Neural Differences between Healthy Adolescents and Those At Risk for Psychological Disorders: An Analysis of P100 and P300 ERPs Using an Emotional Oddball Paradigm

by

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Abstract

Attention Deficit Hyperactivity Disorder (ADHD), Depression, Anxiety, and Conduct Disorder are Axis 1 Psychological Disorders that can affect adolescents' attention and emotion regulation. In this study, we aimed to compare attentional skills and emotional regulation between at-risk and healthy control groups using a modified version of the emotional oddball paradigm while collecting event-related potentials (ERPs). We hypothesized that significant differences in attention related ERPs, particularly in the P100 and P300, would be observed between the two groups.

We collected data from 33 participants in the Clinical group (recruited from CASA Child and Adolescent Services for All mental health services) (females: n=14, males: n=19). And, we included 17 participants in the Healthy Control group (females: n=11, males: n=6). We conducted a modified version of the emotional oddball paradigm, which was designed to observe both emotional and non-emotional responses. To do this, the paradigm included baseline images (scrambled pictures), infrequent distractors (sad, fearful, and neutral pictures of faces), and infrequent targets (circles). Participants were instructed to make a right-hand button press in response to targets and a left-hand button press to all other stimuli.

The behavioral data showed a significant effect of age on accuracy, where older participants were more accurate. Additionally, the amplitude of P100 in response to the distractors was significantly larger for the control group compared to the CASA group. This effect was observed at two electrode sites (POR and Oz). The results also showed significant effects of sex on the right side of the brain for the distractor stimuli (emotions), with males having larger P300 amplitudes. Furthermore, the CASA group showed longer P300 latencies for the target stimuli. As the P300 reflects top-down, or conscious attention processing, these

findings suggest that the CASA group is taking longer to consciously process attentional stimuli, whether because they need to devote more cognitive resources to the task, due to perhaps less efficient processes, or over-attenuating to specific stimuli.

Our study identified significant differences in neural functioning between healthy adolescents and those with Axis 1 Psychological Disorders, particularly at the P100 and P300 ERPs. Group differences at the P100 reflect differences in early attention processes, and group differences at the P300 reflect differences in later, top-down attentional processes. Effects of experimental group occurred mostly where sex was not significant, suggesting sex as a confounding variable. Future research should carefully match for sex and age when analyzing differences between healthy and clinical groups. Our findings suggest the potential usefulness of the emotional oddball paradigm and ERP in detecting and differentiating individuals with psychological disorders from healthy controls.

Preface

The research collected for this thesis was approved by the Health Research Ethics Board at the University of Alberta. The oddball paradigm used was adapted from Wang et al. (2005) and conducted as described in Shafer et al. (2012). All writing appearing here is my own, and all work was completed under the supervision of Dr. Anthony Singhal.

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Chapter 1:

General Introduction

Introduction

1.1 What is Attention?

A classical definition of attention goes back to William James: “the taking possession of the mind, in clear and vivid form, of one out of what may seem several simultaneously possible objects or trains of thought ... it implies withdrawal from some things in order to deal effectively with others” (James, 1890). The second half of this quote is referring to what is now thought of as Selective Attention. This type of attention is where a participant would have to focus on one thing out of many, such as focussing on studying in a busy hall. However, there are other types of attention to consider. For example, Sustained Attention, which is maintaining focus over a long period of time, such as paying close attention during a 3-hour lecture (Ko et al., 2017). Attention also relies on other types of cognitive processes such as perception, awareness, motivation, and emotion. Another way to conceptualize attention is in terms of complexity, task relevance, and volition. For example, attention functions to orient us to novel stimuli to prepare us for potential danger or action. This has often been considered to be an automatic, bottom-up process that works with low-level modality-specific perception. (Graziano & Webb, 2015; Katsuki & Constantinidis, 2013). Another set of attentional processes likely rely on voluntary control. In this case, we can choose what to focus on. This is a more conscious process that is top-down and under attentional control. (Katsuki & Constantinidis, 2013). These two types of attention involve different neural networks (Vossel et al., 2014), and likely interact during complex behaviour. It has been suggested that lower-level, bottom-up attentional processes are mediated by a ventral frontoparietal network, and top-down voluntary attentional processes are mediated by a dorsal frontoparietal network. More recently, research has focussed on how both the ventral and dorsal systems work together to support behaviour (Vossel et al., 2014; Katsuki & Constantinidis,

2013). It has been suggested that bottom-up attentional processes operate on a fast timescale, whereas top-down processes are slower to manifest and may take longer to complete (Delorme et al., 2004).

Attention has also been conceptualized as having components that are sensory-specific, and others that are multi-modal (Bomba & Singhal, 2010). One aspect of attention that is of interest to the present study is how it interacts with other processes such as emotion and motivation. It is not difficult to imagine how stimuli with a strong emotional context could draw attention. It has been argued that emotional stimuli act as natural targets of attention to inform behavioural states such as approach-to desired stimuli and withdrawal-from dangerous or unpleasant stimuli (Heller et al., 2004). One of the main purposes of the present study was to further understand this relationship between attention and emotion.

In the present study we collected event-related potentials (ERPs) as measures of attentional process while participants performed a visual attention task that had emotional and non-emotional stimuli. In this study the two ERPs of interest were the P100 and the P300. The P100 is a positive deflection typically occurring between 80 and 200 ms post-stimulus onset and has been shown to be an early marker of attentional processes strongly involving the visual cortex (Clark et al., 1995). Moreover, the P100 has been shown to be modulated by fearful emotion (Eimer & Holmes, 2002; Smith et al., 2003). The P300 is a large positive waveform that has its largest peak in the range of 300-500 ms post-stimulus onset over midline central and parietal electrode sites (Sutton et al., 1965). It is typically observed when attention is directed to a stimulus train which has both frequent and infrequent stimuli. It has been shown that the peak-latency of P300 increases if the categorization of a target stimulus becomes more difficult, which suggests that it also reflects low-level processes (Kutas et al., 1977; Coles et al., 1995). There is

strong evidence that the amplitude of P300 reflects the intensity of processing (Donchin et al., 1986) as well as perceptual-central resources (Donchin et al., 1986a; Kramer & Spinks, 1991) within a multiple capacity framework (Wickens,1984; Singhal & Fowler, 2004; 2005). Importantly, P300 has been found to be larger in response to emotional stimuli (Carretie et al., 2004).

1.2 What is Emotion?

Prevailing theories suggest that emotions evolved to protect ourselves (i.e. fear) and reproduce (i.e. love) (Hammond, 2006). There are strong social benefits related to emotion, and this has been the subject of much theory and research in Psychology and Neuroscience. There has also been debate about the relationship between psychological and physiological factors of emotion. For example, the James-Lange theory argues that physiological responses impact psychological feelings, whereas the Cannon-Bard theory argues the opposite, that the psychological thoughts or feelings must come first (Cannon, 1987). Not surprisingly, another theory argues that the psychological and physiological are interconnected (Marsh et al., 2019).

Emotional stimuli are often categorized in terms of two continua: valence and arousal (Lang et al., 2005). Valence refers to an emotional spectrum that ranges from pleasant to unpleasant, and arousal refers to an emotional spectrum that ranges from calm to excited (Lang et al., 2005). Valence and Arousal are generally considered separate phenomena that operate orthogonally to each other and are supported by different brain circuitry (Heller, et al., 2004).

1.3 Attention & Emotion Interactions

It has been strongly argued that emotion can both impair and enhance cognition (Chan & Singhal, 2013; Dolcos et al., 2011). It has been established that emotional stimuli capture our attention more easily (Dolcos et al., 2011), perhaps because of their previous importance for

survival and propagation. In many instances this is likely beneficial, however the inability to block-out negative emotional information can be detrimental to behaviour (Wang et al., 2006). It has been suggested that an inability to properly control negative effects of emotion may underlie certain mood disorders (Dolcos et al., 2011; Shafer et al, 2012). It has also been suggested that in cases of depression and anxiety there may be a reduced ability to regulate responses to emotional information, even when that information is not relevant. This further implies that the attention system may not be working efficiently to control responses in these situations (Oliveira et al., 2013). This is related to the argument that emotional stimuli may vie for the same resources as required by both the top-down and bottom-up attentional systems (Hartikainen, 2021).

As described by Dolcos et al., (2011) the traditional view of emotional stimuli processing posits that it is largely automatic, likely due to its relevance for survival. This view supports the role of the amygdala as a primary brain structure associated with emotional processing, and suggests that once an emotional stimulus is present, it must be automatically attended to (Dolcos et al., 2011). However, Pessoa et al. (2002; 2005) suggested a non-traditional view of attention and emotion processing, whereby emotional stimuli compete for attention along with all other stimuli. This view suggests that emotional stimuli require attentional resources. Shafer et al. (2012) found that different types of emotional stimuli, for example negative vs. positive emotional stimuli, have different effects on the brain. Therefore, when different emotional distractors were analyzed individually, they found an effect of emotion by load interaction. At low levels of load, the brain was more susceptible to being distracted by emotional stimuli, likely due to more cognitive resources being available (Shafer et al., 2012). This suggests that these two theories are not necessarily mutually exclusive, and may be situationally dependant. There is further evidence that the brain regions involved in emotional processing (e.g. amygdala) and

regions involved in attention (e.g. lateral and medial PFC) uniquely interact depending on task-relevance of the emotional stimuli (Dolcos et al., 2011).

As briefly described above, EEG is inexpensive, non-invasive, and thus an extremely good option in most neuroimaging studies; it may also be better tolerated for young children (Hajcak et al., 2010). EEG reflects the electrical signals of the brain, which we call ERPs. Different ERPs reflect different neural processes, and we will be focussing on the P100 and P300 ERPs. The main ERPs of the P100 and the P300 are expected when emotionally aroused (Olaffson et al., 2009). The P100 and P300 are commonly associated with emotional tasks, or focusing one's attention on fearful, sad, or otherwise emotive stimuli (Lewis et al., 2006; Dolcos & Cabeza, 2002; Koenig & Mecklinger, 2008). Thus, the waveforms of importance that we focussed on include the P100 and the P300.

An ERP study by Lewis et al. (2006) studied the neurological underpinnings of emotional regulation in children and adolescents. In this study, negative emotion was induced by temporarily losing "points" children had acquired during the experimental task. For the adolescents, the P300 waveform was significantly larger in amplitude following the induction of negative emotions which suggests a heightened attentional state or more effortful processing of negative stimuli (Lewis et al., 2006).

Another study by Shackman et al. (2007) used ERP to examine the effects of physical abuse on children's ability to regulate attention. Early trauma is known to influence brain development (Shackman et al., 2007), but the precise effects on emotion regulation and attention are not well parsed out. The results showed that in abused children, angry voices elicited a larger P300, and the children over-attended to both the task relevant and task-irrelevant anger cues (Shackman et al., 2007). Further, children who have been abused develop a broader boundary for

anger categorization (Pollak & Kistler, 2002). This may be because they devote more attention to negative stimuli, and may also be why anxiety is highly reported in these individuals.

In a critical review of the literature by Philips et al. (2008), they describe emotional regulation, saying: “emotion regulation consists of intrinsic and extrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish one's goals.” This definition describes not only the complexity of emotion regulation, but also the many systems and brain regions involved.

1.3.1 P100 & Emotion

ERP studies have determined that there is an "emotion effect," where emotional stimuli elicit stronger positive ERPs than do neutral stimuli (Dolcos & Cabeza, 2002). In participants with Affective Disorders, this effect could be even more pronounced. The emotion effect has been found specifically in the P100 and P300 waveforms (Cuthbert et al., 2000). As such, there is a known understanding of emotional regulation playing a role in the processing and attention that produce these ERP waveforms. A 2020 review by Schindler and Bublatzky examined the ERP research on attention to faces, and what modulates these emotion effects. Frequently used in scientific studies due to their applicability to the ‘real world,’ tasks involving faces or pictures of faces have been used extensively. In sum, they found larger responses in the P100 for fearful and angry faces. And, they found that threat-related faces were associated with stronger P100 responses than happy faces (Schindler & Bublatzky, 2020). Using a similar task in a healthy group of university aged participants, Muller-Bardoff et al. (2018) found that the P100 responses to emotional face images was independent of task load and was more affected by arousal level than valence, which means that they found increased P100 responses in response to both the positive and negative emotional valence images (Muller-Bardoff et al., 2018).

Carretie et al. (2004) also used ERP to show how attention is captured more strongly by negatively emotionally salient pictures. Following these images, they found an increased P100 amplitude, suggesting that more resources are being dedicated (Carretie et al., 2004). Secondly, regarding the behavioural data, the reaction times show that negative emotional stimuli capture attention *before* neutral stimuli (Carretie et al., 2004).

Lastly, in an EEG study by Pourtois et al (2013), they showed that the evoked potentials for fearful faces evoked a larger response at about 130ms, or in the range of the P100. This is further evidence that responses to emotional stimuli are well established and seen across the board in a variety of research models in the P100 and P300 (Liddell at al., 2004; Krolak-Salmon et al., 2001; Pourtois et al., 2013). This study utilized fearful faces as their stimuli, which is similar to the stimuli we used. Thus, we would expect similar results.

1.3.2 P300 & Emotion

P300 is a mid-latency waveform typically elicited using the oddball paradigm (Olafsson et al., 2008); these responses indicate top-down attention and attentional memory activity. The P300 ERP waveform has been shown to have an elevated positivity in response to emotional stimuli such as that used in the oddball paradigm (Cuthbert et al., 2000). The magnitude of the P300 in ERP data is affected by emotional processing, or level of arousal, which suggests these factors influence attention (Delplanque et al., 2004; Sabatinelli 2007). The P300 shows similar results across many studies, with larger amplitudes in response to fearful faces, and sometimes angry and happy faces (Schindler & Bublatzky, 2020).

Delplanque et al. (2004) showed interesting results with the relationship between emotion and cognition. Using a visual oddball task like that in the present study, they recorded ERPs to evaluate whether emotional stimuli interfered with the cognitive task. Emotionally salient stimuli

are known to frequently elicit larger P300 (Delplanque et al., 2004), which is what they saw in their study. An integrative review by this same group (Hajcak et al., 2010) summarized the ERP surrounding the P300. Their review showed that emotional stimuli capture attention and significantly increase the amplitude of the P300. These data suggest that emotional stimuli receive a prolonged increase in attention, and thus receive additional processing resources that could distract attention away from other task-relevant stimuli (Hajcak et al., 2010).

Emotionality of images such as faces has been shown to significantly affect brain waveforms such as the P300. Pourtois mentions that several studies have seen the P300 influenced by emotion (Poutrois et al., 2013). Lastly, one further study by Rossi & Pourtois (2014) elucidated more precisely that not all emotional stimuli have similar effects.

In developmental cognitive neuroscience, there have been studies examining attentional phenomena. Outside of the neuroscience research, developmental psychology has something to say on the causes of these age differences in attention and emotional regulation. A study by Somerville (2013) suggests that peer relationships become increasingly more important in adolescents, perhaps causing the new emphasis placed on emotional stimuli. Understanding how your peers are evaluating you, and how one 'fits in' becomes more and more valuable at this age. This could be driving the "overemphasis" we see placed on emotional stimuli, and suggests that social evaluation could be the mechanism for these brain differences. In adolescents with Anxiety, Depression, or other Affective Disorders, they may overestimate the social evaluation that peers are placing on them. Although it is difficult to say whether brain development differences drive changes in social evaluation, or vice versa, adolescents show significant differences compared to adults and children (Somerville, 2013). Age-linear patterns, those that increase or decrease with age, are visible (such as those surrounding the P300 amplitude), as well

as those that include adolescent-emergent patterns (such as sensitivity to emotional stimuli), where they emerge or peak in adolescence (Somerville, 2013). These behavioural science findings can complement neuroscience imaging and help explain some of the age-related differences we see. Many parts of the brain are still maturing in adolescence, and can vary greatly between an 11-year-old and 17-year-old adolescent, as well as males and females of the same age, in part, due to the influence of pubertal hormones on neurotransmitters (Ernst, Romeo, & Anderson, 2009; Nelson et al., 2005; Sisk & Zehr, 2005; as in Somerville, 2013).

The Philadelphia Neurodevelopmental Cohort was also examined for sex differences in the brain and behaviour (Gur & Gur, 2016). The database of almost ten thousand 8 to 21-year-olds was used to elucidate sex differences on neurocognitive tests. Sex differences show higher accuracy in females on attention tests, and these effects emerge after age 11 (Gur & Gur, 2016), aligning with the beginning of our age cohort. All age- and sex-related differences start to appear after age 11 and flatten after age 18, indicating that our age population will show significant variation both in age and sex. Further, the authors explain that not only are age and sex important variables for adolescent brain development, but so is the individual themself (Luna et al., 2004; as in Gur & Gur 2016). Interestingly, they note that this within-individual variability is higher for adolescents with developmental disorders such as ADHD (Gur & Gur, 2016).

Thai et al. (2016) also studied the neural markers of attention in children aged 9-16. They found that the N200 and P200 waveforms were significantly correlated with attentional bias and social anxiety, respectively. The N200 reflects early attention, and the P200, like the P300, can reflect the amount of neural resources dedicated to stimuli. The authors suggest these findings of a larger P200 amplitude in social anxiety suggests increased use of attentional resources, which may act as a compensatory mechanism that attenuates social anxiety in children.

Wauthia & Rossignol, in their 2016 review, saw that children showed longer P100 latencies than adults on the same task (Batty & Taylor, 2006). Their task was similar to ours, as participants viewed a variety of negative and neutral emotional faces. They also explained how the P300 follows a slow developmental course in childhood (Davies et al., 2004). Their review included studies (of 9- and 10-year-olds) with significant age effects (Jonkman et al., 2003), just as we would expect in our study. This review data shows that the P300 response does not begin to develop until around 10 years of age, which could be a significant confound in our study involving 11-17 year olds. This developmental process, and the fact that it begins so late, could explain the difficulty that children and youth have regulating attention and emotions, especially those who also have ADHD, anxiety, depression, or conduct disorder.

Using an oddball task similar to ours, where 1st year university students viewed angry and fearful faces as distractors, while looking for oddball "targets," Rossignol et al. (2012) found a main effect of emotion, such that angry and fearful faces were detected slower. The P100 showed an effect of group such that those with anxiety scores had increased waveforms. The P300 also showed similar effects, but for emotion. These effects of group and emotion on P100 and P300 amplitude, respectively, showed that the neural patterns of attention are impacted by these variables. Precisely, an increased amplitude means that more neural resources are being devoted to processing negative emotional stimuli. Most importantly, although this study was done in older teenagers, they showed similar trends in the effect of emotion on the P100 and P300 ERPs.

Zhao et al. (2023) also used an oddball paradigm and studied the relationship between ERPs and the risk for suicide in adolescents. They found that adolescents more prone to suicidal tendencies showed a larger P300 amplitude. Interestingly, they found brain lateralization effects in the LPP (the Late Positive Potential, occurring after the P300) and N250 (Negative 250, a

negative ERP occurring at approximately 250 ms) ERPs, but not in the P300 (Zhao et al., 2023), where we expect to see lateralization effects along with the P100.

