating Scale (EES) measures. These two measures were found to be highly correlated, $r(41) = .84, p < .001$. Subsequently, binge-eating behavior groups were generated by dichotomizing, via a mean split, the binge-eating behavior composite measure. One group was comprised of those whose scores fell within the upper half of the mean split (participants who scored high in binge-eating behaviors). The other group (Non-binge eaters) was comprised of those whose scores fell within the lower half of the mean split.

2.2.1a The Dutch Eating Behavior Questionnaire (DEBQ) is a measure of eating behavior (Van Strien et al., 1986). The DEBQ consists of three subscales: emotional eating, restraint, and

externality scales, for a total of 33 items answered on a 5-point Likert scale ranging from “never” to “very often”(Van Strien et al., 1986). Only the 13 items from the emotional eating subscale was used in the present investigation (see Table 1 for descriptive values). The emotional eating subscale addresses eating behavior in the context of emotion with questions such as, do you have the desire to eat when you are depressed or discouraged. The internal consistency for this measure was good ($\alpha = .93$).

2.2.1b The Emotional Eating Scale (EES) is a measure of emotion induced eating behavior (Arnou et al., 1995). The EES consists of 25 mood descriptions (e.g. bored or nervous) answered on a 5-point Likert scale ranging from “no desire to eat” to “an overwhelming urge to eat” (Arnou et al., 1995). After reading the mood description (e.g. irritated), participants indicate what their usual desire to eat level would be (e.g. an overwhelming urge to eat). See Table 1 for descriptive information. The internal consistency for this measure was good ($\alpha = .93$)

Table 1. Mean and standard deviations for scales assessing binge-eating behavior

	DEBQ	EES
	Mean (SD)	Mean (SD)
Overall values	2.20 (.82)	2.02 (.68)
High binge-eaters	2.93 (.46)	2.61 (.40)
Low binge-eaters	1.57 (.45)	1.49 (.36)

All scores are unstandardized values; DEBQ = The Dutch Eating Behavior Questionnaire; EES = The Emotional Eating Scale

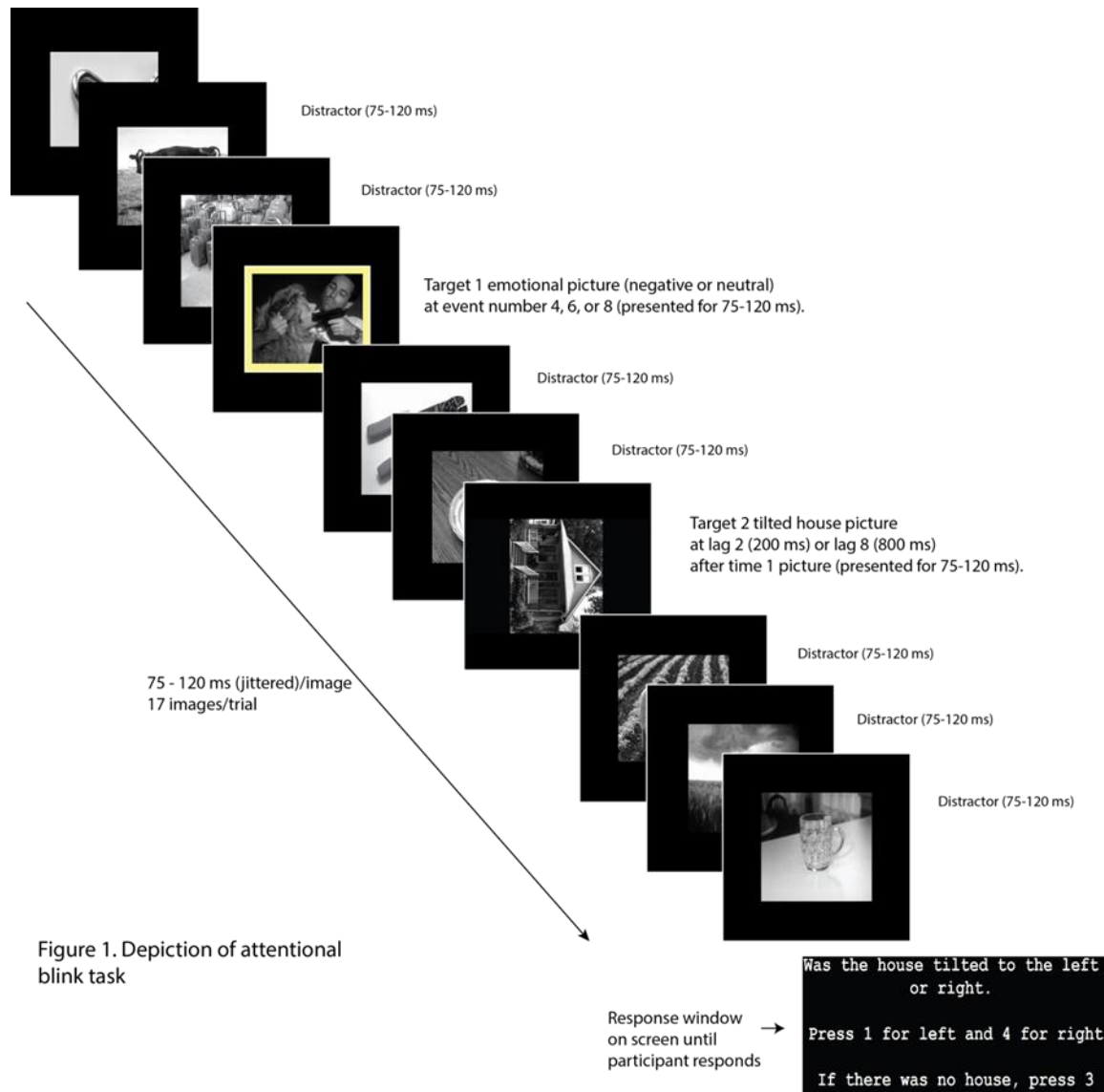
2.2.2 Body Mass Index (BMI). BMI is an indicator of body fatness (World Health Organization, 1995). BMI is calculated by dividing weight in pounds (lbs) by height in inches (in) squared and multiplying by a conversion factor of 703 to change units from metric (meters

and kilograms) to imperial (inches and pounds). Formula: weight (lbs) / [height (in)]² x 703. BMI does not measure body fat directly, but research has shown that BMI correlates to direct measures of body fat, such as underwater weighing and dual energy x-ray absorptiometry (Mei et al., 2002). Participant weight was measured using a standard analogue scale and height was measured using a sliding measuring device attached to the weight scale. Participants remained fully clothed during the acquisition of these measurements except for their shoes.

2.2.3 Task. Raymond and colleagues (1992) introduced the term attentional blink (AB), a psychological construct in which attention is momentarily inaccessible due to the processing of previous information. When two targets are to be identified among non-target distractors most individuals show an AB in reporting the second target. Correct identification of the first target (T1) impedes the detection of a second target (T2) that appears within 500 ms of T1 (Chun & Potter, 1995; Raymond et al, 1992). The failure to report a T2 is believed to happen because a large amount of attentional resources have been allocated to T1 (Shapiro et al., 2006). The attentional blink is believed to be induced when salient stimuli cause a focus of attention (Shapiro, Schmitz, Martens, Hommel, & Schnitzler, 2006). Moreover, Olivers and Nieuwenhuis, (2005) found that the size of an attentional blink is determined by an individual's psychological state and that a strong focus on T1 promotes the attentional blink. Shapiro and colleagues (2006) found that performance on T2 could be predicted from the amount of resources used in the processing of T1; more resources used for T1 equated to larger blink magnitude for T2. EEG and fMRI studies have shown reduced activity in the frontoparietal cortex when T2 cannot be reported, even though both targets activate early visual areas (Sergent, Baillet, & Dehaene, 2005; Krancioch, Debener, Schwarzbach, Goebel, & Engel, 2005; Williams, Visser, Cunnington, & Mattingley, 2008). However, these studies did not use emotionally salient images. Most, Chun,

Widders, and Zald (2005) found negative T1s to cause greater deficits in T2 processing than neutral T1s. However, the study was only behavioral and did not examine associated neural correlates. Furthermore, currently no studies have explored the link between attentional blinks and binge-eating behavior.

For the current task (Figure 1), participants began with a 10 trial practice session with instructions emphasizing that T2 will follow the picture with the yellow frame (T1). The task consisted of 4 blocks of 120 trials each. At the end of each trial, participants pressed either “1” for a house tilted left, “4” for a house tilted to the right or “3” if no house was seen. To prevent participants from looking at their hands to indicate the correct button, which would lead to EEG eye artifact, button 3 was marked by a large fuzzy sticker that could easily be identified by touch alone.



Between each block of the task, participants were instructed to stretch and blink their eyes in order to get comfortable and ready to proceed with the next block. Stimuli were black and white photographs: 120 T1 images framed in yellow (60 negative T1 images and 60 neutral T1 images), 200 neutral distractor images (each used randomly 9 times), and 100 T2 images (house photos, 50 tilted 90° to the left and 50 tilted 90° to the right) all presented on a 34 cm wide x 27 cm high LCD monitor. We also included 20 trials where no T2 image was presented to make missed T2 trials a true option and thereby prevent random responding when T2 was not observed. Emotional and neutral pictures were drawn from the International Affective Picture

System (IAPS; Lang, Bradley, & Cuthbert, 2008). House pictures were drawn from publicly available sources. Negative IAPS pictures were of people or animals and included graphic images of violence and mutilation. The neutral pictures were balanced with the negative pictures for numbers of depictions of people and animals. Trials consisted of a rapid serial visual presentation (RSVP) stream of 17 images, presented for 75-120 ms, and jittered trial-by-trial to aid in ERP processing. Depending on the trial, T1 was presented as the 4th, 6th, or 8th stimulus. T2 was presented either two or eight pictures after the T1 (lag 2 and lag 8). This task was adapted from a previous version (Most et al., 2005), but differed in many ways. The previous task used color photos throughout the task along with scrambled negative photos as a control. However, to avoid the possibility of participants reacting to color salience potential differences rather than the difference between neutral and negative images, we used black and white photos. Furthermore, our use of black and white rather than color photos rendered the use of scrambled photos as a control unnecessary. Also, the previous task used tilted landscape/architectural photos for T2. In order to make the T2 photos clearly different from the neutral distractor photos, we used only tilted house photos. Neutral distractor photos did not include any house images. Lastly, in order to avoid participants guessing house tilt direction for T2s that they did not see, we included 20 trials in each block in which no house was present at T2 and gave the button 3 option.

2.3 Procedure

After obtaining written consent, height and weight measurements were taken and questionnaires were completed. Participants completed the questionnaires in the EEG testing room. Questionnaires took an average of 35 minutes to complete. Following the completion of questionnaires, an electrode sensor net was applied to the participants' heads and participants sat

67cm from a computer monitor. Instructions on how to do the task were given and participants completed a practice block, identical to the main task, of 10 trials. When proficiency was shown on the practice block (on average it took only one practice block to show proficiency) participants went on to perform the actual task. On average, the task took 30 minutes to complete.

2.4 EEG data collection and analysis

EEG was recorded using a 128-channel Geodesic Sensor Net and sampled at 250 Hz, using EGI software (Net Station; Electrical Geodesic, Inc., Eugene, OR). Data acquisition was started after all impedances for all EEG channels were reduced to below 50 k Ω . All channels were referenced to Cz (channel 129) during recording and were later re-referenced against an average reference corrected for the polar average reference effect (PARE correction; Junghoefer, Elbert, Tucker, & Braun, 1999). Data was filtered using a FIR bandpass filter with a lowpass frequency of 50 Hz and a highpass frequency of .3 Hz. To best capture eye blink artifacts, the threshold was set to 140 μ V threshold (peak-to-peak) and all trials in which this threshold was violated were excluded from analyses. Furthermore, signal activation change (peak-to-peak) exceeding 100 μ V across the entire segment were marked as bad and interpolated. P2, N2, and P3 data were time-locked to the T2 and was baseline corrected to 400 ms before T2 onset. Because of the CNV latency range of -200 to 0 ms before T2, CNV data was time-locked to the T2 and was baseline corrected from 400 ms to 200 ms before T2 onset. Data from trials with correct T2 detection and trials in which T2 was erroneously not detected were analyzed for Lag 2 trials. Based on grand averaged data, a latency range of -200 to 0 ms before T2 was used to measure CNV activation, a range of 239-316 ms after onset of T2 was used to measure P2 activation, a range of 267-395 ms after onset of T2 was used to measure N2 activation, and a

range of 355-559 ms after onset of T2 was used to measure P3 activation. P2, N2, and P3 activation was measured as maximum or minimum activation within the previously outlined latency ranges. However, because of the drawn out nature of its waveform, CNV activation was measured as mean activation. All ERP and behavioral data that showed values greater or less than 2SD from the mean were modified to reflect exactly 2 SDs from the mean (outlier correction) thereby preventing statistical analyses from being skewed by outliers.

2.5 Data analyses

Analyses consisted of 4 mixed model repeated-measures ANOVAs computed separately for each ERP component (CNV, P2, N2, P3). Analyses were conducted separately for each ERP component rather than for each hypothesis to avoid redundancy of analysis. More specifically, the mixed model repeated-measures ANOVA can show results for all hypotheses (accuracy, emotion, and group) for one ERP component. However, to avoid confusion, all analyses below are marked by hypothesis number. In each model, BMI, sex, age, and trial count were added as covariates. BMI was included as a covariate due to literature indicating ERP differences for obese individuals compared to individuals of normal weight (Kamijo et al., 2012). Sex and age (18 to 36) were controlled because of documented activational differences for (Hodges & Gust, 1995). Furthermore, since the number of trials comprising an ERP can affect ERP amplitude, trial count was added as a covariate in all subsequent analyses (Lamm et al., 2014). Within each ANOVA binge eating group (Binge-eaters vs. Non-binge eaters) was used as a between subject factor, emotion (negative vs. neutral) and trial type (correct T2 detection vs. incorrect T2 detection) were within subject factors. Post hoc analyses were conducted using Emeans analyses with LSD correction in order to correct for family wise error.