An early childhood study in participants aged 3-4 looks at these mechanisms of emotional regulation. In Rothbart et al.'s (2011) study, they found that infants' ability to disengage attention was correlated with lower amounts of negative emotion, highlighting the connections between the attentional and emotional regulation pathways (Rothbart et al., 2011).

Besides differences seen in age, there are important differences seen in sex, particularly in child and adolescent research (Barry et al., 2022). Sex difference research in children is sparse, and particularly so in ERP data. Thus, in an individually matched study on 8-13 year olds, Barry et al. (2022) evaluated sex differences in children using EEG. Much adult research has shown sex differences in the visual oddball, with larger female amplitudes and longer male latencies for the P300 (Conroy & Polich, 2007), or larger P100 amplitudes in response to emotional faces in females compared to males (Pfabigan et al., 2014). Barry et al. (2022), however, studied children, and used an Auditory Go/NoGo task and showed that females had greater accuracy than males, and the response inhibition processing in females was significantly slower. The later ERPs, including the P300, showed larger ERP amplitudes in females than males, indicating a female advantage in conducting this task (Barry et al., 2022). Behavioural performance was also significantly better in girls with fewer errors and faster reaction time.

Overall, emotional regulation is seen to improve with age (Ahmed et al., 2005). A review by Dickey et al. (2021) suggests a developmental pattern of a broad decrease in emotional reactivity from adolescence onwards. Blakemore (2008), explains that there is a small amount of research on adolescent developmental changes in emotion and social development. However, some research shows significant differences in face processing between children in early puberty

(10, 11, 12 years of age), and those in late puberty (16, 17 years of age), leading to questions about the differences in brain development prompting this (Blakemore, 2008). Structural differences are often seen, but their translation to behavioural reactions can be difficult to explain.

1.3.3 Comorbidities

A review by Ollendick et al. (2008) also discusses the risk of comorbidity, including anxiety disorders, depressive disorders, ADHD, and oppositional defiant disorder. As their review is done on youth, this population is representative of our population. As they state, for the most part, "comorbidity is the rule, not the exception," and clinical studies need to attend to these variables in regard to treatments and outcomes (Ollendick et al., 2008). When viewed as a moderator variable, or a variable that can influence the strength or direction of the relationship between treatment and outcome, comorbidities can have a significant effect on what treatments "work" or are "evidence based."

Ladoceur (2012) suggests the importance of age in affective disorders, and that certain adverse events, when presented at the vulnerable time of adolescence, could lead to the development of anxiety and mood disorders. Neuroimaging research suggests that sex steroids can also influence the connections prefrontal cortical and subcortical limbic regions that regulate emotions (Ladoceur, 2012). They also suggest that heightened emotional reactivity in adolescents could contribute to an imbalance, which, especially in vulnerable youth, could start a cascade towards the development of an affective disorder.

1.4 The Present Study

The main purpose of the present study is to examine the nature of attention and emotion interactions in a population of high-risk youth, primarily suffering from affective disorders,

compared to matched healthy controls. To do so, we used an emotional oddball paradigm to examine responses to emotional stimuli, non-emotional stimuli, and the relationship between the two. In this task, participants viewed emotional pictures interleaved with non-emotional distractors, and non-emotional rarely occurring targets. Our goal was to examine behavioral responses as well as the P100 (early attention) and P300 (late attention) ERP waveforms in response to the pictures and the targets. We hypothesized that the clinical group would be less accurate and slower on this task compared to controls. Null differences on the behavioural measures could mean either that the differences in neural functioning between the clinical and control groups are not large enough to be seen outside of EEG, or, that there are other compensatory mechanisms being employed by the clinical group.

Furthermore, we expected that both groups would differ on the two ERP waveforms in response to both emotional stimuli and targets. One possibility is that the clinical group would elicit larger P100 and P300 waves during the emotional stimulus presentation suggesting a stronger emotional response compared to controls, followed by smaller or later peaking waveforms in response to non-emotional targets. This type of pattern might suggest the initial emotional reactivity has a negative effect on the attentional mechanisms required for non-emotional task performance in the clinical group compared to controls. Finally, in the P100 we expected healthy controls to show higher amplitude and longer latency in the distractor conditions. In the target conditions, we also expected the healthy controls to show a larger amplitude.

Regarding P300, we expected the clinical group to show longer latency, and larger amplitude, suggesting that more of their neural resources are being devoted to emotion and attentional processing than for the controls.

For both groups, we expected to see some effects of age and gender, in which we expected younger participants (and males) to perform worse, as indicated by a longer latency and response times. Lastly, we expected to see an effect of experimental condition, such that responses will differ for both groups for the negative emotional images compared to the neutral distractors.

Chapter 2:

Methods

Methods

2.1 Participants

Participants were adolescents aged 11 to 17, recruited from the Edmonton area in Alberta, Canada. The mean age for males, in both the control and CASA group, was approximately 1 year younger than the mean age for females. (CASA Group: Females 15.47, n=14, Males 14.65 years, n=19; Control Group: Females 15.11, n=11, Males 14.2 years, n=6). Most participants were right-handed, with only 2 left-handed participants (self-reported handedness data). All participants were recruited from the Edmonton area, with the adolescent clinical group participants from a local mental health treatment facility, in Edmonton, Alberta, called CASA, which stands for Child and Adolescent Services for All. Participants were not randomly selected, but recruited based on the nature of their diagnoses in order to have roughly equal age- and sex-matching between the CASA and control groups. Informed consent was received both from the adolescents as well as parental guardians. Those in the CASA group had clinical diagnoses of a variety of Axis 1 Disorders, previously diagnosed using the then-current DSM-IV. The most common diagnoses included: anxiety and depressive disorders, conduct disorder, oppositional defiant disorder, PTSD (Post-Traumatic Stress Disorder), and ADHD (not listed in order). Many participants had co-morbid disorders, with many adolescents also on medications. All participants had normal or corrected-to-normal vision.

The data used in this study was collected from 33 participants in the clinical CASA group and 17 participants in the control group. Original sample sizes included additional participants, with some of these individuals removed from analysis because of extremely noisy ERP data, incomplete or incorrect responses to trials, or otherwise unusable data (e.g. overly delayed responses). All trials that were removed had been independently analyzed by three researchers

for inter-rater reliability, as well as run through a computer analysis program with defined amplitude and latency cut-offs for the P100 and P300 waveforms. Due to the nature of the multivariate analysis used in the methods, participants were only included if they had usable trials in all of the analyzed electrode categories (ex. a participant's electrode must have clean data for amplitude and latency for all experimental conditions being tested). Further examples of exclusion were participants whose reaction time was either unnaturally early, or extremely late and thus outside the analysis window. Participants who made incorrect responses had those trials removed. Data was screened for outliers, and no participants were removed as outliers in the initial analysis.

2.2 Experimental Procedures

2.2.1 Task

The task used in this study was an emotional "oddball task." The basis of an oddball task is that most stimuli are the same, although occasionally, a different type of stimuli appears. This infrequent, different stimulus is then called the "oddball," which is what participants were told to pay attention to. Like most oddball tasks, the paradigm we used consisted of frequent "baseline" stimuli, and infrequent "oddball" targets. The participant was directed to respond differently to the oddball targets, which tests the participant's levels of attention. Our task varied slightly from other oddball tasks in that it also uses visual emotional stimuli as distractors. The paradigm had been adapted from Wang et al.'s 2005 paper, and was conducted as described below and in the Singhal et al. 2012 paper. The infrequent emotional distractors included three different types of distractors: sad, fearful, and neutral emotional stimuli (i.e. a picture of a woman's face displaying sadness). As this oddball task was a visual task, participants viewed images that fell into the three categories: baseline stimuli, oddball targets, or the emotional distractors. The baseline stimuli

were simply scrambled pictures, and were provided as filler between the emotional distractors and targets in order to prevent spillover effects, ensuring that participants' emotional responses did not affect consecutive trials. The emotional distractors were sad, neutral, or fearful pictures of people (ex. a picture of a sad woman). The images were retrieved from the International Affective Picture System (IAPS) for validity and standardization of emotional classification. The oddball targets, which were a picture of a circle, were more sophisticatedly grouped, based on the emotional distractor they followed. Labelled as "target after sad," "target after neutral," "target after fearful," or "target-after-target," the oddball targets were sorted based on the type of emotional stimuli preceding them. Due to the nature of our CASA group's affective disorders, the response of interest is how the emotional distractors modify the response to the target, hence this grouping format. Secondly, we were interested in how responses to emotional distractors differs from the response to neutral distractors. Positive emotional pictures were also included, but only to provide a baseline of the level of typical emotional response for each participant. The positive trials were only used in behavioural analysis and not ERP analysis due to a small number of clean trials. An example sequence of the task is displayed below, with an image of a sad woman (an infrequent negative distractor), a scrambled image (a frequent baseline stimuli), and an image of a circle (the target). This particular target is labelled as a "target-after-negative" target type, because it follows an image of a sad woman, which is a negative emotion.

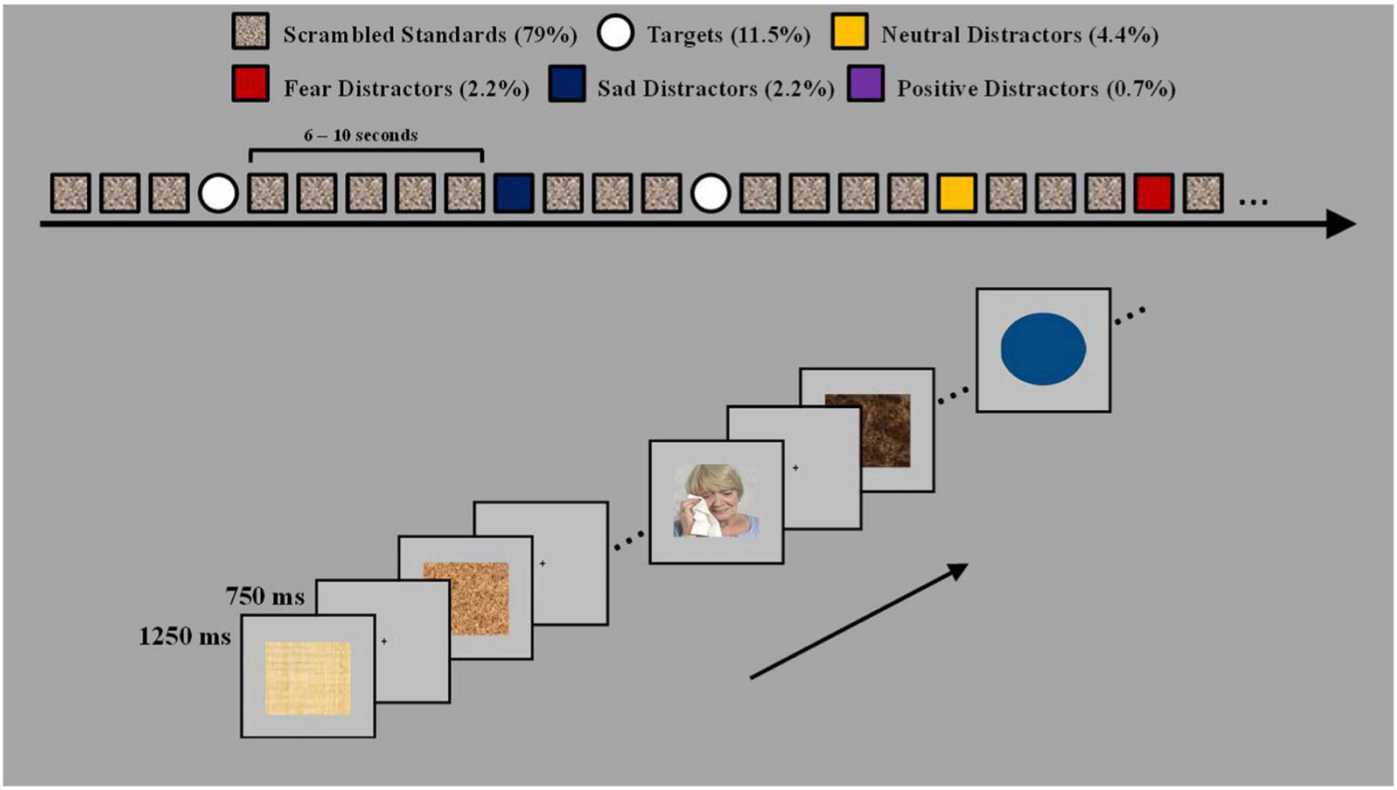


Figure 1

Example Task Sequence. Example task sequence diagram (Singhal et al., 2012). Upper part of image displays frequency of scrambled baseline images (79%), distractors (4.4% Negative Distractors, 4.4% Neutral Distractors), and target images (11.5%). Note example image of "sad woman" as taken from IAPS (International Affective Picture System). Other emotional images include fearful, angry, neutral, and happy images of faces from the IAPS.

2.2.2 Procedures

Conducting the task itself involved two actions for participants: they would press a button with their left hand when they viewed distractor stimuli (which consisted of the emotional distractors, whether positive, neutral, sad, or fear), and press another button with their right hand when viewing the target stimulus. Further, all participants were told to respond as quickly as

possible while remaining accurate. Reaction time and accuracy data was recorded for both groups, to exclude incorrect trials, as well as those with abnormally fast or slow reaction time. Behavioural data was also used to compare any significant differences between accuracy or reaction time in control and patient groups. EEG data was also recorded for both groups, specifically to compare the P100 and P300 waveforms' amplitude and latency between the control and CASA groups.

For all participants, the oddball task consisted of one run of 24 trials, and four runs of 25 trials. Each trial began with the presentation of the stimulus: scrambled picture distractor, emotional distractor, or target (a circle). The stimulus was presented for 750 ms, followed by a fixation screen for 1250 ms, with a spacing of 2 seconds between trials (from the onset of the first stimuli to onset of next stimuli). Importantly, the negative emotional distractor oddball trials were pseudo-randomized, in order to avoid affecting the participants' mood states. This meant that no more than two trials of the same emotional state were presented consecutively. Lastly, participants were informed they could "experience any feelings and thoughts the pictures might trigger" (Singhal et al., 2012). As this task is testing responses following emotional stimuli, any emotions felt by participants are pertinent.

2.3 Event-Related Potential (ERP) Recordings

Event Related Potentials (ERPs) were recorded with a high-density 256-channel Geodesic Sensor Net EEG (Electrical Geodesics Inc., Eugene, OR). ERPs were amplified at a gain of 1000 and recorded at a sampling rate of 250 Hz [Impedance <50KOhms and initially referenced to the vertex electrode (Cz)] (Singhal et al., 2012). Data was also baseline corrected (-300 to 0ms) and adjusted for eye-movement artifacts using an ocular artifact algorithm (Gratton et al., 1983). The events of interest, the P100 and the P300, were used to determine

segments of data to analyze. For the P100 analysis, the segments analyzed were between 50 and 200 ms. For the P300 analysis, the segments between 250 and 500 ms were analyzed for the largest peak in the specified time period. Various studies of the P300 use different time windows ranging from 300 to 600ms (as reported in Hajcak et al., 2010). Research from Foti and Hajcak (2009), however, suggest that the P300 is strongest around 350ms, within the time window that we used.

For each participant that was included, the individual waveforms were also visually inspected to ensure that the components of interest were clearly visible and in the proper response time range. This means that the P100 and P300 waveforms were notable enough to be visible to the naked eye, and were independently cross-checked with multiple researchers (three independent researchers in most cases). Waveforms were identified at electrode sites known to display maximal amplitudes for the P100 and P300, which included the Oz, Pz, P3, P4, PO3, and PO4 electrodes (Singhal et al., 2012) (see Figure 2). Since data were collected with a high-density 256-channel electrode net, we included three nearby electrodes for components of interest. Thus, the P100 analysis included the Oz, PO3, and PO4 electrodes, and the P300 analysis the Pz, P3, and P4 electrodes. The Oz and Pz electrodes are midline electrodes (Occipital Midline and Parietal Midline, respectively). P3 is also known as Parietal Left or PL, and P4 is known as Parietal Right or PR. PO3 is also known as Parietal-Occipital Left or POL, and PO4 is also known as Parietal-Occipital Right or POR. These naming conventions will be used throughout instead of the more complex numerical system of the 256-electrode map (see Figure 2). These electrode names are simplified for the sake of discussion as the older 10-20 EEG mapping system has less electrode locations than the high-density 256-electrode net used in our study (see Figure 2). When mapped on to the 256-electrode naming system, we see the Oz

electrode as electrode 137, and the Pz electrode as electrode 101. Electrode P3 is equivalent to electrode 86, and electrode P4 equivalent to electrode 162. Finally, electrode PO3 is mapped to electrode 109.

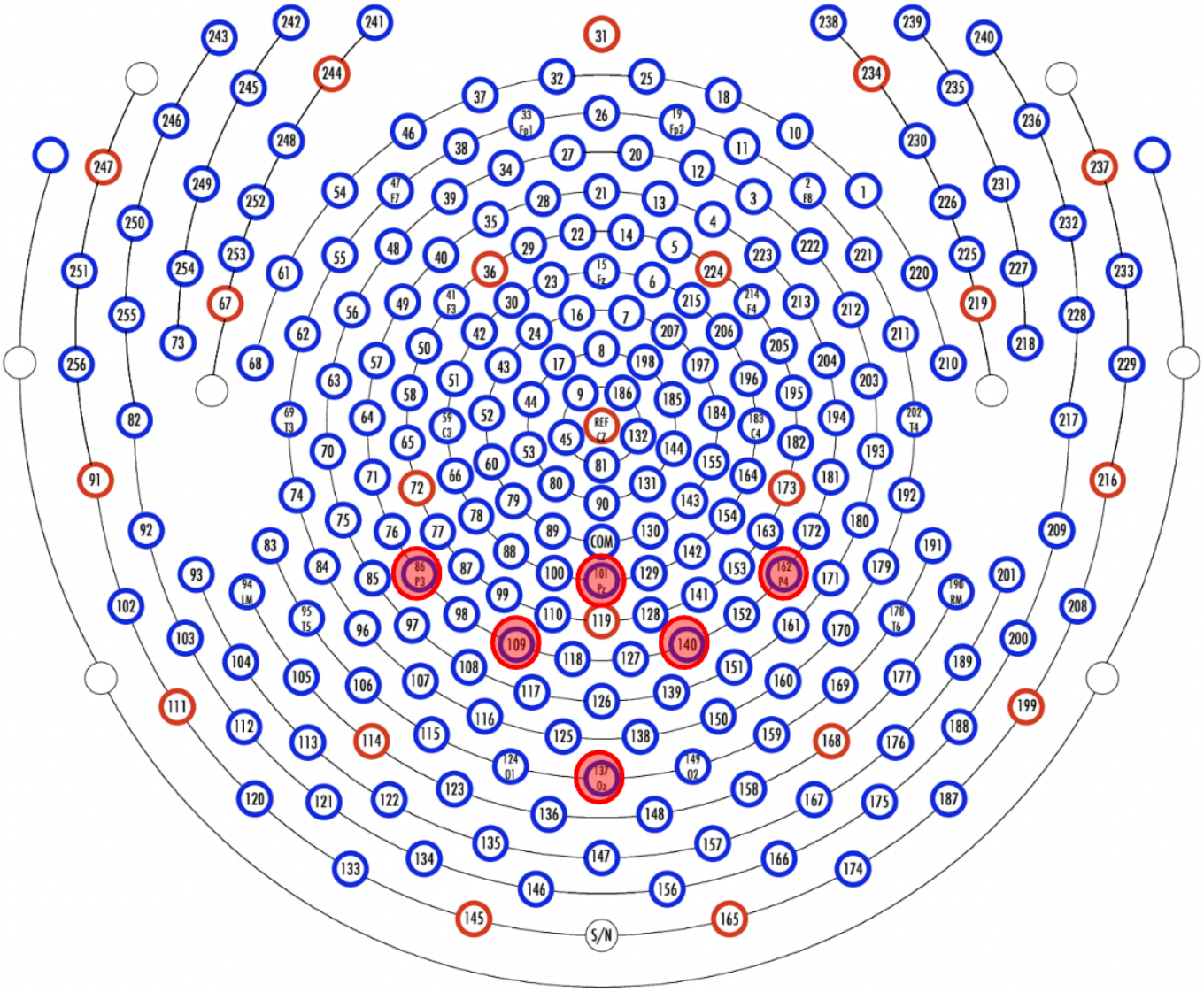


Figure 2

An Image of the 256-Electrode Map with Highlighted Electrodes of Interest. Electrode names are simplified for ease of discussion. When mapped on to the 256-electrode naming system, the Oz electrode is electrode 137, and Pz is electrode 101. Electrode P3 (PL) is electrode 86, and P4

(PR) is electrode 162. Finally, electrode PO3 (POL) is mapped onto electrode 109 in the 256-electrode system, and PO4 (POR) maps onto electrode 140 (Luu & Ferree, 2005). Electrodes of interest are highlighted with red circles. Adapted from Electrical Geodesics Inc. (Eugene, OR). 109 in the 256-electrode system, and PO4 maps onto electrode 140 (Luu & Ferree, 2005) (see Figure 2). Time windows for the P100 and P300 were determined from visual inspection and assessing common patterns in regard to early or late peaks. Again, at least 3 researchers independently analyzed the ERPs.