Results

3.1 Accuracy

A 2 (emotion; negative vs. neutral) x 2 (lag; lag 2 vs. lag 8) x 2 (binge-eating group; bingers vs non-bingers) mixed model repeated-measures ANOVA was computed with age, sex, BMI, and trial count as covariates. Findings showed main effects for emotion [$F(1, 42) = 88.70$, $p < .001$] and lag [$F(1, 42) = 21.84$, $p < .001$]. A trend-level emotion x lag interaction also emerged [$F(1, 42) = 3.33$, $p = .08$]. Contrasts revealed reduced accuracy in neutral, compared to negative condition, for both lag 2 ($p < .001$) and lag 8 ($p < .001$) and reduced accuracy in lag 2 vs lag 8, for both neutral (p

$< .001$) and negative ($p = .01$) conditions (see Figure 2). Given that the attentional blink has been shown to appear between 200 and 500 ms after T1 (Raymond et al, 1992), the fact that we found

decreased accuracy for lag 2 trials compared to lag 8

trials indicates that the task was administered correctly.

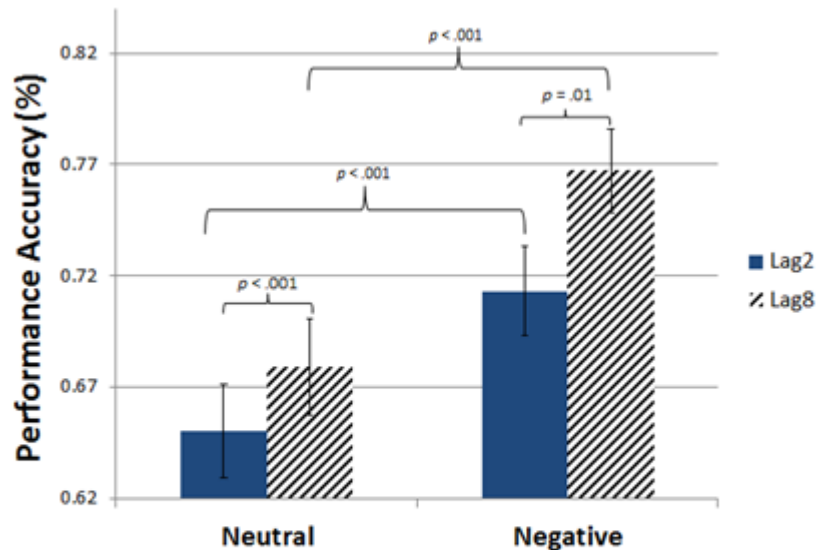


Figure 2: Bar graph showing reduced accuracy in Neutral vs. Negative condition in Lag 2 and Lag 8, and reduced accuracy in Lag 2 vs. Lag 8, in both Neutral and Negative conditions.

ERPs

3.2 Visualization

Visualization of correct T2 identification trial waveforms (CNV, P2, and N2 are shown in Figure 3; P3 is shown in Figure 4) showed clear P2, N2, and P3 components, while visualization of incorrect (blinked) T2 identification trial waveforms showed less distinct components. No clear CNV was observed for any of the conditions or groups.

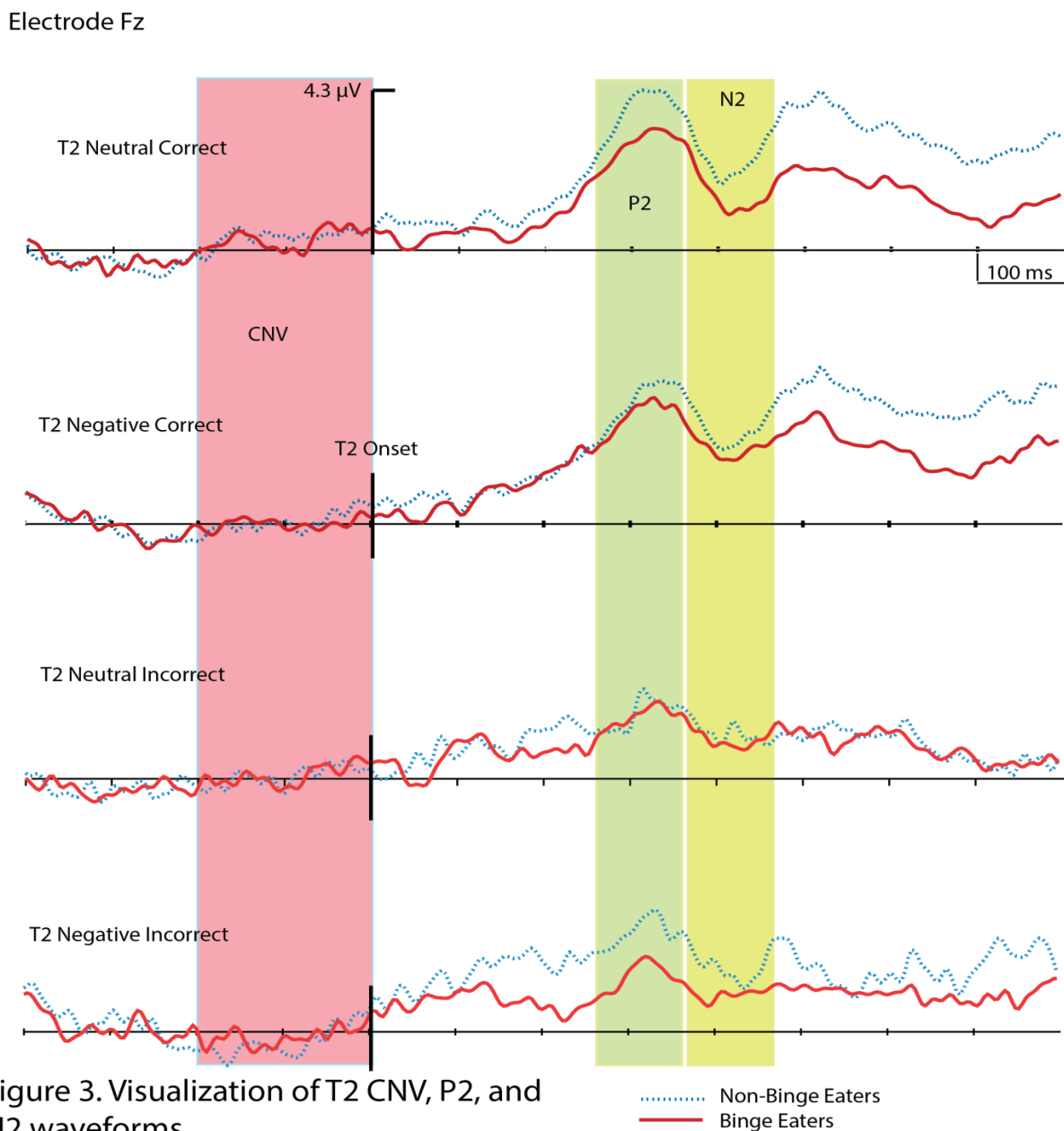


Figure 3. Visualization of T2 CNV, P2, and N2 waveforms

Electrode Pz

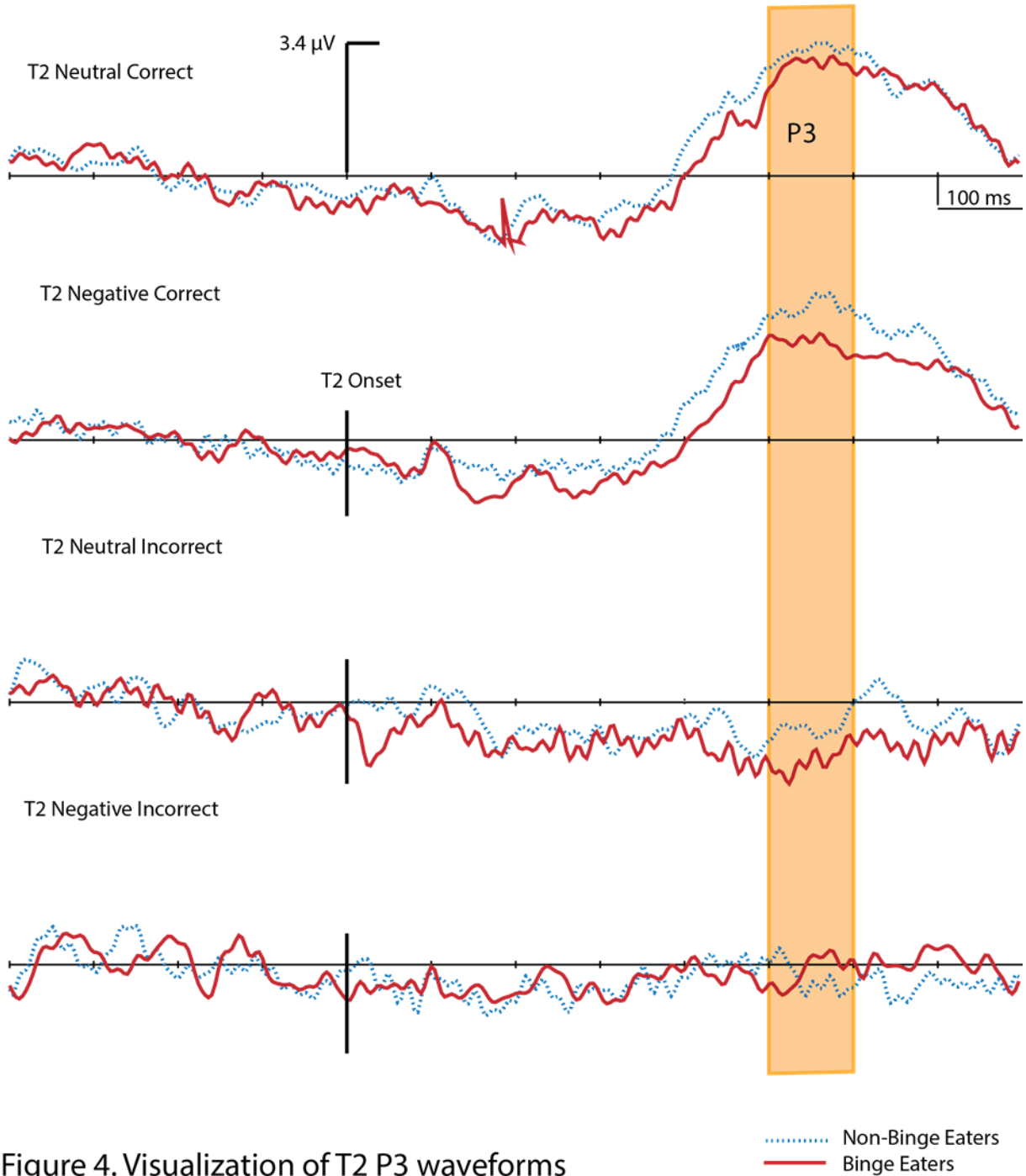


Figure 4. Visualization of T2 P3 waveforms

3.3 CNV data analysis

Based on literature indicating activation significance for the CNV at mediocentral electrodes (Smith et al., 2007; van Wouwe et al., 2011; Verleger et al., 2006), electrodes Fz and FCz were analyzed for each participant. A 2 (emotion; negative vs. neutral) x 2 (trial type; correct T2 detection vs. incorrect T2 detection) x 2 (binge-eating group; bingers vs non-bingers) mixed model repeated-measures ANOVA was computed with age, sex, BMI, and trial count as covariates. No statistically significant results emerged for CNV at either electrode site. Effect sizes ranged from $.000 \eta^2$ to $.085 \eta^2$ for electrode Fz and from $\eta^2 = .000$ to $\eta^2 = .065$ for electrode FCz, thus indicating that these non-significant findings were not exclusively due to low power brought about by the number of participants.

3.4 P2 data analysis

Based on literature indicating activation significance for the P2 at mediofrontal electrodes (Eichele et al., 2005; Kanske, Plitschka, & Kotz, 2011), electrodes Fz and FCz were analyzed for each participant. A 2 (emotion; negative vs. neutral) x 2 (trial type; correct T2 detection vs. incorrect T2 detection) x 2 (binge-eating group; bingers vs non-bingers) mixed model repeated-measures ANOVA was conducted with age, sex, BMI, and trial count as covariates.

Electrode FCz: An emotion x trial type interaction, at the level of a trend, was found [$F(1, 35) = 3.09, p = .09$]. Contrasts revealed that for incorrect T2 detection trials, when compared with correct T2 detection trials, P2 activation was reduced

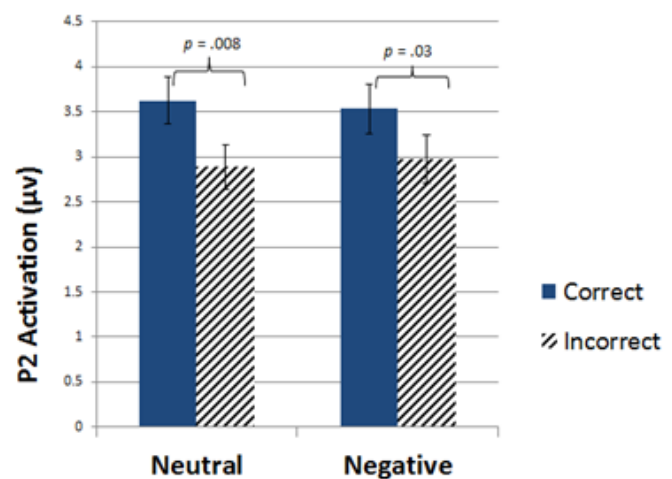


Figure 5: (electrode FCz) Bar graph showing reduced P2 activation for incorrect T2 detection trials vs. correct T2 detection trials, for both Negative and Neutral conditions.

for both negative ($p = .03$) and neutral ($p = .008$) conditions (hypothesis 1.17.1.b; see Figure 5). Thus, results showed decreased P2 activation for incorrect trials.