P100 waveforms were originally filtered through beginning at 100ms, however upon manual inspection of the ERP data, P100 waveforms beginning at 50ms were included. Similarly, upon visual inspection of the data, P300 windows were adjusted to include P300 peaks beginning at 250ms. Because our primary goal was to study emotional dysregulation and its effects on cognition in a clinical population, we compared the ERP results during the emotional oddball tasks of those in the clinical population with those in the control group. Specifically, we compared the amplitude and latency of the P100 and P300 ERP peaks through statistical analysis and a grand averaging of the ERP results.

2.4 Statistical Analyses

Clinical (CASA) and control groups' responses to the emotional oddball task were compared. Reaction time data, accuracy, as well as ERP data for the P100 and P300 were compared. In analyses of the ERP data, maximum amplitude was used for the P100 and P300. Windows of 50-200ms were used for the P100 and a window of 250-500ms was used for the P300. The maximum peak in the window was used as the P100 or P300 value in analyses. Trials were removed from analyses with extreme noise, as determined with cross-checked independent analysis between three researchers. If binning for "good" and "bad" waveforms was not

consistent among researchers, ERP data was re-analyzed. If there was still no consensus, trials were removed from analysis.

In the behavioural analysis, distractor type data was taken and analyzed for reaction time and accuracy effects between the CASA and control groups. Participants' trials were excluded if reaction time was less than 175ms or more than 2000 ms, or if the trials were completed incorrectly. Error rates were not seen to differ between control and CASA groups. Data was also analyzed across target type and compared between the CASA control groups for reaction time and accuracy differences. Age and sex were also analyzed for significant effects. This resulted in four separate analyses: Distractors Reaction Time, Distractors Accuracy, Targets Reaction Time, and Targets Accuracy. Analyses were assessed for effects of age, sex, condition, and group.

In statistical analyses of ERP data, the between-subject variable was "group," such that the CASA and control groups' data were compared against each other. The within-subject factor of "condition" was also tested, such that participants' responses to different conditions were compared, including the negative and neutral distractor stimuli (the negative distractor condition included a combination of the fear and sad distractor stimuli), as well as target-after-target and target-after-neutral conditions. Further, age and sex were included as variables in the analysis, and were removed from analysis when they were shown not to have a significant effect. If age or sex showed no significant effect, they were removed from the analysis which was then run again. Reaction time and accuracy of the CASA group and control group were also tested, for both the distractors and target conditions separately.

Although many neuroscience studies use ANOVA analyses, likely due to the smaller sample sizes seen in neuroimaging research, we chose to conduct MANCOVA analyses. MANCOVA analyses reduce Type 1 error, and most importantly, account for correlation between

variables, but need enough data points to have sufficient power. Due to the interconnectivity between different areas of the brain, and potential correlation between our different electrodes of analysis, this was the deciding factor in choosing MANCOVA.

Ideally, again due to the interconnectivity of brain regions, and thus correlations between data from different electrode locations involved in the P100 analysis, the Oz, PO3, and PO4 would all be included in the same analysis. Similarly, for the P300, it would have been ideal to include the Pz, P3, and P4 in the same multivariate analysis. However, due to missing values and a small sample size, we were unable to complete our multivariate analysis in this fashion with enough power. Because participants were only included in analysis if they had clean ERPs for every electrode condition (i.e. no missing values), we chose to separate the analyses by electrode to reduce the issue of missing values and increase power. Separating by electrode meant that most participants now had clean ERP values for every experimental condition, which increased power and sample size for this analysis. P100 and P300 were analyzed separately, further separated into electrode location, resulting in 6 separate analyses. Because the P100 analysis was separated into 3 individual electrode analyses, and similarly for the P300, our new p-value for statistical significance was set at $p=0.017$ ($p=0.05 / 3$). However, borderline-significant results up to $p=0.05$ were still included in this paper for the sake of discussion. Sex was included in the analysis when significant, and otherwise removed. Age was included as a covariate and was also removed where not significant. Power was maintained by doing separate multivariate analyses to reduce the number of outcome variables, and by lowering the significance level accordingly.

This resulted in a 2 x 2 design where sex (male / female) and group (Control / CASA) were the independent variables. Dependent variables were amplitude and latency, and because we had two types of stimuli, or two conditions, in each analysis (“Negative” (fear and sad) and

"Neutral" image types), this was a repeated-measures MANCOVA. (Outcome variables included in Pz analysis: Pz Amplitude NEG Distractor, Pz Amplitude NEUTRAL Distractor, Pz Latency NEG Distractor, Pz Latency NEUTRAL Distractor). Univariate analyses were still considered as a method of analysis and were tested in some cases, normally where results had borderline significance. When ANOVA results are reported, they are specified as such.

"Distractors" analysis was completed comparing the neutral and "negative" distractors as the within-subject variable. The "negative" distractor stimuli included the fearful and sad emotional images, and the neutral distractor stimuli included only the neutral distractors as described (no scrambled baseline images or positive distractors were included). Maintaining power was the main reason for combining the "fear" and "sad" distractors into one larger "Negative" category, which then had more data points. The "Targets" analyses was conducted with the "target-after-target" and "target-after-neutral" stimuli as the within-subject variables. This means that the participants' response to targets following an initial target stimulus were compared against their response to targets following a neutral or negative stimulus. Again, the between-groups variable was CASA vs. control group, and age and sex were included as covariates when significant. Final analyses, including all participants that had ERP data for each stimuli type and did not have any missing values, included 33 participants from the CASA group, and 17 participants from the control group. Note that precise number of females and males, as well as control and CASA participants, varied slightly between electrode analyses. Again, this is because some participants would have clean data for some electrodes and not others, and were only included in analyses were they had no missing values. For example, in a P300 analysis of target-after-target vs. target-after-neutral, if a participant had no clean data for the target-after-target stimuli type, they would not be included. This is to ensure that both data groups being

compared were the same size. Post-hoc comparisons were done where necessary, and all significant and borderline significant p-values are reported below.

Chapter 3:

Results

Results

3.1 Behavioural Data

Regarding the behavioural data comparing CASA and control groups, we had two separate analyses: “Distractors” and “Targets.” The distractor analysis compared reaction time and accuracy across all different distractor types and the baseline standards (Fear, Neutral, Positive, and Sad Distractors, as well as the Scrambled “Standards,” or baseline). The target analysis compared reaction time and accuracy across all different target types and the baseline standards (Target-After-Fear, Target-After-Neutral, Target-After-Sad, Target-After-Target, in addition to the Scrambled “standards”). Age and sex were considered as confounding factors, and experimental group was analyzed. ANCOVA analyses were conducted on behavioural data.

3.1.1 Behavioural Analysis of Distractors

In the analysis of reaction time, there were no significant differences noted between groups ($F(9, 294) = 2.633$, $p=0.162$). There were also no significant effects of age or sex. In the analysis of accuracy for the distractors stimuli, sex was a significant predictor of participants accuracy ($F(9, 288) = 1.279$, $p=0.015$) such that female participants performed more accurately (see Figure 4). This correlates with the behavioural analysis effects of age, with older participants being more accurate, as the females were on average 1 year older than males in both experimental groups. There was no effect of group.

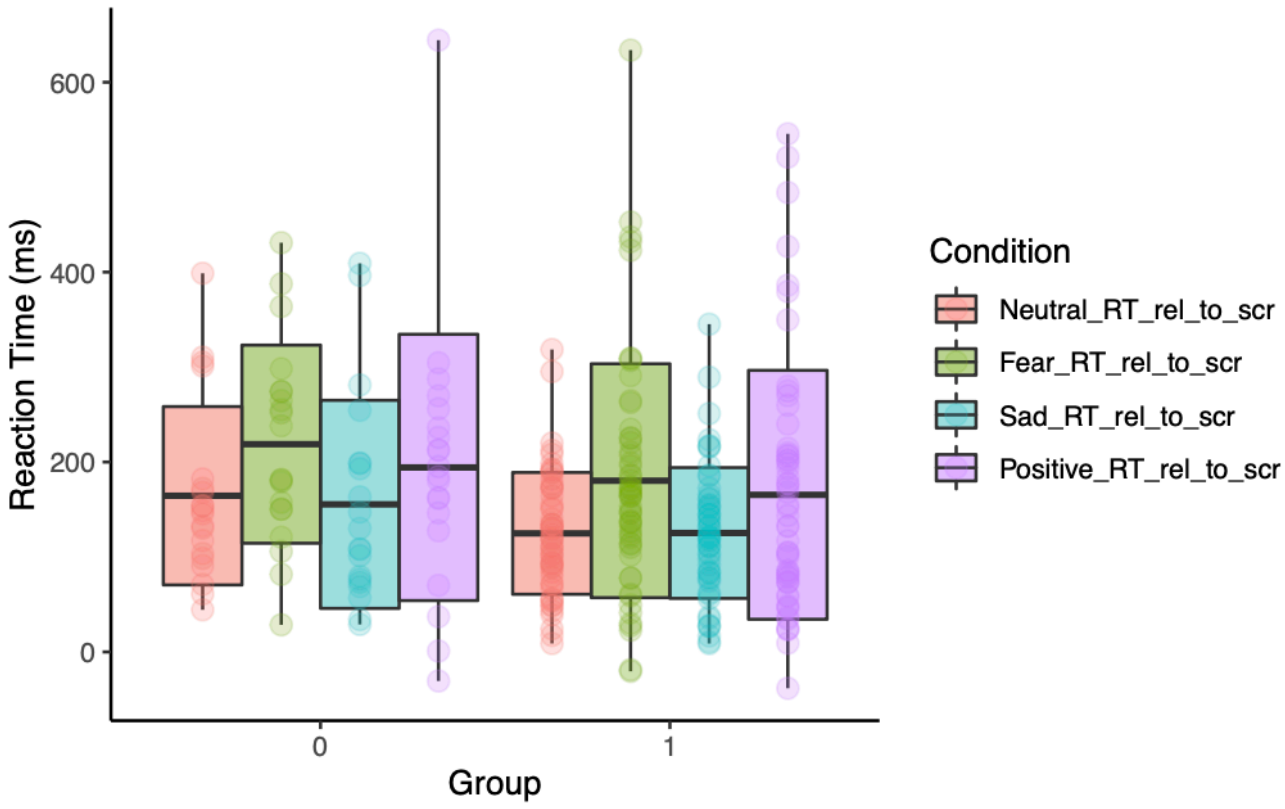


Figure 3

Reaction Time to Distractors. Error bars plot of the effects of Reaction Time on Group and Condition. No effects of group were seen. Controls are reference group 0, CASA is group 1.

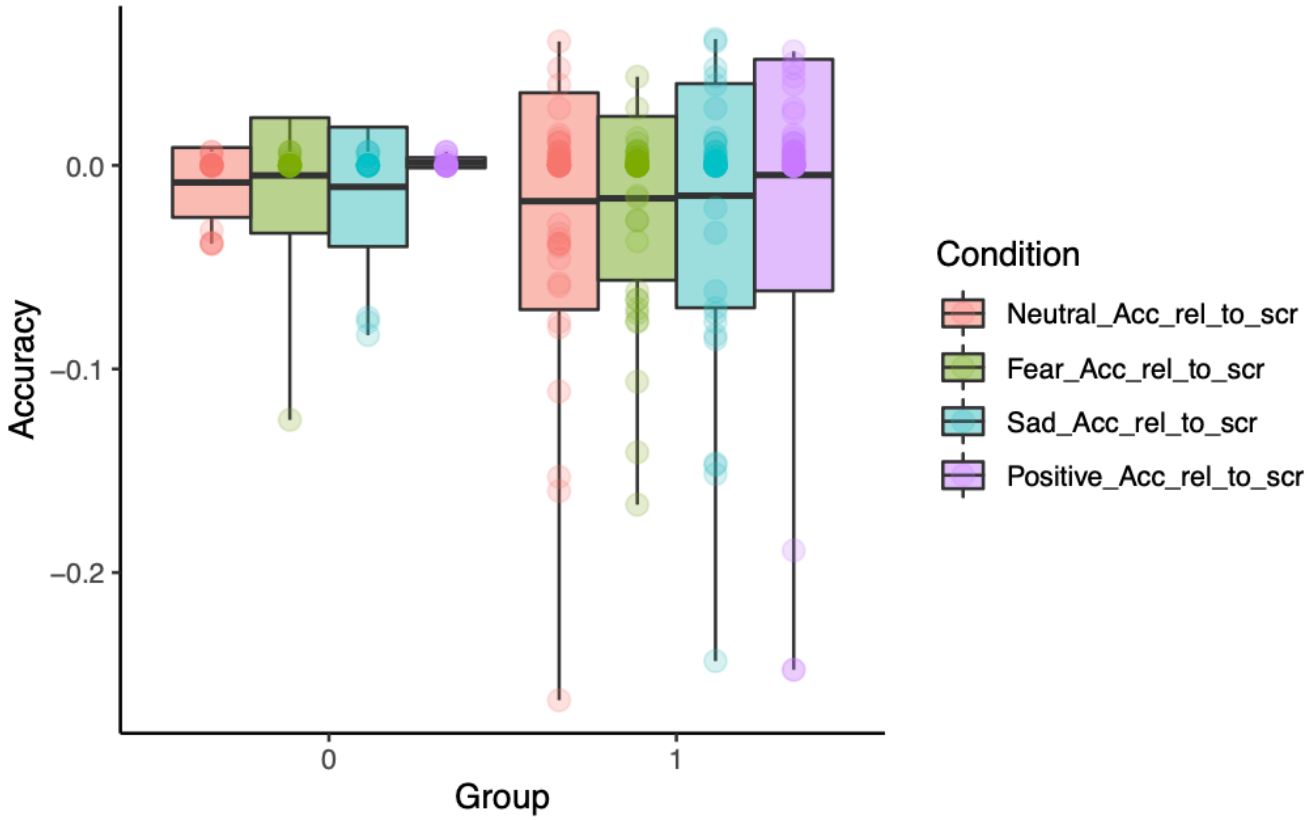


Figure 4

Accuracy for Distractors. Error bars plot of the effects of age and condition on accuracy. Significant effects of accuracy were seen (F(9, 288) = 1.279, p=0.015) such that the older participants were more accurate. Controls are reference group 0, CASA is group 1.

3.1.2 Behavioural Analysis of Targets

In the targets analysis of reaction time, there were no significant effects, but a trend in age (F(9, 294) = 0.51, p=0.089) such that older participants had a shorter reaction time (see Figure 5). Lastly, in the targets analysis of accuracy, we saw effects of age (F(9, 293) = 3.935, p=0.033) and group (F(9, 293) = 3.935, p=0.033). The control group and the older participants were more accurate on the task (see Figure 6). These results, although only mildly significant,

showed that responses to emotional stimuli were influenced by age and whether the participant is diagnosed with Axis 1 Neuropsychological Disorders.

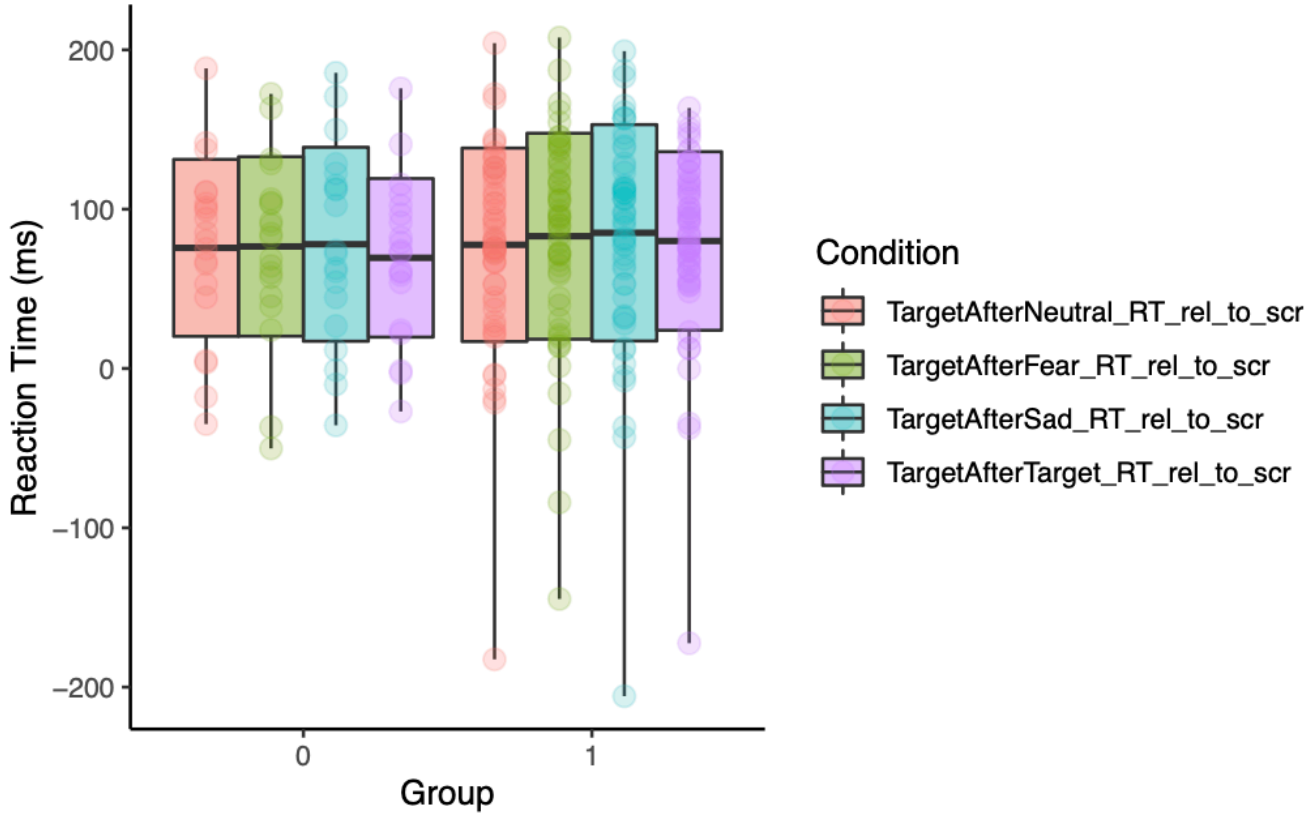


Figure 5

Reaction Time to Targets. Error bars plot showed no significant effects, but a trend showed older participants have a shorter reaction time ($F(9, 294) = 0.51$, $p=0.089$). Controls are reference group 0, CASA is group 1.

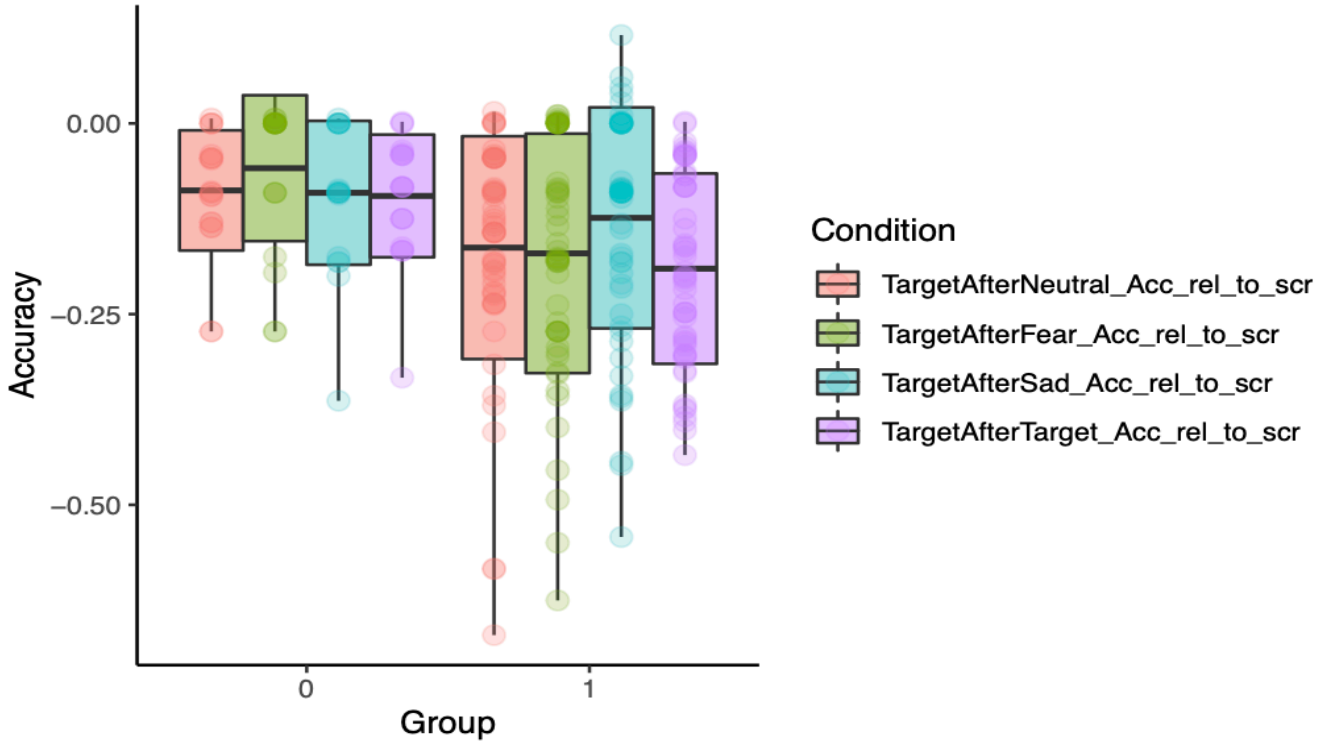


Figure 6

Target Accuracy. Error bars plot of the slight effects of Age (F(9, 293) = 3.935, p=0.033) and Group (F(9, 293) = 3.93, p=0.033) such that the older participants and control group were slightly more accurate on the task. Controls are reference group 0, CASA is group 1.

3.2 ERP Data

Analysis of the ERP data was separated by stimuli type: Distractor or Target. Analysis was also organized by ERP, P100 or P300, and electrode (POR, POL, Oz, PR, PL, and Pz; see Figure 2). Results were reported accordingly, with distractors analyses and targets analyses separated, followed by P100 and P300 ERPs. Significant results were reported, with non-significant covariates and interactions reported in the appendix. We identified significant differences in neural functioning, as measured by amplitude and latency, between healthy adolescents and those with Affective Disorders, particularly at the P100 and P300 ERPs. The

P100 and P300 measure neural activity at 100 ms and 300 ms, respectively, and are well-known metrics of attention.

3.3 ERP Data: Analysis of Distractors

3.3.1 P100 POR Analysis of Distractors

Analysis of the P100 showed significant effects in both distractors and targets analyses. The POR electrode is located on the Parietal-Occipital Right region of the head (electrode 140, Figure 2). Distractors' analysis included comparison of the negative distractors (Fear + Sad) against the neutral distractors, and comparison of the CASA and control groups with age and sex as covariates. In the P100 Distractors analysis of the POR electrode (PO4 electrode), we saw significant effects of age and experimental group. Effects of experimental group ($F(2, 45) = 6.118$, $p=0.005$) showed the healthy controls with a larger response to the emotional distractors in both the negative and neutral stimuli types (see Figure 7). Further analysis with ANOVAs showed this response was driven by amplitude ($F(2, 45) = 11.247$, $p=0.002$), with a non-significant latency. We also saw a within-groups effect of experimental condition ($F(2, 45) = 11.118$, $p<0.001$) such that both control and CASA groups showed a significantly larger amplitude in response to the negative distractors compared to the neutral distractors (see Figure 8). Lastly, we saw a significant effect of age ($F(2, 45) = 7.771$, $p=0.001$) such that younger participants showed a larger amplitude in response to both negative and neutral distractors (see Figure 9). ERP grand averages at POR showed the same difference of healthy controls having a larger amplitude compared to the CASA group in response to the distractors.

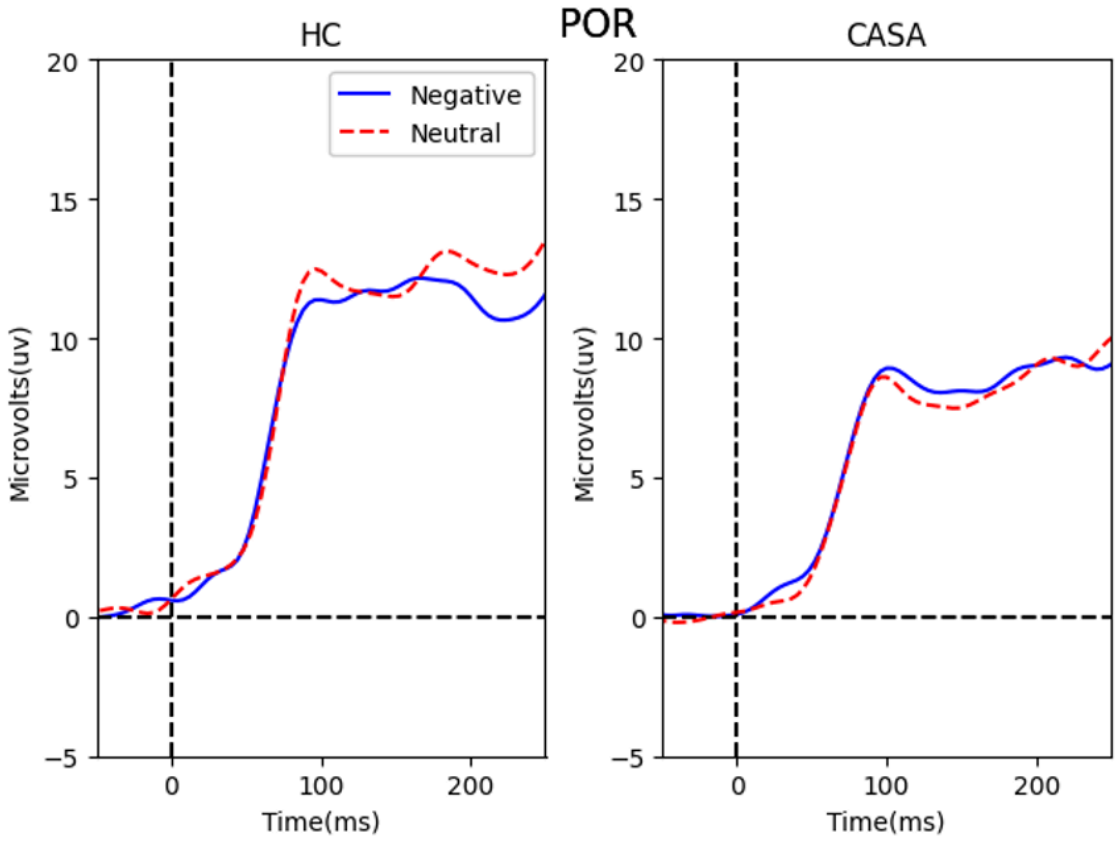


Figure 7

P100 POR Grand Averages of Distractors. POR (Parietal-Occipital Right) electrode showed significant effects of experimental group ($F(2, 45) = 6.118$, $p=0.005$). Further analysis with ANOVA showed results were driven by amplitude ($F(2, 45) = 11.247$, $p=0.002$), with a non-significant latency. The healthy controls (HC) showed a larger amplitude compared to the clinical group, denoted as "CASA" (recruited from CASA Mental Health Program), in response to the emotional distractors in both the negative and neutral stimuli types.

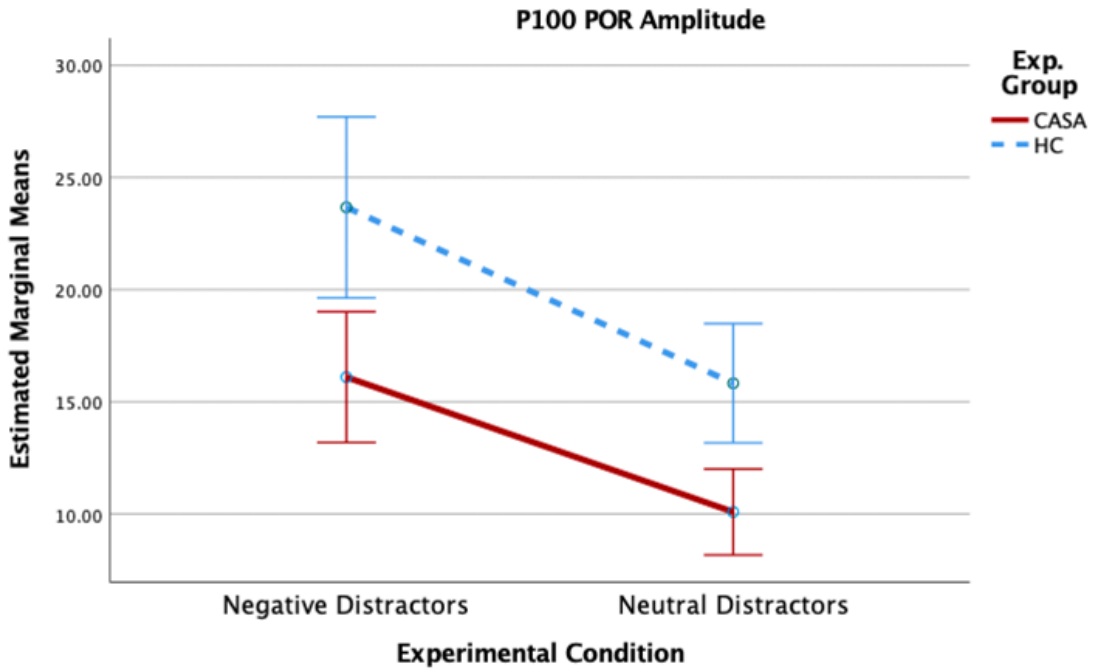


Figure 8

P100 POR Distractors Amplitude. POR electrode analysis of means (microvolts) showed significant effects of experimental group ($F(2, 45) = 6.118$, $p=0.005$). Further analysis with ANOVA showed results were driven by amplitude ($F(2, 45) = 11.247$, $p=0.002$), with a non-significant latency. Healthy controls had a larger amplitude in response to the emotional distractors in both the negative and neutral stimuli types. Mean age 15.00, 95% confidence intervals.

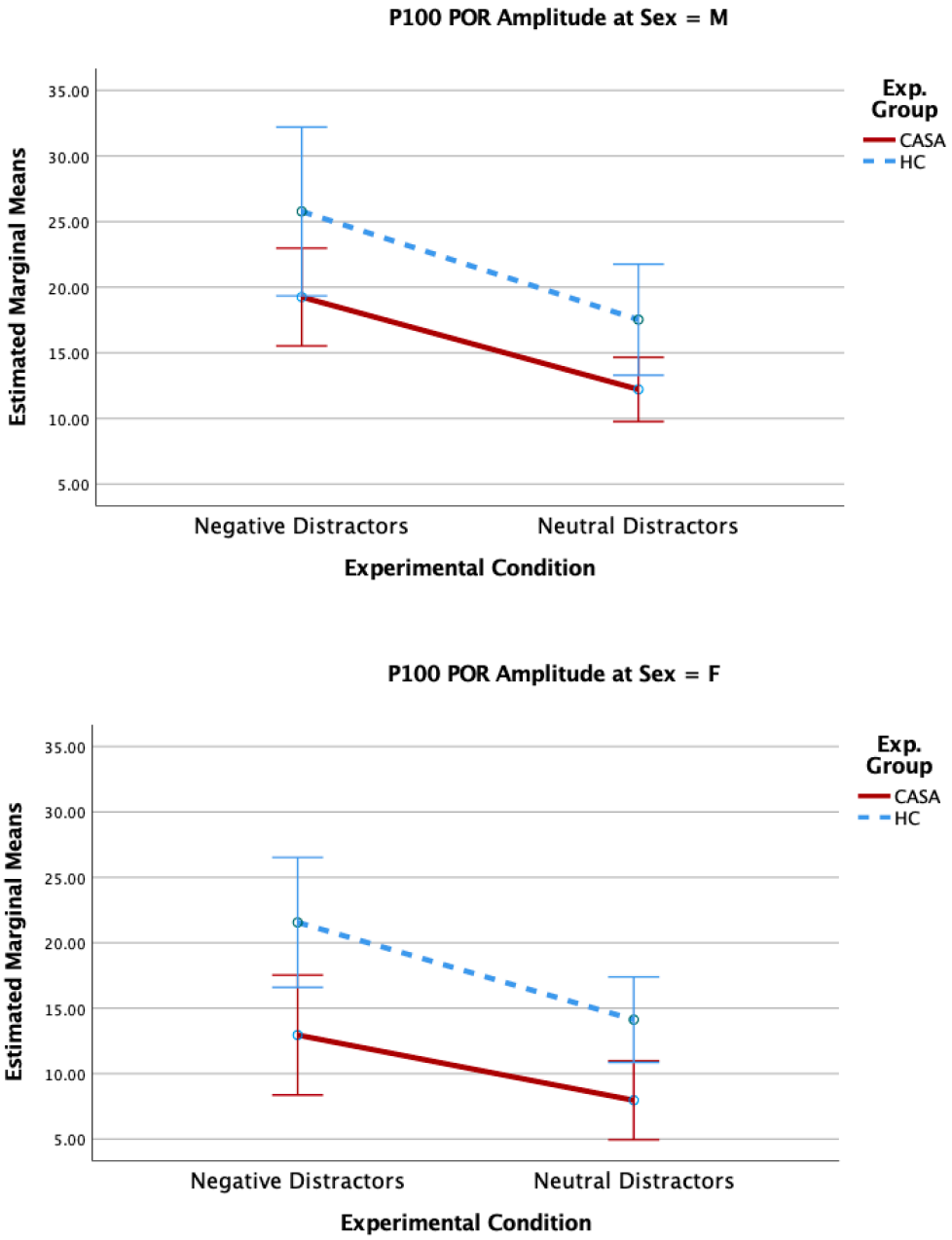


Figure 9

P100 POR Distractors Amplitude by Sex. Significant effect of age (F(2, 45) = 7.771, p=0.001) such that younger participants showed a larger amplitude in response to both negative and neutral distractors. Healthy controls (HC, dashed line) and clinical groups (CASA, solid line) show different amplitudes in response to the stimuli. Data is separated by sex, with younger

participants represented by the top graph (males' average age across both groups is approximately 1 year younger than females). Males are top panel, Females are bottom panel. Mean age 15.00, 95% confidence intervals.

3.3.2 P100 Oz Analysis of Distractors

P100 Oz Distractors analysis, located on the Occipital midline region (electrode 137, Figure 2), shows significant effects of sex ($F(2, 44) = 4.663$, $p=0.015$), such that males from both CASA and control groups showed a larger amplitude in response to both negative and neutral distractor stimuli types (see Figure 10, Figure 11). In line with this effect, we see a strong effect of age ($F(2, 44) = 6.061$, $p=0.005$), which is tied to sex in this study due to the average age of males being approximately one year younger than the average age of females (in both CASA and control groups) (Figure 11). The results showed that younger participants had a larger amplitude at POz for both negative and neutral distractors (see Figure 10). We also saw a strong within-groups effect of experimental condition ($F(2, 44) = 5.816$, $p=0.006$) such that both CASA and control groups have significantly larger amplitude following the negative distractors (see Figure 12). Lastly, there was a borderline-significant effect of group ($F(2, 44) = 3.600$, $p=0.037$) such that the healthy controls showed a larger amplitude for both distractor types. Further analysis with ANOVA showed these results were driven by amplitude ($F(2, 44) = 5.647$, $p=0.022$), with a non-significant latency. ERP grand averages also showed sex differences (which are associated with age) and group differences (see Figure 10). It is important to note that both results for the P100 Distractors stimuli were localized to the midline (Oz) and right (PR) electrodes, supporting the laterality of brain function, specifically regarding emotional processing and the effects of our emotional distractors. In sum, we only saw significant differences in emotional processing (quantified by ERPs of emotional distractors) on the electrodes located on the right/midline areas

of the brain, which supports the localization of emotional processing to the right side of the brain.