Electrode Fz: A two-way Emotion-by-Trial type interaction was found [$F(1, 35) = 5.32, p = .03$], which was subsumed by an emotion x trial type x binge-eating group three-way interaction [$F(1, 35) = 14.05, p = .001$]. Contrasts revealed for non-binge eaters a decrease in P2

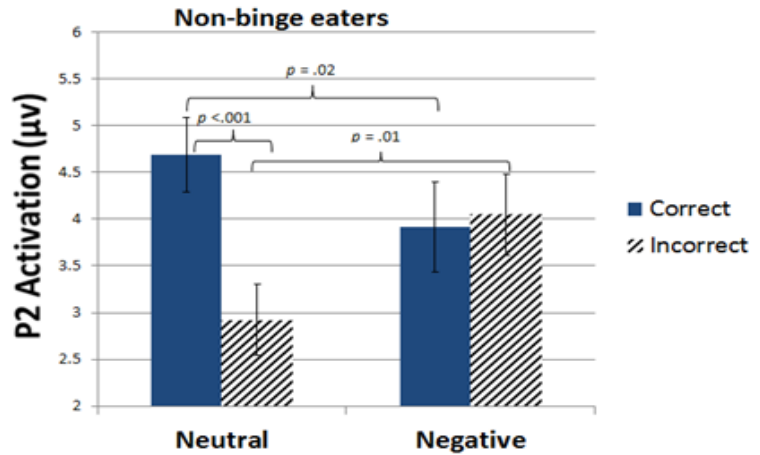


Figure 6: (electrode Fz) Bar graph showing, for Non-binge eaters, decreased P2 activation in the Negative vs. Neutral condition, for correct T2 detection trials, increased P2 activation in the Negative vs. Neutral condition, for incorrect T2 detection trials, and decreased P2 activation in the Neutral condition for incorrect T2 detection trials vs. correct T2 detection trials.

activation from the neutral to the negative condition ($p = .02$) for correct T2 detection trials and an increase in activation from the neutral to the negative condition ($p = .01$) for incorrect T2 detection trials (hypothesis 1.17.2.b; see Figure 6). Also, we found a difference between Trial types. For non-binge eaters in the neutral condition less P2 activation was found for incorrect T2

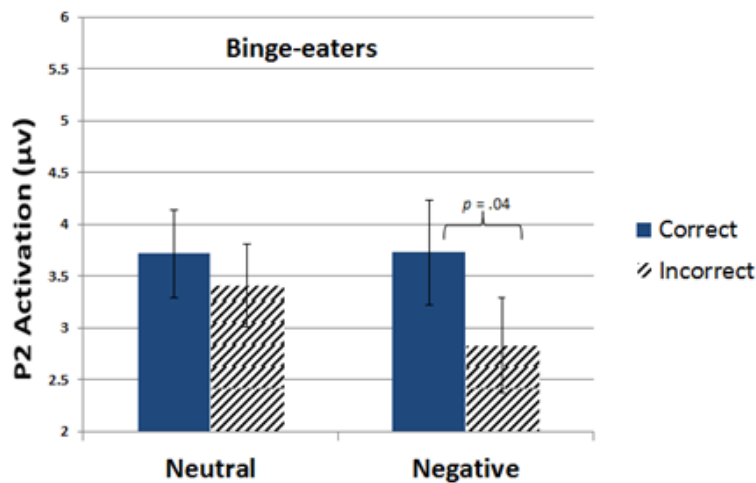


Figure 7: (electrode Fz) Bar graph showing, for Binge-eaters, decreased P2 activation in the negative condition for incorrect T2 detection trials vs. correct T2 detection trials.

detection trials than for correct T2 detection trials ($p < .001$; hypothesis 1.17.1.b; Figure 6).

However, for binge-eaters, in the negative condition P2 activation decreased during incorrect T2 detection trials when compared with correct T2 detection trials ($p = .04$; hypothesis 1.17.1.b; Figure 7).

Supporting our hypothesis, we found a trend level group difference for incorrect T2 detection trials in the negative condition; binge-eaters showed decreased P2 activation compared to non-

binge eaters ($p = .08$;

hypothesis 1.17.3.b; Figure 8).

Interestingly, we only found

the predicted decreased P2

activation for binge-eaters as

compared to non-binge eaters

in erroneous trials.

3.5 N2 data analysis

Based on literature

indicating activation significance for the N2 at mediofrontal electrodes (Lamm et al., 2013; Swainson et al., 2003), electrodes Fz and FCz were analyzed for each participant. A 2 (emotion; negative vs. neutral) x 2 (trial type; correct T2 detection vs. incorrect T2 detection) x 2 (binge-eating group; bingers vs non-bingers) mixed model repeated-measures ANOVA was conducted with age, sex, BMI, and trial count as covariates.

Electrode FCz: An emotion x trial type interaction was found [$F(1, 35) = 7.13, p = .01$].

Counter to our hypothesis, contrasts revealed increased (more negative) N2 activation for

incorrect T2 detection trials, when compared with correct N2 detection trials, for both negative

($p < .001$) and neutral ($p = .02$) conditions (hypothesis 1.17.1.c; see Figure 9).

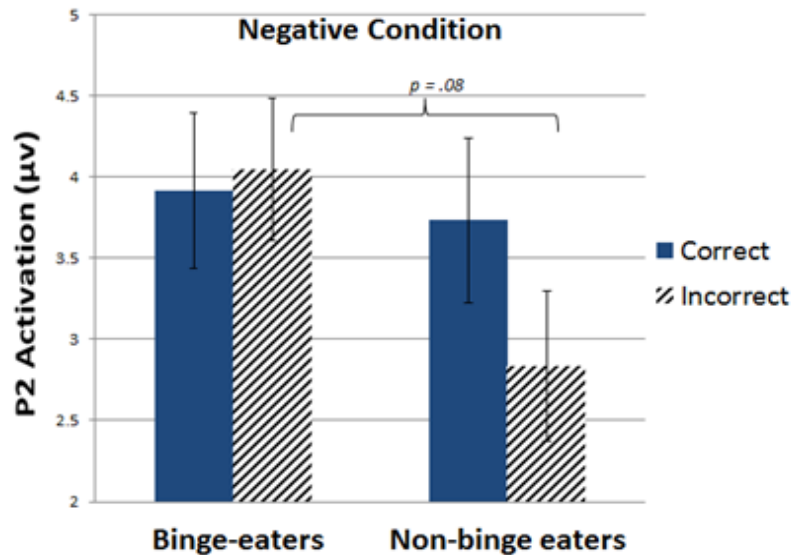


Figure 8: (electrode Fz) Bar graph showing decreased P2 activation, for Binge-eaters vs. Non-binge eaters, for incorrect T2 detection trials in the Negative condition.