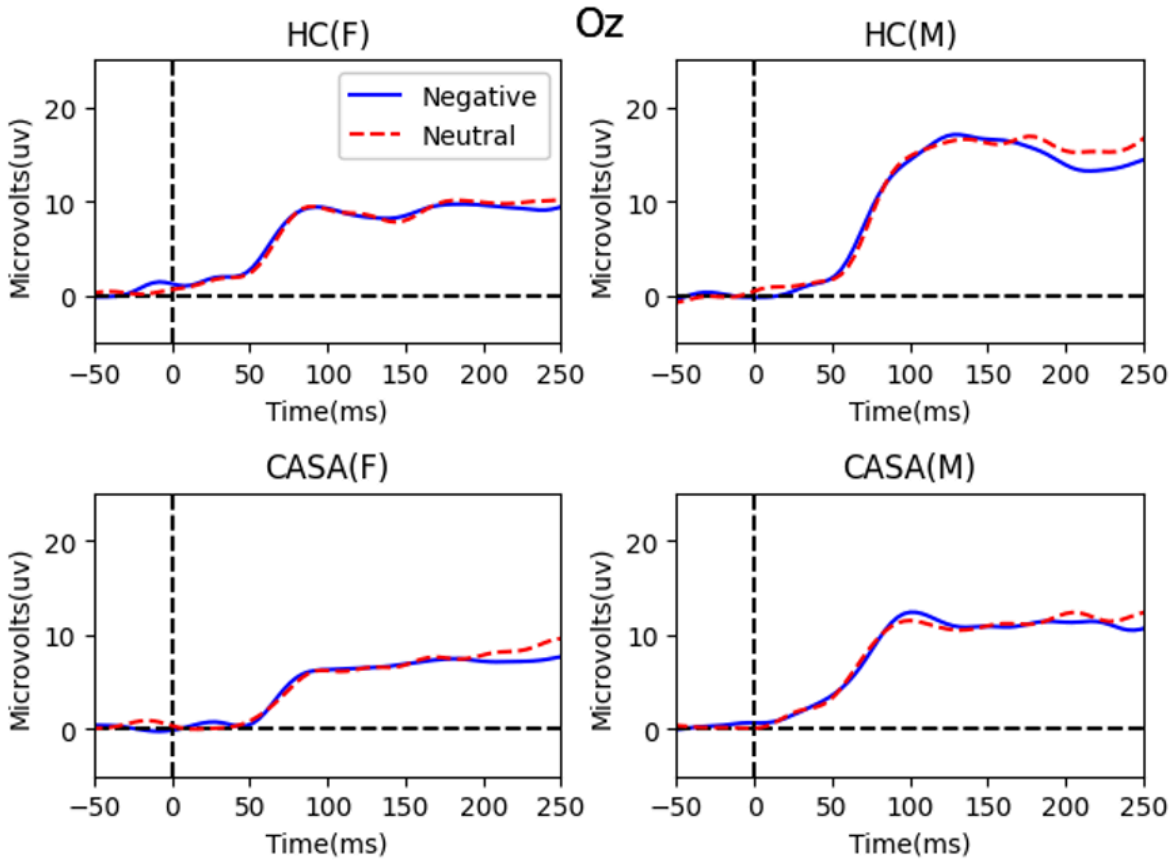


Figure 10

P100 Oz Grand Averages of Distractors. Significant effects of sex seen ($F(2, 44) = 4.663$, $p=0.015$), such that males from both CASA and control groups showed a larger amplitude. Strong effect of age also seen ($F(2, 44) = 6.061$, $p=0.005$). Further, strong within-groups effect of experimental condition seen ($F(2, 44) = 5.816$, $p=0.006$) such that both CASA and control groups had significantly larger amplitude following the negative distractors. Lastly, a borderline-significant effect of group was visible ($F(2, 44) = 3.600$, $p=0.037$) such that the healthy controls showed a larger amplitude. Further analysis with ANOVA showed group effects were driven by amplitude ($F(2, 44) = 5.647$, $p=0.022$), with a non-significant latency.

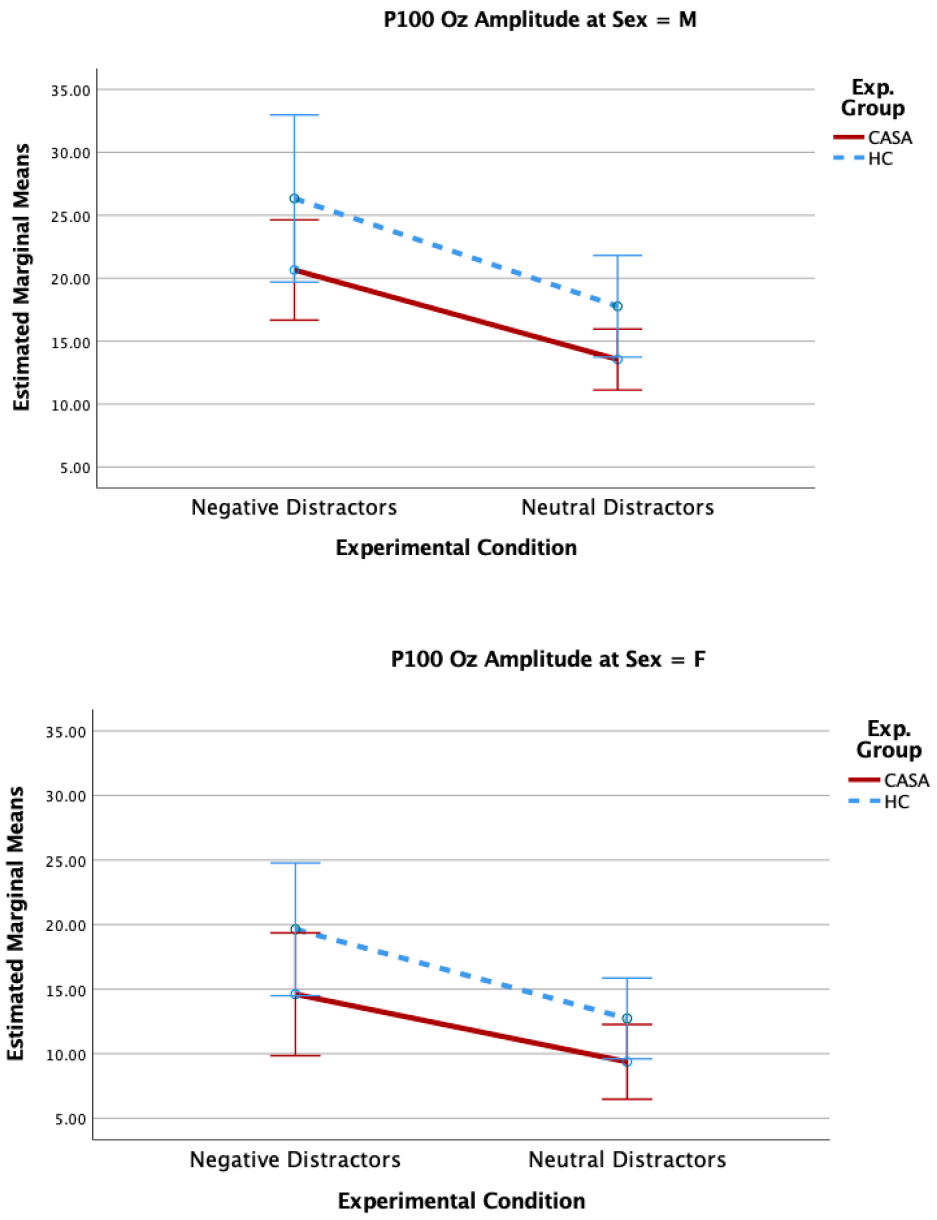


Figure 11

P100 Oz Distractors Amplitude by Sex. Significant effects of sex seen (F(2, 44) = 4.663, p=0.015), such that males from both CASA and control groups showed a larger amplitude. Strong effect of age also seen (F(2, 44) = 6.061), which correlates with sex (males ~1 year younger). Males are top panel, Females are bottom panel. Mean age 15.00, 95% confidence intervals.

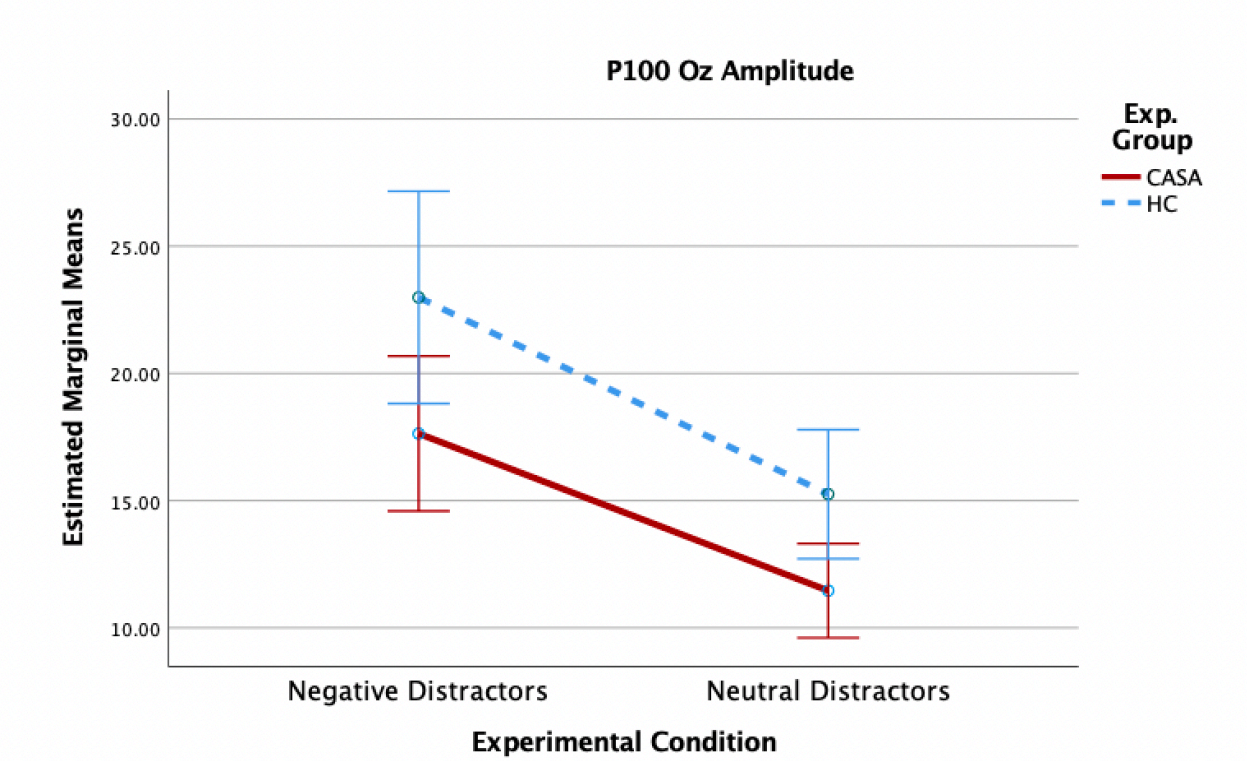


Figure 12

P100 Oz Distractors Amplitude. Strong within-group effect of experimental condition ($F(2, 44) = 5.816$, $p=0.006$) such that both CASA and control groups had significantly larger amplitude following the negative distractors. Borderline-significant effect of group ($F(2, 44) = 3.600$, $p=0.037$) such that the healthy controls showed a larger amplitude for both distractor types. Further analysis with ANOVA showed these results were driven by amplitude ($F(2, 44) = 5.647$, $p=0.022$), with a non-significant latency. Mean age 15.00, 95% confidence intervals.

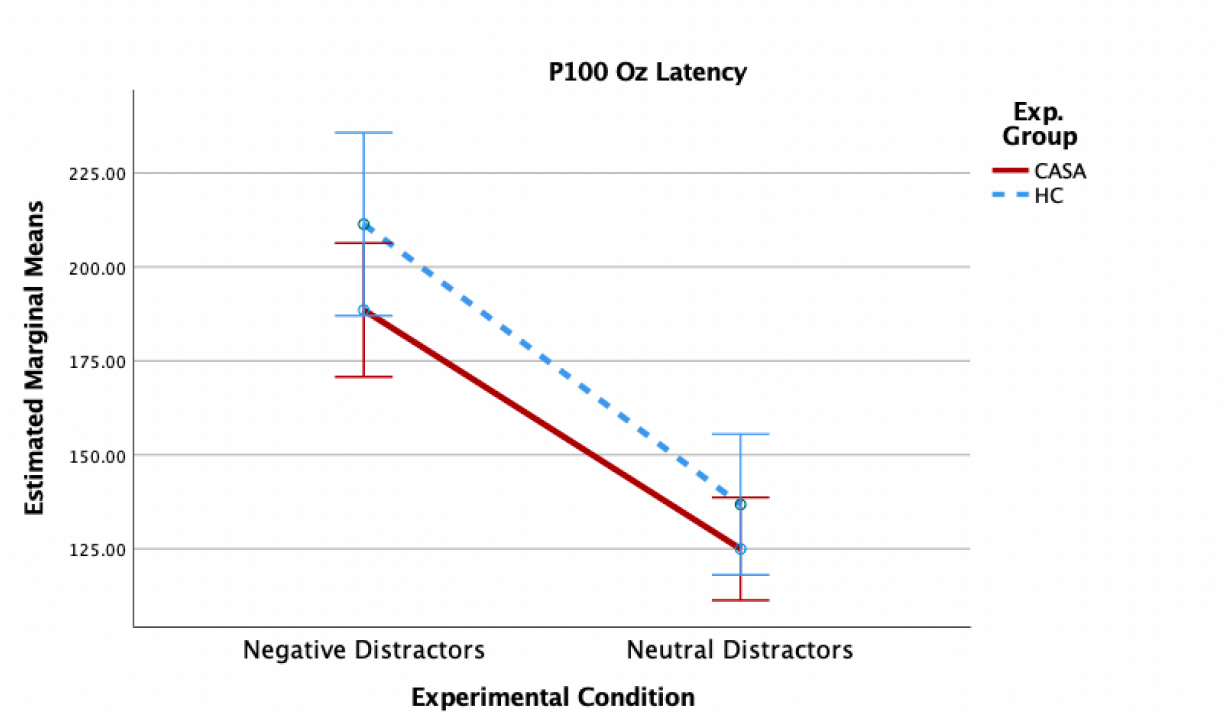


Figure 13

P100 Oz Distractors Latency. Non-significant effect of group, but trend is visible. Within-group effects of experimental condition (F(2, 44) = 5.816, p=0.006) such that the healthy controls showed a larger amplitude for both distractor types. Mean age 15.00, 95% confidence intervals.

3.3.3 P300 PR Analysis of Distractors

Analysis of the P300 ERP amplitude and latency also showed significant results of sex and experimental group. The PR electrode is located on the Parietal-Right region of the brain (electrode 162, Figure 2). The P300 PR Distractors analysis showed no significant effects of group, but showed significant effects of sex (F(2, 40) = 7.044, p=0.003) such that males had a larger amplitude in response to both distractor types (see Figure 17). This effect was driven primarily by the clinical CASA group, with the control group showing smaller differences (see Figure 16). ERP grand averages of sex and experimental groups at P300 PR showed this same

trend, with the CASA group having very significant differences between sexes, and the healthy controls showing lesser differences.

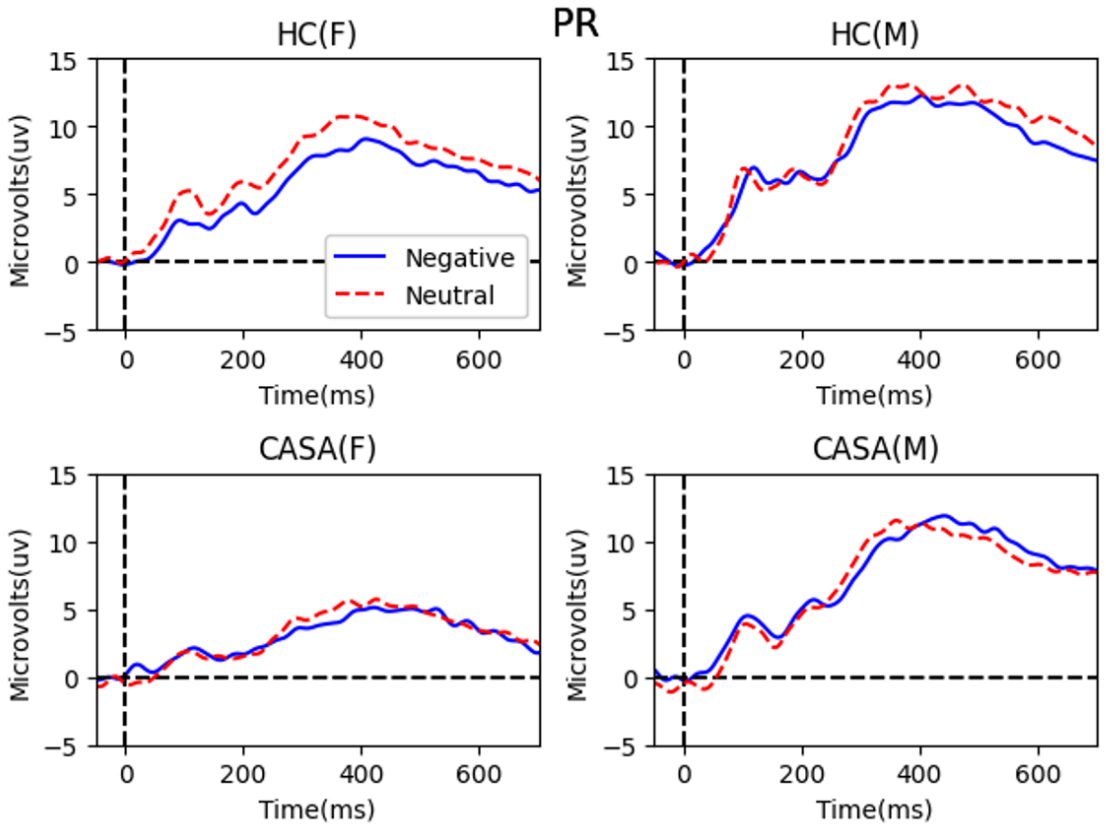


Figure 14

P300 PR Grand Averages of Distractors. Significant effects of sex ($F(2, 40) = 7.044$, $p=0.003$) such that males had a larger amplitude in response to both distractor types.

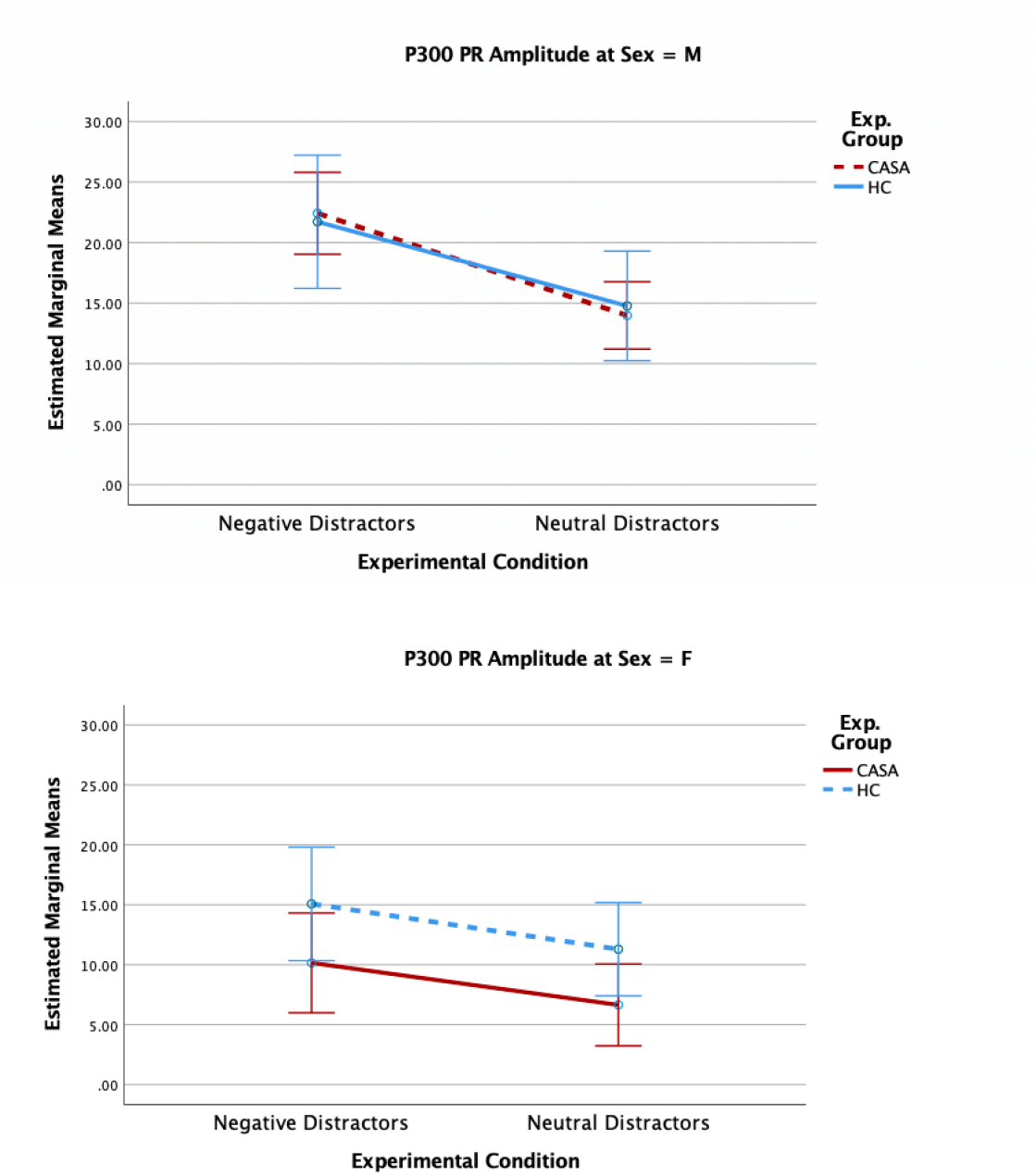


Figure 15

P300 PR Distractors Amplitude by Sex. Significant effects of sex (F(2, 40) = 7.044, p=0.003) such that males had a larger amplitude. Males are top panel, Females are bottom panel. Mean age 15.00, 95% confidence intervals.

3.4 ERP Data: Analysis of Targets

3.4.1 P300 PL Analysis of Targets

P300 Targets analysis showed results on the left, right, and centre electrodes. The PL electrode is located on the Parietal-Left area of the brain (electrode 83, Figure 2). The analysis of the emotional distractors (with no significant results on the left hemisphere), versus the more wide-spread results of the targets analysis, further showed differences between attentional processing of the targets and emotional distractors. The P300 PL Targets analysis showed trends of effects of sex. Further analysis with ANOVA showed borderline-significant effects of sex on latency ($F(2, 30) = 5.209$, $p=0.031$) with males having a longer latency for both stimuli types (see Figure 18, 19). This effect was more pronounced in the healthy controls. ERP grand average analysis displays these effects of sex on latency.

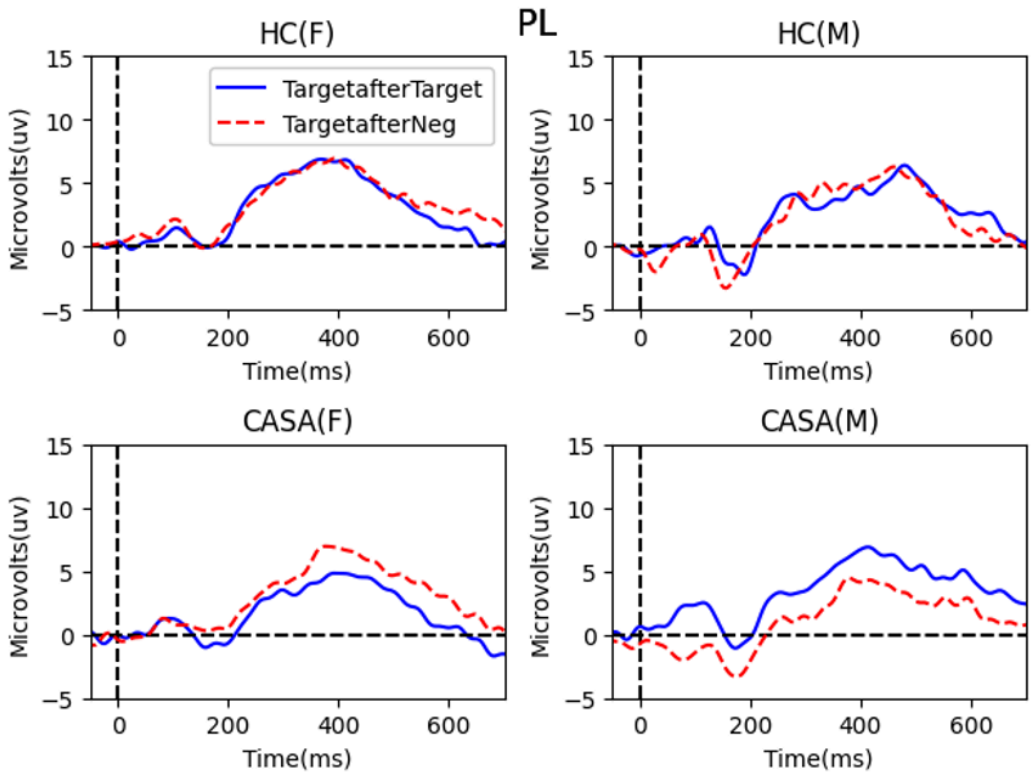


Figure 16

P300 PL Grand Averages of Targets. Analysis shows borderline-significant effects of sex on latency ($F(2, 30) = 5.209$, $p=0.031$) such that the males have a longer latency for both stimuli types.

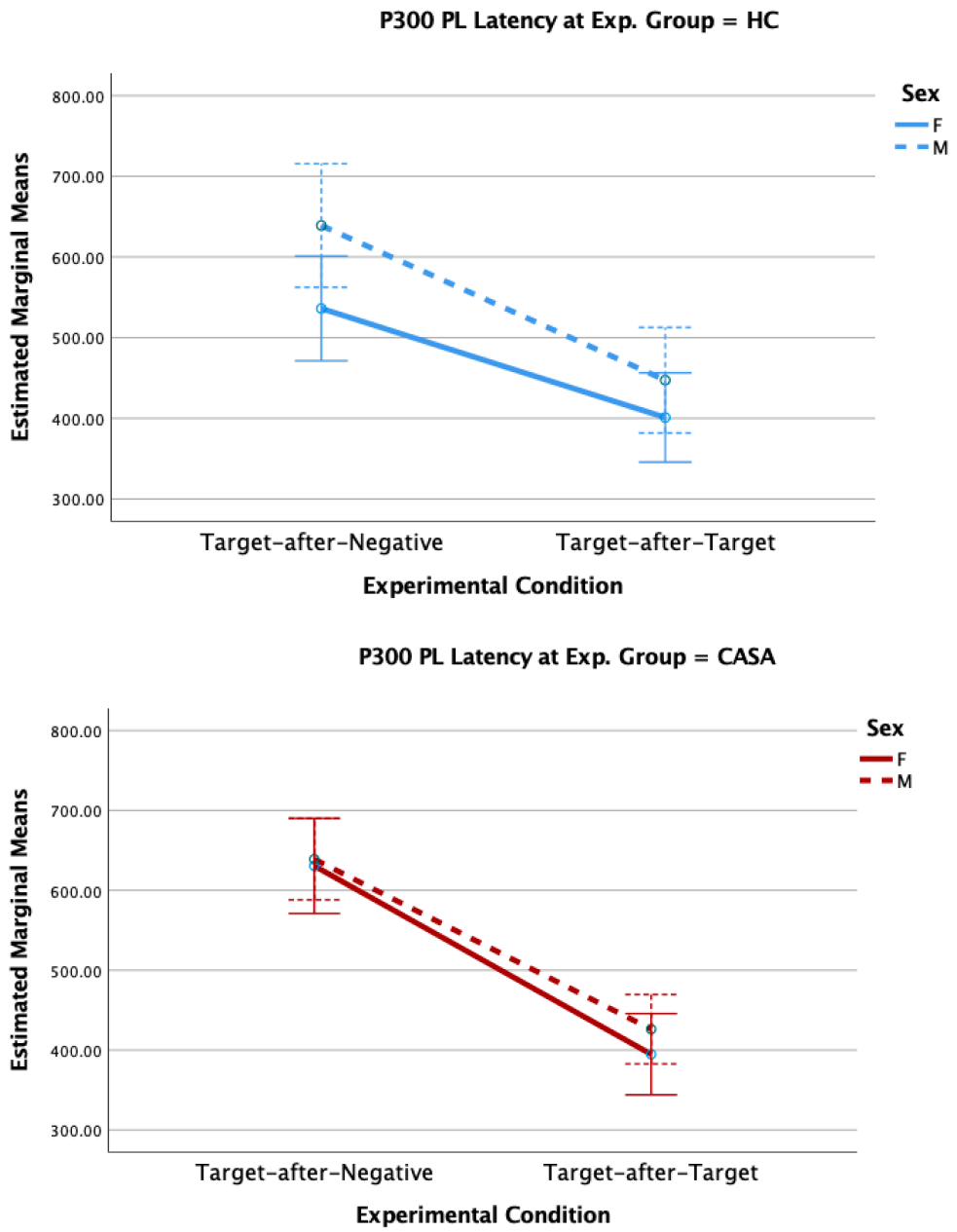


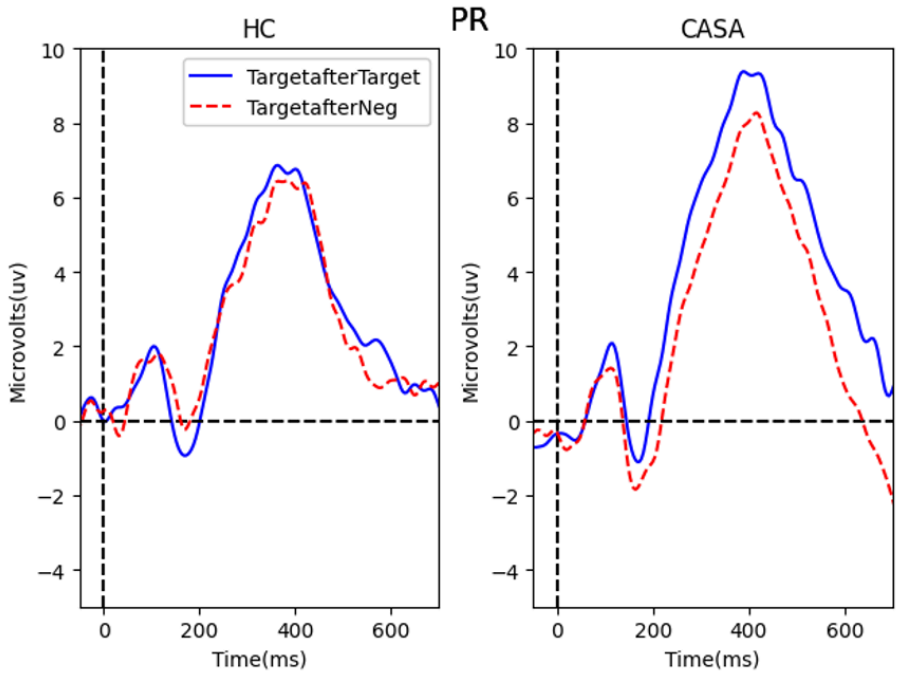
Figure 17

P300 PL Targets Latency. The P300 PL Targets analysis showed borderline-significant effects of sex on latency ($F(2, 30) = 5.209$, $p=0.031$) such that the males had a longer latency in both stimuli types. Mean age 15.00, 95% confidence intervals.

3.4.2 P300 PR Analysis of Targets

P300 PR (Parietal-Right, electrode 162, Figure 2) Targets analysis showed significant effects of experimental group ($F(2, 29) = 3.896$, $p=0.033$, Latency $p=0.017$) such that the CASA group showed a significantly longer latency for both stimuli types (see Figure 22). Further analysis with ANOVA showed these results were driven by latency ($F(2, 29) = 6.457$, $p=0.017$), with a non-significant difference in amplitude (see Figure 18).

There was also a within-group effect of experimental condition such that both CASA and control groups had a significantly longer latency and larger amplitude in response to the Target-after-Negative stimuli ($F(2, 29) = 178.436$, $p<0.001$) (see Figure 21). ERP analysis grand averages below clearly showed the difference between experimental group for both amplitude and latency, although only borderline significant ($F(2, 29) = 3.896$, $p=0.033$) (see Figure 20).



P300 PR Grand Averages of Targets. ERP analysis grand averages clearly showed the difference between experimental group for both amplitude and latency, although only borderline significant ($F(2, 29) = 3.896$, $p=0.033$).

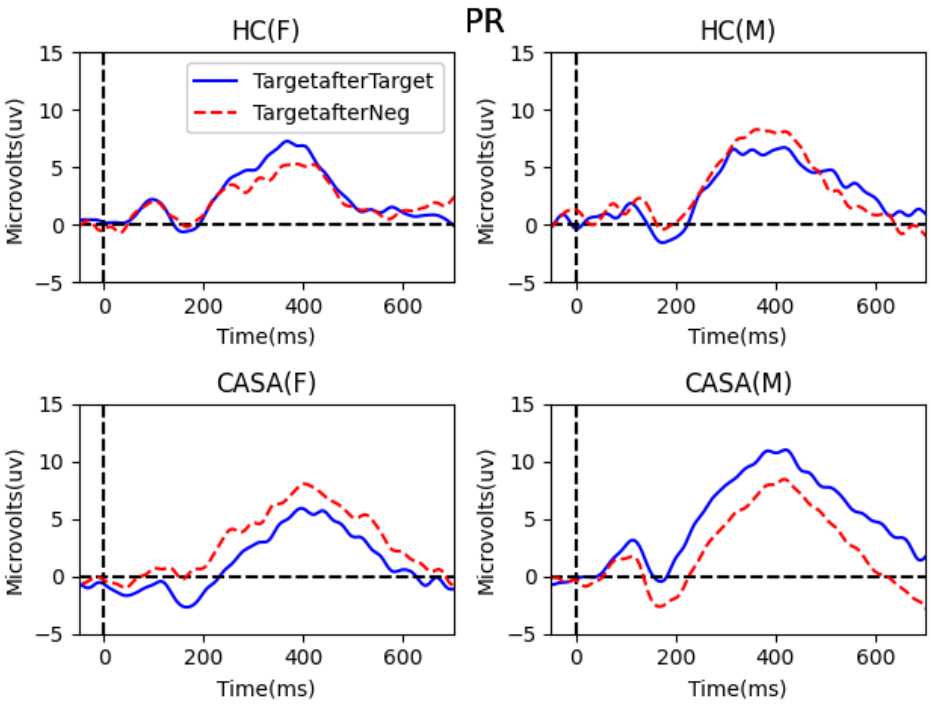


Figure 18B

P300 PR Grand Averages of Targets, Separated by Sex. ERP shows a non-significant effect of experimental condition by sex.

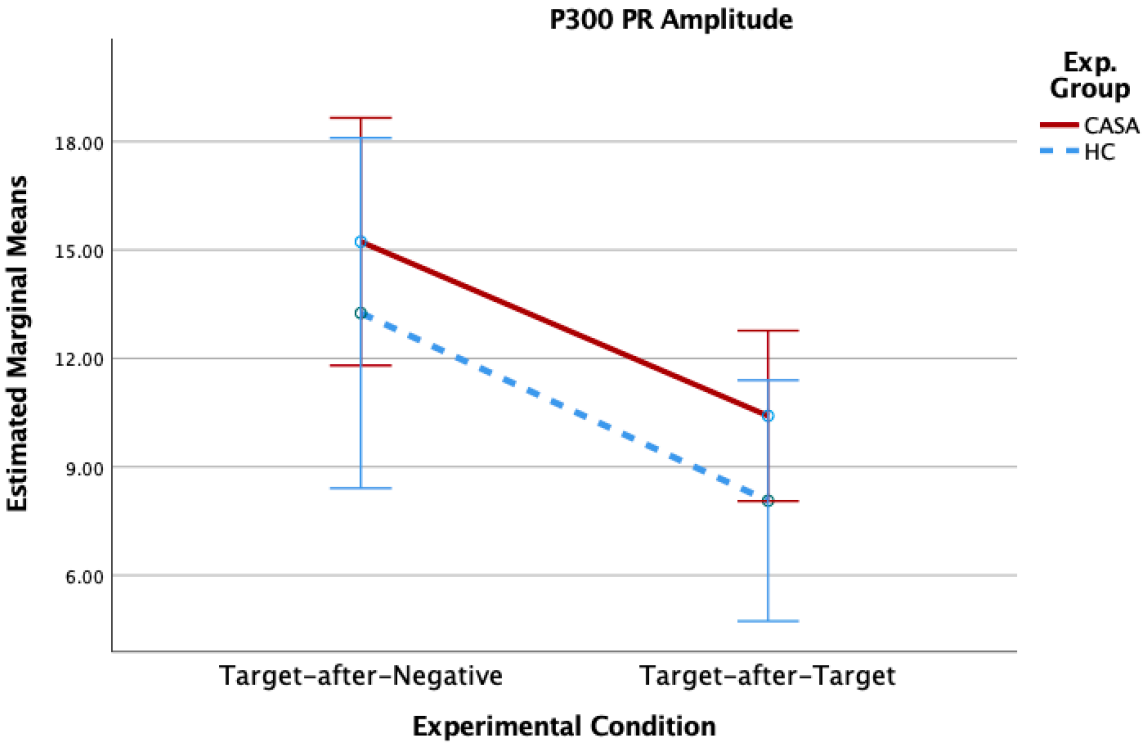


Figure 19

P300 PR Targets Amplitude. Analysis showed mildly significant effects of experimental group ($F(2, 29) = 3.896$, $p=0.033$) such that the CASA group showed larger amplitude for both stimuli types. Further analysis with ANOVA showed these results were driven by latency ($F(2, 29) = 6.457$, $p=0.017$), with a non-significant difference in amplitude (see Figure 18). Mean age 15.00, 95% confidence intervals.