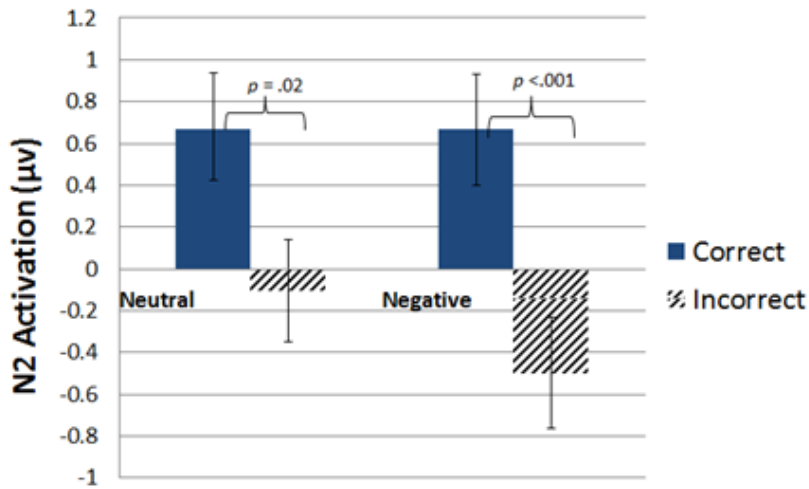


Figure 9: (electrode Fz) Bar graph showing increased (more negative) N2 activation, for incorrect T2 detection trials vs. correct T2 detection trials, for both Negative and Neutral conditions.

Electrode Fz: An Emotion-by-Trial type interaction was found [$F(1, 35) = 5.83, p = .021$] along with an emotion x binge-eating group interaction trend, [$F(1, 35) = 3.89, p = .057$] both of which were subsumed by an emotion x trial type x binge-eating

group three-way interaction [$F(1, 35) = 5.76, p = .02$]. Contrasts revealed three significant effects. 1) For non-binge eaters, increased N2 activation for incorrect T2 detection trials ($p = .003$) compared with correct N2 detection trials in the neutral condition (hypothesis 1.17.1.c; see Figure 10). 2) For binge-eaters, increased (more negative) N2 activation was found for incorrect T2 detection trials, when

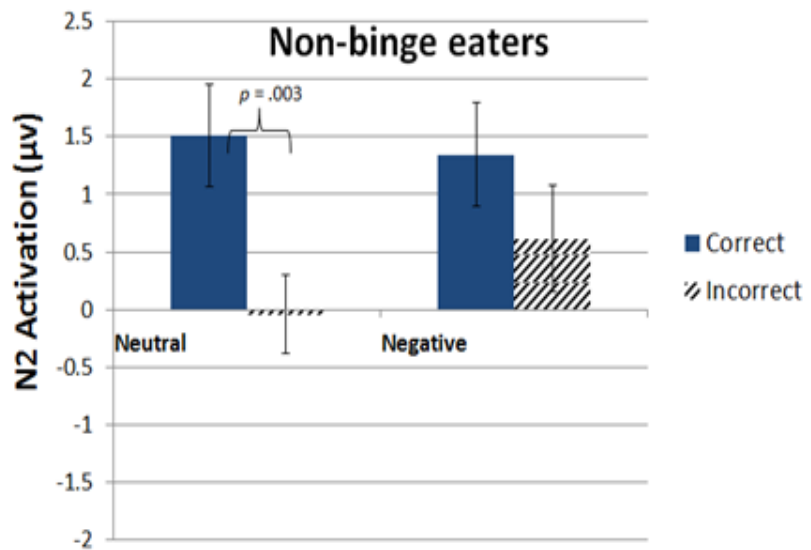


Figure 10: (electrode Fz) Bar graph showing increased N2 activation (more negative) for Non-binge eaters, for incorrect T2 detection trials vs. correct T2 detection trials, in the Neutral condition.

compared with correct T2 detection trials, for both neutral ($p = .04$) and negative ($p = .001$) conditions (hypothesis 1.17.1.c; see Figure 11). In line with our hypothesis, we found increased N2 activation in the negative condition compared to the neutral condition ($p = .009$; hypothesis

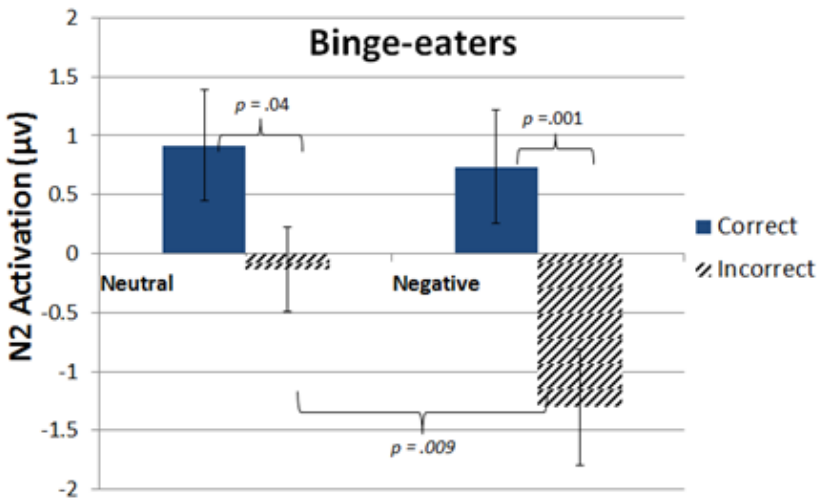


Figure 11: (electrode Fz) Bar graph showing increased N2 activation (more negative) for Binge-eaters, for incorrect T2 detection trials vs. correct T2 detection trials, for both Neutral and Negative conditions, and increased N2 activation in the Negative condition vs. the Neutral condition.

1.17.2.c; see Figure 11) for binge-eaters but not for non-binge eaters. We found this effect only for erroneous trials and not for correct trials. Also, contrary to our prediction, we found more N2 activation for binge-eaters than non-binge

1.17.3.c; see Figure 12); however, we only found this in negative trials when T2 was erroneously

not detected. Thus, results revealed that binge-eaters recruited more neural activation for more difficult (incorrect T2 detection) trials in the negative condition.

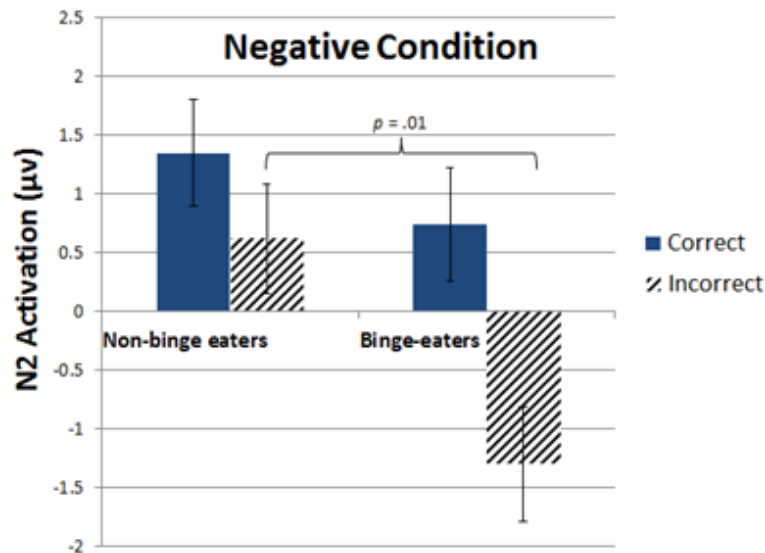


Figure 12: (electrode Fz) Bar graph showing increased activation (more negative) for Binge-eaters vs. Non-binge eaters, in the Negative condition, for incorrect T2 detection trials.