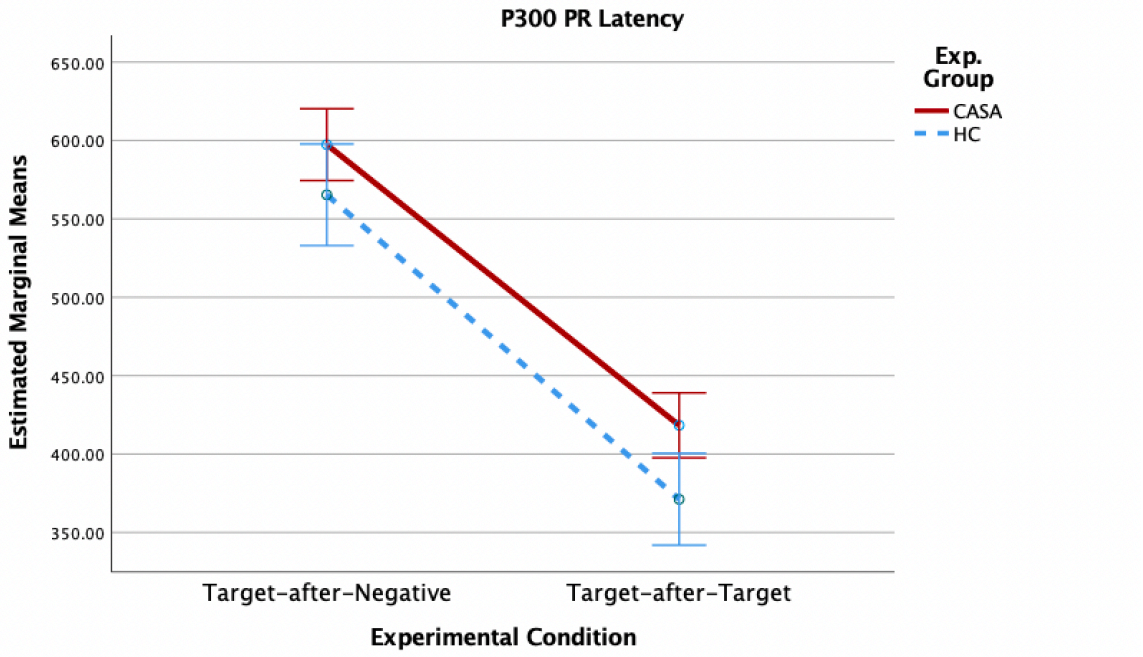


Figure 20

P300 PR Targets Latency. Analysis showed significant effects of experimental group (F(2, 29) = 6.457, Latency p=0.017) such that the CASA group showed a significantly longer latency for both stimuli types. Mean age 15.00, 95% confidence intervals.

3.4.3 P300 Pz Analysis of Targets

P300 Pz Targets analysis showed a significant effect of sex (F(2, 36) = 7.355, p=0.002), such that males showed a longer latency on both Target-after-Target and Target-after-Negative stimuli (see Figure 24, 25). The Pz electrode is a midline electrode located in the Parietal region (electrode 101, Figure 2). There was also a significant effect of experimental condition (F(2, 36) = 83.001, p=0.001) for both the CASA and control groups, such that both groups showed a significantly larger amplitude and longer latency in the Target-after-Negative stimuli types (see Figure 26). Lastly, we saw a slight 3-way effect of ExperimentalGroup*Sex*ExperimentalCondition (F(2, 36) = 3.352, p=0.048), but this result seemed to be driven primarily by experimental condition. ERP results of the Targets analysis of

P300 Pz showed some noise specifically in the CASA (Female) group, and this is likely due to a small number of trials remaining after removing those that had significantly worse noise. Regardless, these grand averages show very clearly the significant results of sex (see Figure 23).

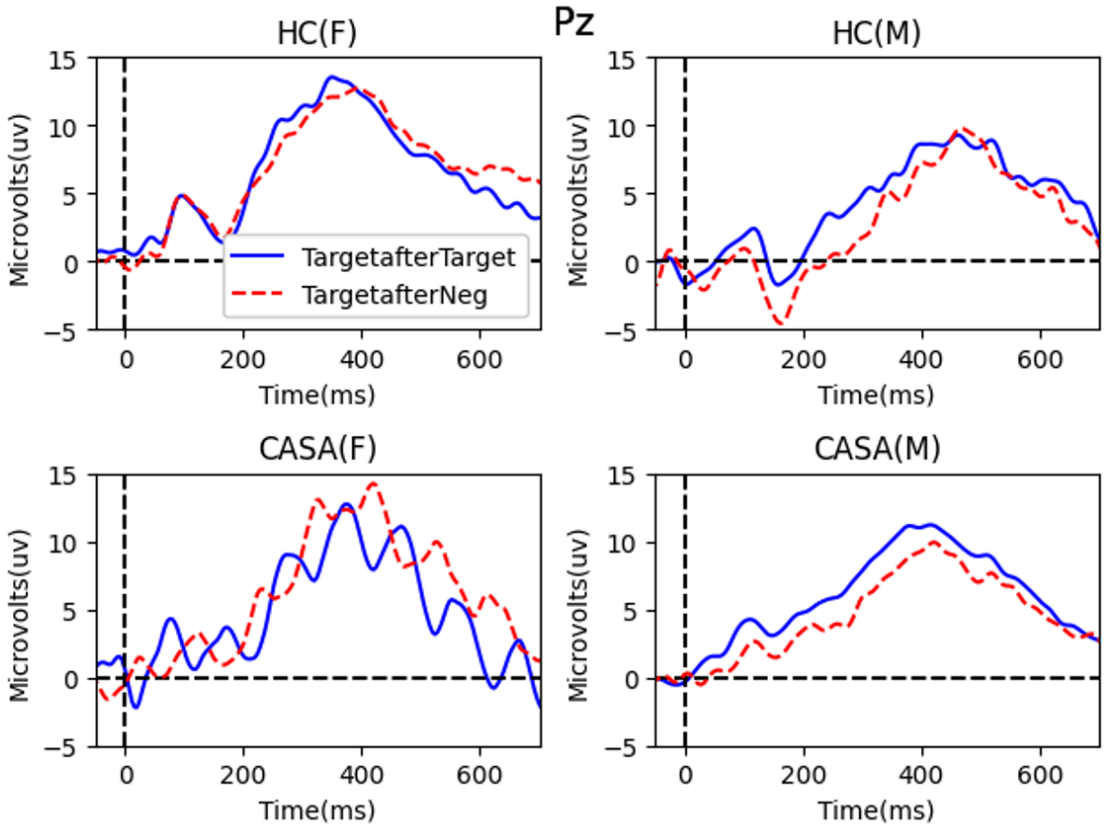


Figure 21

P300 Pz Grand Averages of Targets. Analysis showed a significant effect of sex ($F(2, 36) = 7.355$, $p=0.002$), such that males showed a longer latency, females a larger amplitude. There was also a significant effect of experimental condition ($F(2, 36) = 83.001$, $p=0.001$) for both the CASA and control groups, such that both groups showed a significantly larger amplitude and longer latency in the Target-after-Negative stimuli.

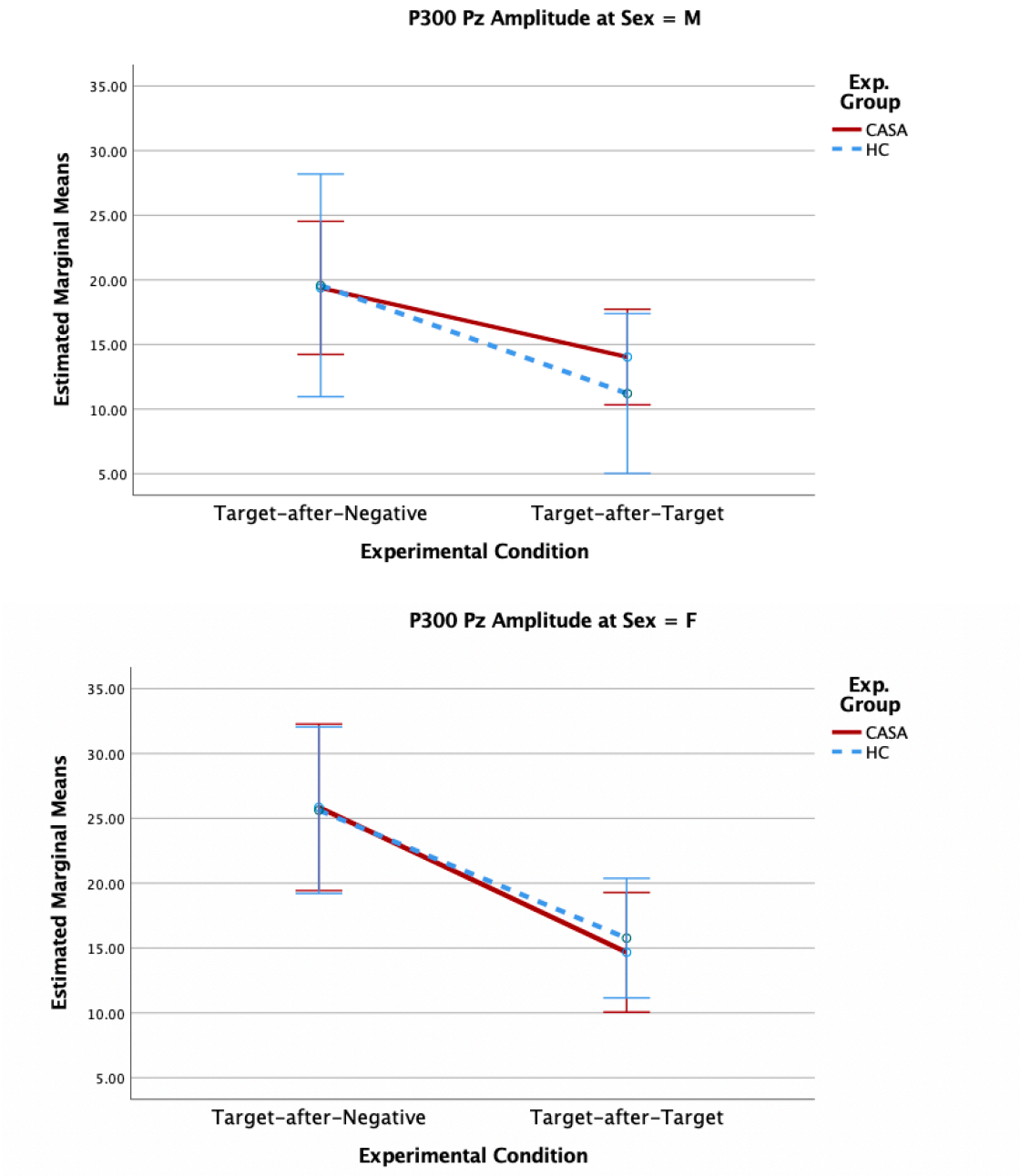
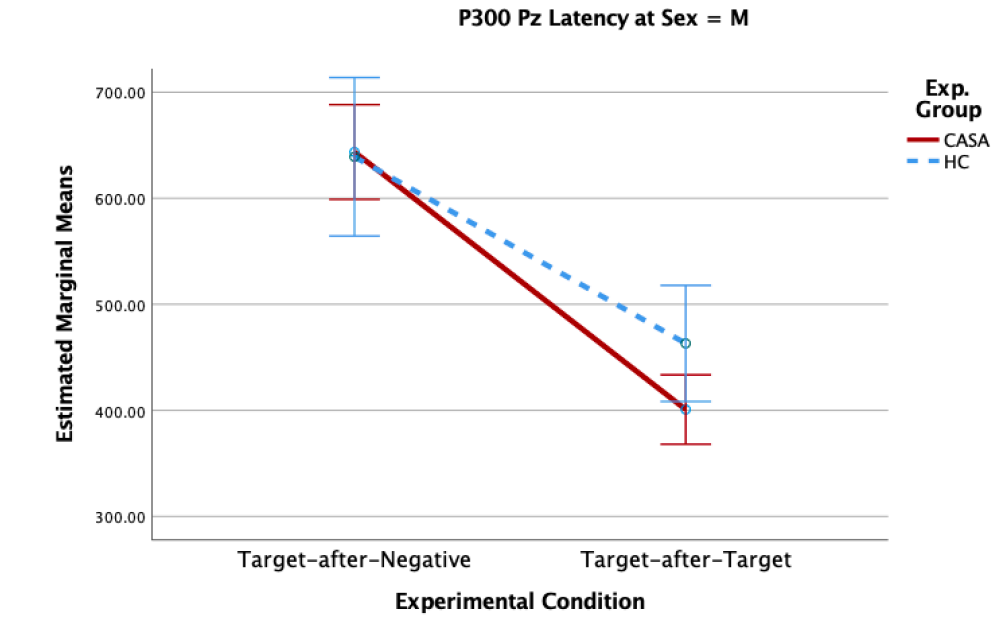


Figure 22

P300 Pz Amplitude by Sex. Significant effect of sex (F(2, 36) = 7.355, p=0.002), such that females showed a larger amplitude on both Target-after-Target and Target-after-Negative stimuli types. Males are top panel, Females are bottom panel. Mean age 15.00, 95% confidence intervals.



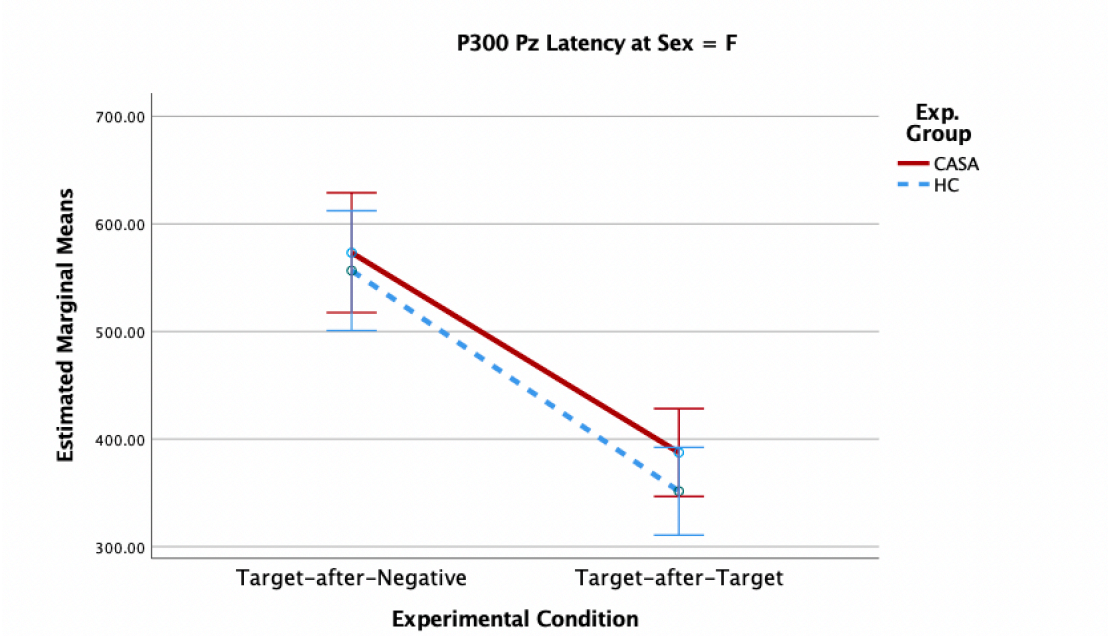


Figure 23

P300 Pz Latency by Sex. Analysis showed a significant effect of sex (F(2, 36) = 7.355, p=0.002), such that males showed a longer latency on both Target-after-Target and Target-after-Negative stimuli types. Males are top panel, Females are bottom panel. Mean age 15.00, 95% confidence intervals.

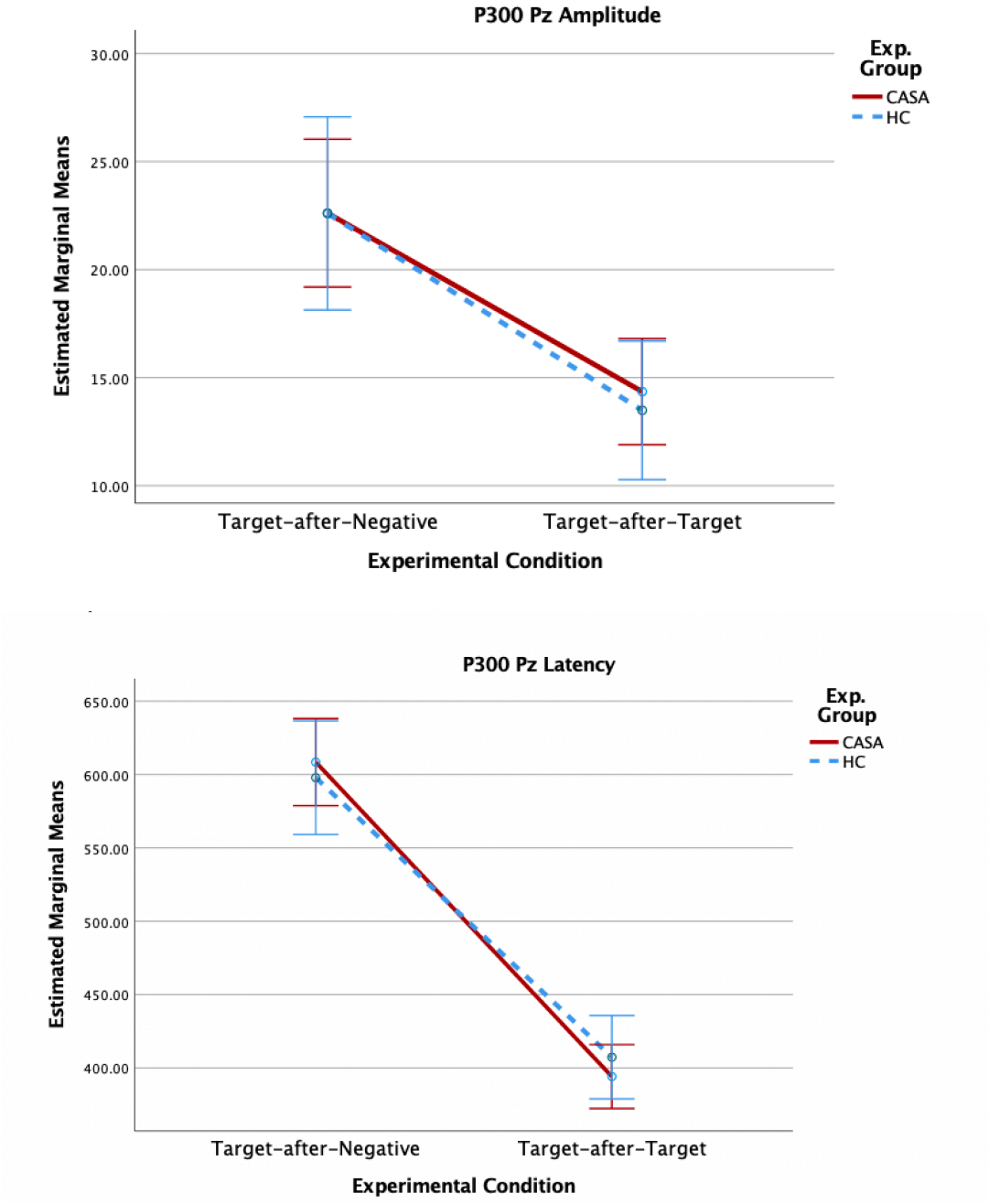


Figure 24

P300 Pz Amplitude & Latency. See a slight 3-way effect of ExperimentalGroup*Sex*ExperimentalCondition ($F(2, 36) = 3.352$, $p=0.048$), which seems to be driven primarily by effects of experimental condition. Mean age 15.00, 95% confidence intervals.