3.6 P3 data analysis

Based on literature indicating activation significance of Posterior midline electrodes (Sergent et al., 2005; Vogel et al., 1998), electrodes PCz and Pz were analyzed for each participant. A 2 (emotion; negative vs. neutral) x 2 (trial type; correct T2 detection vs. incorrect T2 detection) x 2 (binge-eating group; bingers vs non-bingers) mixed model repeated-measures ANOVA was conducted with age, sex, BMI, and trial count as covariates. No significant effects were found for electrode PCz.

Electrode Pz: A main effect was found for emotion [$F(1, 35) = 7.51, p = .01$] and an interaction was found between emotion and trial type [$F(1, 35) = 4.32, p = .05$]. Contrasts revealed increased P3 activation for correct T2 detection trials, when compared with incorrect T2

detection trials, for both the neutral ($p < .001$) and negative ($p < .001$) conditions (hypothesis 1.17.1.d). Also, increased activation was found in the negative condition, when compared with the neutral condition ($p < .001$), for incorrect T2

detection trials (hypothesis

1.17.2.d; see Figure 13). Therefore, while we did not find a group difference, we did find the predicted trial type effect supporting the validity of our paradigm.

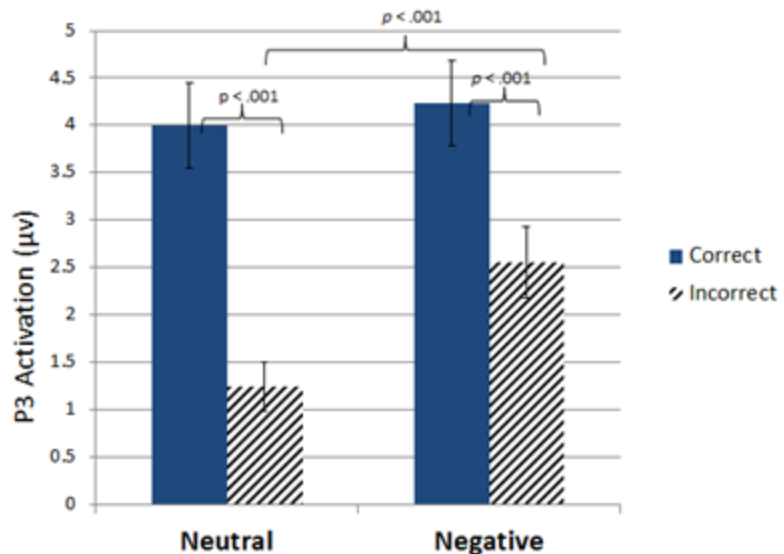


Figure 13: (electrode Pz) Bar graph showing increased P3 activation for correct T2 detection trials vs. incorrect T2 detection trials, for both the Neutral and the Negative conditions, and increased P3 activation in the Negative condition vs. the Neutral condition, for incorrect T2 detection trials.

4.0 Discussion

An attentional blink is believed to result from the large amount of attentional resources used to focus on particular stimuli, which interferes with attending to temporally close subsequent stimuli (Raymond et al., 1992). Furthermore, missed stimuli do not enter working memory and do not activate the P3 ERP component, a measure of stimuli entering working memory (Kranzloch et al., 2003). In line with these previous findings, we found significantly reduced accuracy for trials in which the second target (T2) was in close temporal proximity to the first target (T1) as compared to when T2 was temporally further away. Interestingly, and also in line with previous research, we found significantly more P3 activation for trials in which T2 was accurately detected as compared to when T2 was not accurately detected. Given that these findings are in line with previous attentional blink research, these results suggest that we affectively captured the neural activation underlying the attentional blink phenomena and can now move forward to apply this paradigm to the study of obesity and binge eating.

Obesity is a multifaceted problem which affects individuals as well as society as a whole (Falkner et al., 2001; Finkelstein et al., 2009; Flegal et al., 2012; Reilly et al., 2003). Binge-eating is one of many factors that affect the obesity problem (CDC, 2012). Attentional deficits have been found to contribute to binge-eating through cognitive narrowing (Heatherton et al., 1991). Additionally, emotion has also been implicated as a precursor to binge-eating behavior (Arnoult et al., 1995; Cattanach et al., 1988; Chua et al., 2004; Elmore et al., 1990) and binge-eating behavior has been associated with cognitive rumination in the context of negative events (Fairburn et al., 1995; Kubiak et al., 2008; Nolen-Hoeksema et al., 2007). Therefore, it may be that when binge-eaters are faced with negative events or emotions it leads them to cognitively narrow their focus of attention or ruminate about the negative events and thus preventing

subsequent events from entering their focus of attention. More specifically, because of this emotion-induced cognitive narrowing (attentional process), dietary restrictions may not enter attentional focus and thus contribute to unrestricted eating behavior.

The current study decomposes this attentional deficit by applying a frequently used model of attention, i.e., the Posner Model of attention, and using ERPs to characterize the sub-processes of attention outlined by the Posner Model. The Posner Model of attention outlines that 3 sub-processes of attention—alerting, orienting, and executive attention—can contribute to attentional deficits (Posner et al., 1971). In this study, we use three ERP components (CNV, P2, and N2) that have been associated with alerting, orienting, and executive control of attention (Carretié et al., 2001; Delplanque et al., 2004; Donkers et al., 2004; Kanske et al., 2011; van Wouwe et al., 2009; Woodman, 2010) to measure the neural activation underlying each of these sub-processes of attention, and then examine differential activation patterns for binge-eaters and non-binge eaters. Thus, the current study adds to the extant literature by examining the neuromechanistic chronometry underlying attentional deficits that contribute to binge-eating behavior.

The CNV component has been associated with alerting or cueing a subsequent event (Walter et al., 1964). Thus, it may reflect the alerting aspect of the Posner model of attention (first attentional sub-process). We did not find any group differences for the CNV. This was perhaps due to using negative stimuli, as opposed to food stimuli, for the T1 cue. Though both negative and food stimuli have been shown to affect attention, negative stimuli may not interfere with the cueing process to the extent that food stimuli would for binge-eaters. More specifically, for binge-eaters, a food stimulus attracts attention to a greater extent than negative stimuli. This

greater attentional focus on a food T1 could result in interference to the cueing process where no interference was observed for the negative stimuli.

The second attentional sub-process discussed in the Posner Model is orienting. P2 activation signifies the neural activation underlying attentional orientation toward stimuli (Huang et al., 2006) and thus may function as a marker of orienting as depicted in the Posner Model. Furthermore, orienting to stimuli in the context of emotional stimuli has been shown to require additional P2 activation (Carretié et al., 2001; Delplanque et al., 2004; Kanske et al., 2011). Subsequently, it may be that P2 activation is a measure of the neural resources used to orient away from some stimuli and towards other stimuli, and perhaps in the context of negatively-charged photos, one requires additional neural resources to effectively orient. In the current study, binge-eaters had reduced P2 activation (trend level) compared to non-binge eaters for erroneous T2 detection trials in the negative condition. Thus, in the presence of negative stimuli, binge-eaters may have not been able to recruit sufficient neural resources to effectively orient away from negative T1 events and towards subsequent stimuli. This ineffective orienting may thus have contributed to attentional blinking of the subsequent T2 event.. Given that binge eaters have been shown to have attentional narrowing (Heatherton et al., 1991), the presence of negative stimuli (e.g. a bad day at work) may cause sustained attentional focus (ineffective orienting) on the key components of the negative stimuli (e.g. rumination about the most troubling aspects of a bad day) and/or they may shift their attention toward food (used as an escape mechanism; Heatherton et al., 1991). Thus excessive attentional focusing on either of these two types of stimuli (negative or food), could result in depleted attentional resources thereby preventing effective attentional orienting away from these events. For binge-eaters,

inadequate attentional orienting may prevent thoughts about preplanned dietary restraint measures from interrupting food focused or ruminative behavior.

The third attentional sub-process within the Posner Model is executive control of attention. N2 activation has been found to reflect the neural response to cognitive conflicts occurring between incompatible streams of attentional processing, i.e. conflict monitoring (Van Veen & Carter, 2002). Thus, N2 activation may be a neural marker of executive control of attention as depicted in the Posner Model. N2 activation has also been shown to respond to emotional stimuli (Lamm et al., 2013). Therefore, in terms of our attentional blink task, N2 activation may represent a large amount of attentional resources focused on T1 (emotionally arousing stimulus) conflicting with the attentional resources required to process T2. Consistent with this argument, binge-eaters had significantly greater N2 activation (more negative), compared with non-binge eaters, in the negative trials when T2 was present but not detected. Binge-eaters were more affected by negative stimuli than non-bingers, which may have resulted in increased conflict between T1 and T2 stimuli and thus caused T2 to be missed. In terms of binge-eating behavior, as outlined above, negative stimuli (e.g. a bad day) could cause attention to be focused on the events of the day, or on food (i.e. as an escape mechanism), and thus cause a heightened attentional conflict between this food focused behavior and thoughts about their preplanned dietary restraint measures, and thus contribute to unrestrained eating behavior.

P3 activation is believed to indicate information processing within working memory, with increased activation found for detected targets and little or no P3 activation found for missed targets (Kranzloch et al., 2003; Ritter & Vaughn, 1969). More specifically, Kranzloch et al., (2003) found that in the context of the attentional blink paradigm, P3 activation may represent information making it into working memory. Based on this research, we expected increased T2

P3 activation for accurate trials than inaccurate trials (blinked trials) and this hypothesis was confirmed. We also expected differences between conditions and groups, neither of which were found. It may be that our negative T1s were not distracting enough for binge-eaters to prevent succeeding T2s from entering working memory. In future studies, using food images in place of negative images for T1 may result in more distraction and thus reductions in subsequent P3 activation for binge-eaters.

Our study utilized ERPs in order to examine if binge-eaters differed from non-binge eaters in the neural correlates underlying attentional sub-processes outlined in the Posner model of attention. Our findings suggest that binge-eaters do differ from non-binge eaters in terms of orientating and executive control of attention. Binge-eaters were found to have decreased P2 and increased (more negative) N2 activation for events that followed negative stimuli and contributed to erroneous responding. As indicated above, P2 activation has been associated with attentional orienting (Huang et al., 2006) and N2 activation has been associated with attentional conflict (Van Veen et al., 2002). Thus, this pattern of results might reflect a dual deficit in attentional processing for people who display binge-eating behavior. More specifically, binge-eating behavior may be brought about in part by first, an inadequate orienting toward new stimuli after exposure to a negative event and second, a heightened conflict between the first and second stimuli. In other words, inadequate orienting away from emotional events and towards subsequent events prevents appropriate attentional resources from being allocated to the second event thereby potentially causing elevated conflict between the first and second events. This dual processing deficit likely prevented adequate processing of a subsequent stimulus and thus contributed to the erroneous response. A real-world example of this dual processing deficit for binge-eaters might be that exposure to a negative event leads to attentional focus on the event

(dwelling on the event) or shifted attentional focus to food (in order to avoid thinking about the event). Focused attention upon either of these targets (negative events or food) could result in preventing effective attentional orientation to preplanned dietary restraint plans as well as increased conflict between thoughts about the events and thoughts of preplanned dietary restraints. Subsequently, these attentional deficiencies may be key contributing factors for binge-eating behavior.

5.0 Limitations

There are limitations to the current study. Firstly, the group differences in P2 and N2 activation showed opposite effects, i.e., binge eaters showed less P2 activation and more N2 activation compared to non-binge eaters. This pattern of effects could be generated by a number of factors including differences in baseline activation, which might undermine the meaningfulness of these results. Upon visual inspection of the grand-averaged waveforms, it is evident that this is not the case, i.e., no group differences are evident in the baseline activation. Thus, it is likely that this pattern of opposite ERP effects is due to patterns of underlying neural activation and not due to an artifact.

Secondly, some participants had relatively few trials comprising the ERP and trial count has been correlated with ERP amplitude (e.g., Lamm et al. 2014). To control for this problem, we entered trial count as a covariate. However, future research should include more trials to avoid this potential issue.

Thirdly, our sample of binge-eaters was most likely comprised of individuals who would not meet the clinical criteria for binge-eating. In the future this study should be replicated using a clinical sample of binge-eaters.

Lastly, in the current study, we are using a laboratory environment to emulate real world emotion-induced binge eating. While this is a useful approach to decomposing the attentional mechanisms contributing to binge-eating behavior, it is likely not as salient as real world emotions. Given that we cannot induce the level of emotional arousal, as might be realistic in the real world (e.g., breakup of a relationship or after a car accident), due to ethical considerations, we can only speculate that the attentional neuromechanistic chronometry revealed in the current study is similar to what might be expected for real world levels of emotional arousal.

6.0 Conclusion

This study found differences in cortical activation, believed to be associated with attentional deficits, between binge-eaters and no-binge eaters in the context of negative images. Binge-eaters showed deficiencies in allocating attention to stimuli that followed negative images. These results enhance our understanding of attentional deficits that may contribute to binge-eating behavior. Furthermore, elucidating these attentional deficits could be beneficial in terms of treatment and intervention, i.e., if the attentional blink is a precursor for binge-eating behavior, then targeting treatment with attention focused training, such as meditation, could curtail this behavior.

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