3.5 Summary of ERP Analysis

In analysis of the P100 and P300, significant effects of sex and experimental group were found at various electrodes. There were trends at most locations showing either sex effects or an effect of experimental group, suggesting that age, sex, and perhaps brain development stages have an influence on emotional processing and attention in this task. Further, we saw laterality effects such that the emotional distractor effects were localized to the right and midline electrodes, with no distractor effects seen in the left hemisphere. This is in line with literature suggesting emotion lateralization differences in the brain, with asymmetrical processing of emotions focussed in the right hemisphere (in right-handed individuals) (Demaree et al., 2005). Our study included only 2 participants who were left-handed (48 right-handed), and with most left-handed people showing the same brain lateralization as right-handed people, these participants are unlikely to affect our results.

Chapter 4:

Discussion

Discussion

The present study was designed to examine the nature of attention-emotion interactions in adolescents. In this study of attention, a broad, overarching neural process, we primarily focussed on selective attention, or the ability to focus on one thing while ignoring others (Ko et al., 2017). Attention can be split into bottom-up, or environmental-mediated attention, and top-down, or voluntarily-controlled attention (Vossel et al., 2014). The top-down, voluntarily-controlled attention is typically referred to as selective attention. Emotion regulation is the ability for certain neural networks to control and direct the psychological and physiological responses to arousal and valence, or emotion intensity and emotion type, respectively (Hartikainen, 2021). In our population of adolescents, we had two groups: clinical (33 participants) and control (17 participants). The clinical (CASA) group consists of at-risk adolescents with Axis 1 psychological disorders, such as ADHD, anxiety, depression, and conduct disorders, recruited from CASA mental health services. These disorders are thought to affect both attentional control and emotion regulation (Steinberg & Drabick 2015). We took measurements of the participants' neural ERP responses, and specifically quantified and analyzed the P100 and P300 ERPs that occur at approximately 100ms and 300ms respectively. Both of these waveforms reflect attention, with the P100 reflecting more "automatic" attentional mechanisms, and the P300 reflecting top-down, more "conscious" attentional mechanisms. Thus, in addition to behavioural differences, we expected to see significant differences between the CASA and control groups on both the P100 and P300 waveforms in response to different aspects of the task. The CASA and control groups were matched for age and sex.

Our study used an emotional oddball task and ERP to compare participants' attention and emotion processing between the control and CASA groups. In the oddball task, most stimuli are

the same (baseline stimuli), but occasionally a different type of stimuli appears. This infrequent, different stimulus is then called the "oddball target." In our oddball paradigm, we also included infrequent emotional distractors. These emotional distractors can affect neural responses and performance. The emotional distractors included three different types of distractors: sad, fearful, and neutral emotional stimuli. The task itself involved two actions for participants: press a button with their left hand when they viewed distractor stimuli, and press another button with their right hand when viewing target stimuli. ERP analyses was recorded while participants completed the task.

Behavioural data was collected, where reaction time and accuracy was analyzed for both the target and distractor stimuli types. Similarly, age and sex were also included in the analysis.

The P100 and P300 ERPs were collected while conducting the emotional oddball task. The POR, POL, Oz, PR, PL, and Pz electrodes were analyzed (see Figure 2). We compared both amplitude and latency between the CASA and control groups, as well as analyzed confounding variables such as age and sex. An increased P100 latency can reflect dysfunction in the early processes of the attentional processes, including visual processing, perception, and awareness. An increased P300 latency reflects how long it takes the participants' brain to consciously respond to the stimulus, so a longer latency likely means they are taking longer to process that stimulus. The amplitude reflects the significance of the neural response, or how much their brain is responding, and, in the context of this study, the strength of the emotional or attentional response. There is a great deal of literature suggesting a larger amplitude means more neural resources are being devoted to this stimulus (Singhal & Fowler 2004; 2005). For example, for the P100, a larger amplitude is thought to be indicative of high reactivity in early attention, and for our study, reactivity to visual emotional stimuli (Pourtois et al., 2005). In the P300, a larger

amplitude may indicate that the participant is allocating more neural resources to process the visual emotional stimuli (Singhal et al., 2012). Significant results were seen in both the behavioural and ERP analyses, with effects of Group, Age, Sex, and Experimental Condition found separately across the various analyses.

We will now discuss each of the major findings. To follow the results as described in the methods and results sections, we will discuss first the behavioural findings followed by the ERP findings. Accordingly, we will then discuss results seen in the distractor stimuli, followed by results found in the target stimuli.

4.1 Behavioural Data

4.1.1 Reaction Time & Accuracy

Although there were no effects with Reaction Time in response to distractors or targets in this study, this result is important when also considering the accuracy data. Since there were accuracy differences, but no reaction time differences, one interpretation is that the overall performance strategies used by the participants were similar between groups.

Regarding the distractor stimuli, where the participants viewed emotional stimuli (positive, negative (fear + sad), or neutral images of faces), accuracy was significantly better in older participants. Although narrowly meeting the threshold of our adjusted p-value of 0.017, this result fits with the expected findings. One argument is that these types of emotional stimuli hold a greater evolutionary importance, for example for reproductive purposes, such that older adolescents (of reproductive age), therefore pay attention to these stimuli in order to reproduce and survive (Somerville, 2013; Blakemore, 2008). Developmental patterns also indicate increased attentional control as the brain develops with age, perhaps accounting for improvements in accuracy (Davies et al., 2004). Luna et al. (2001) suggested that there are many

neurodevelopmental factors that play into poor attention and top-down modulation in adolescents, including still-developing pre-frontal cortices and parietal areas of the brain, as well as ongoing synaptic pruning and myelination.

In response to the target stimuli, we also saw a small effect of accuracy where the control group was higher. Furthermore, the older participants were more accurate. We know that older adolescents have increased brain development in areas that support visual processing (Luna et al., 2004; Gur & Gur 2016), so it is logical that we saw older participants show more accuracy on the task.

Our slight effect of group shows that with the control group being more accurate, the CASA group may be showing some of the same lack of brain development that the younger participants show, with less prefrontal development and synaptic pruning compared with the healthy participants. Effectively, the CASA group neural patterns could align with those of younger, healthy participants, and this may be an interesting area to explore in future research with MRI. Accuracy on both the distractors and the targets also differed as a result of age. For both early and late types of attention, increased brain development with age is thought to improve accuracy on attention tasks (Gur & Gur, 2016). This was reflected in our research, with improved accuracy with age shown on both the distractors and the targets stimuli. My analysis of the literature has shown that there are significant age differences in brain development (Yurgelun-Todd, 2007; Blakemore, 2008; Somerville, 2013), but how that brain development is differentially affected in children and adolescents with Axis 1 disorders is less known.

4.2 ERP Data

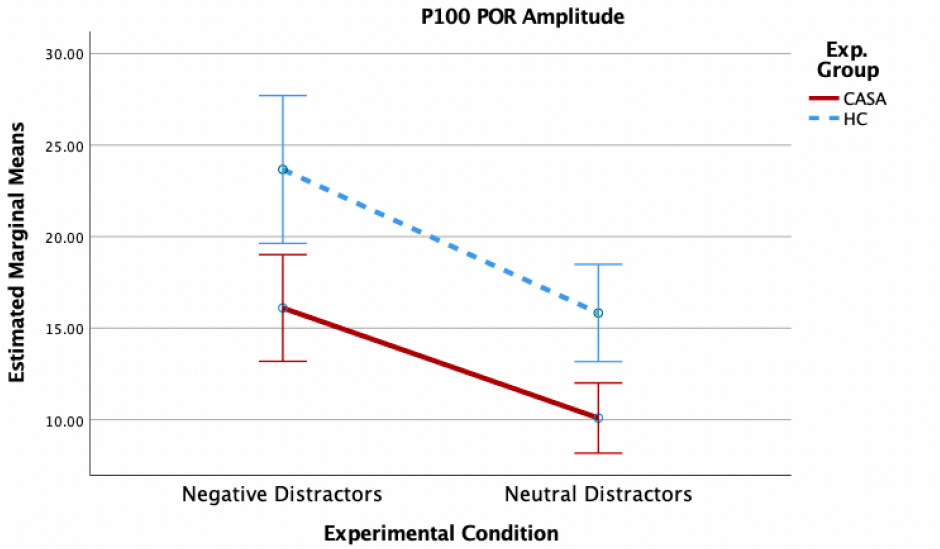


Figure 25A

P100 POR Distractors, Healthy Controls with Larger Amplitude. Healthy controls show a significantly larger amplitude on the P100 POR in response to the distractor condition when compared to the CASA group (F(2, 45) = 11.247, p=0.002).

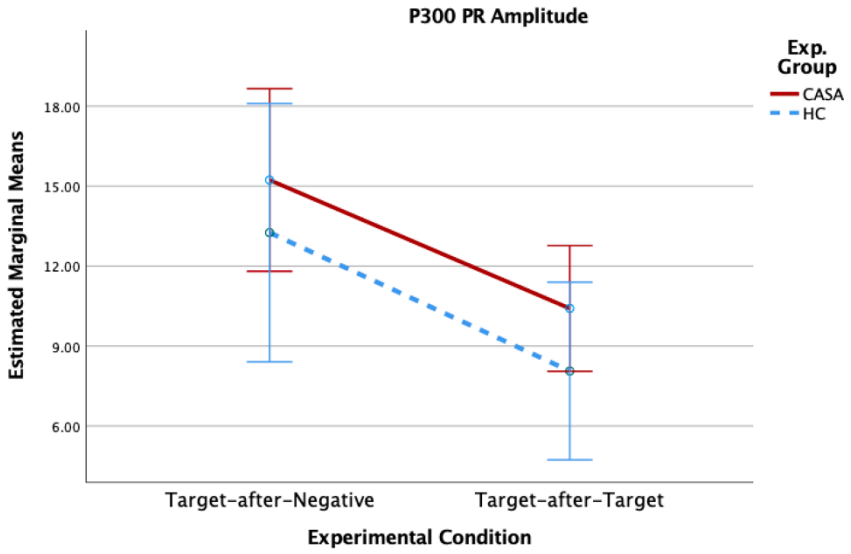


Figure 25B

P300 PR Targets, CASA Group with Larger Amplitude. CASA group shows a significantly larger amplitude and longer latency than controls on the P300 PR in response to the target condition when compared to the healthy controls (F(2, 29) = 3.896, p=0.033).

4.2.1 P100 in Response to Distractor Stimuli

P100 in response to distractors at POR and Oz showed strong effects of group, with the healthy controls having a larger P100 amplitude in response to emotional stimuli. One interpretation of this larger P100 suggests that the healthy participants have more attentional processing or resources available to them to complete the task (Woodman et al., 2010). Another possibility is that the larger P100 is indicative of attentional orientation to the stimuli (Woodman et al., 2010).

P100 in response to distractors at POR also showed significant effects of age, such that the younger participants (and accordingly, males), showed a larger amplitude in response to the emotional stimuli. As previously discussed, there is research supporting increased reactivity to emotional stimuli in adolescence, especially during a certain critical period correlating with puberty (Yurgelun-Todd 2007; Blakemore 2008). Due to the age range of our participants, 11-17, there are pre-pubescent, pubescent, to potentially some teens that are nearly finished puberty all included in this age group. Thus, the younger participants, who are likely at the early stages of puberty, showed a larger amplitude than the older participants due to the importance of the emotional stimuli for young adolescents (Somerville, 2013). However, we did not collect data on stages of pubertal development, so we can only hypothesize this as a potential explanation.

The P100 at Oz was larger for younger participants, showing a significant effect of age. This effect may simply be reflecting the effects of brain development, not age (Blakemore, 2008; Monk et al., 2003). The increased amplitude could indicate that younger participants are more attenuated to emotional stimuli than are the older participants, as has been suggested in papers on developmental social psychology (Somerville et al., 2013).

The P100 at the Oz electrode showed an effect of sex, where males had a larger amplitude compared to females. Although not as strong as the previous result, this finding may be related to the fact that younger, male, children are showing an increased amplitude to emotional stimuli, likely due to increased attention being placed on these emotional stimuli. Being at a critical period of development where relationships are at a high importance could be leading to different attentional emphases placed on emotional stimuli. The P100 may be reflecting an increase in attention because of the importance of emotional stimuli for social development in this age group.

Lastly, for the P100 Distractors stimuli we found significant results of experimental condition on both the POR and the Oz electrodes, such that participants' responses were significantly larger in regards to the negative emotional stimuli. This is an important aspect to our study, as it verified that the task did indeed test what we wanted it to and gives validity to the study design. We expected both groups to have an increased amplitude in response to the negative distractors, for the reason that they are more emotionally salient, and of greater importance to the brain when allocating attentional resources – specifically in adolescence.

It is also pertinent to notice that all the significant results for the emotional distractors condition showed on the right and midline electrodes. There are only null results related to emotional stimuli on the left side electrodes. This is significant, because it supports lateralization of emotional processing to the right side of the brain in adolescents. Lateralization differences are commonly researched in adults, but not in adolescents. For example, Bruder et al. (2017) studied the differential lateralization of emotion processing in adults with clinical disorders, and found reductions in activity in the right parietal regions associated with depression. A study on anxiety suggests differential lateralization of processing in the dorsolateral prefrontal cortex

(dlPFC) as a mechanism for regulating anxiety (White et al., 2023). However, these studies were done in adults. In our study, we saw differential activation on target and emotional distractor stimuli. The emotional distractors showed significant results only on the right and midline electrodes, with the target effects seen throughout all areas. These results would indicate that the regions in the brain for attention and emotional processing show lateralization differences in adolescents.

It is known that in adults the pathways involved in emotion processing include the ventrolateral, dorsolateral, and dorsomedial prefrontal cortex, as well as the anterior cingulate (Urry et al., 2006; Lewis et al., 2006, Monk et al., 2003). Reductions in prefrontal cortex activity, as well as decreased activity in the right parietal regions specifically, have also been linked to depression (Bruder et al., 2017), supporting the laterality of emotion processing in adults. Similarly, yet this time studying anxiety, research by White et al. (2023) studied the laterality of the dorsolateral prefrontal cortex (dlPFC) in regulating anxious emotions. In line with the lateralization found in our study, they found that the right dlPFC regulated responses to emotional stimuli. Even in adults, however, the research is not conclusive on laterality of emotions (Turnbull & Salas 2021). Further, adults are thought to regulate emotion and attention differently than children (Lewis et al., 2006). Our brain lateralization results, though, showed that emotion processing occurs in the same areas of the brain for adolescents and adults. Finding a laterality effect in a population of adolescents is important for understanding how the brain develops, as many other processes differ in children as compared to adults (Lewis et al., 2006).

4.2.2 P300 in Response to Distractor Stimuli

There was one significant effect on the P300 in response to the distractors at the PR electrode. The males showed a larger P300 amplitude compared to females. This result indicates

late attention (dorsal processes, reflected by the P300), may vary by sex. This may be due to the effect of increased emotional sensitivity in the younger males, perhaps owing to the stage of pubertal development and the importance placed on emotional stimuli in the social context at that age (Ernst, Romeo, & Anderson, 2009; Nelson et al., 2005; Sisk & Zehr, 2005; as in Somerville, 2013).

4.2.3 P300 in Response to Target Stimuli

The P300 in response to targets also showed significant effects of experimental condition. Specifically, the P300 at Pz showed a significantly larger amplitude and longer latency on the target-after-negative stimuli in both groups. One interpretation is that the negative emotional stimuli are taking longer to process and are requiring more attentional resources (Somerville, 2013; Monk et al., 2003; Ahmed et al., 2015).

We also saw a significant effect of sex on the P300 in response to targets. Specifically, males showed a longer latency on the P300 at Pz. This likely suggests that it is taking the males more time to evaluate the targets because they are still processing the emotional stimuli that preceded the targets (Singhal et al., 2012).

The P300 at the PR electrode showed a significant result of experimental condition, which is nice to see the validity of the task arrangement across many electrodes. Both CASA and control groups had a significantly longer latency and larger amplitude in response to the Target-after-Negative stimuli, which is what was expected. Adolescents showing a larger amplitude to negative emotional stimuli highlights the importance that are placed on these images. The longer latency taken to process these stimuli falls in line with the aforementioned importance of emotional stimuli, as well as the attentional load theory. The attentional load theory suggests that emotional stimuli can cause increased “load” on the brain, resulting in less resources for

attention (Oliveira et al., 2013). With a higher emotional salience, and greater perceived importance, the brain is putting more importance on these stimuli and processing them more carefully, causing a higher load and more processing time than neutral stimuli. These results all indicate the importance of emotional stimuli in adolescence, such that they are processed much more carefully and slowly.

For the P300 Target stimuli analysis, the PL showed a borderline effect of sex, such that males have a longer latency on both stimuli. In keeping with all our results thus far, the males take slightly longer to evaluate the negative emotional stimuli, indicating more resources are being dedicated to processing these stimuli. This might be due to the increased dedication to emotional stimuli for social reasons (Somerville, 2013), or owing to less brain development and synaptic pruning and myelination (Luna et al., 2001), or perhaps both.

Lastly, the P300 PR Target analysis showed borderline significant effects of group. Although under the p=0.05 significance threshold, they are not significant after our adjusted p-value of 0.017, yet the results are quite obvious on the ERP Grand Averages, hence why they are of interest here. In the P300 stimuli analysis, we saw the CASA group with larger responses (larger amplitude) to emotional stimuli. Moreso, the CASA group showing longer latency in the late, top-down measures of attention, as reflected by the P300, indicates that this system is likely the mechanism of dysfunction in adolescents with Axis 1 psychological disorders. Either a decreased efficiency of attentional processes in the dorsal attention system, or needing to dedicate more resources to process emotional stimuli, could lead to the differences we are seeing in the CASA group.

4.3 General Discussion

It has been argued that the P100 and P300 reflect early and late attention, respectively (Olafsson et al., 2009; Graziano & Webb, 2015). The P100 likely reflects more immediate reactions to attention and emotion stimuli, and is modulated mostly by bottom-up processes, meaning it is mediated by the environment (Graziano & Webb, 2015). When a stimulus is deemed important by our brain, such as an image of an emotional face, attention is automatically directed to that source and the brain directs resources to process that stimulus. The P100 is stimulated by visual stimuli, and thus originates in visual areas of the brain in the occipital cortex (Zhao et al., 2023). This type of externally-mediated attention is controlled by the ventral network and less so by controlled, top-down processes. The P300, however, is likely more of a reflection of controlled or top-down attention and emotion regulation. This type of attention regulates focussed attention on our target stimulus, and the processing and responses that includes. As the P300 is a more complex reflection of conscious attention, there are many areas involved. Primarily, prefrontal cortex and parietal regions are involved for visual attention such as in our task (Linden 2005). Although both systems of attention work together, the P100 and P300 reflect distinctly different types of attention, and thus we see different results for each ERP (Vossel et al., 2014). Amplitude, in this context, likely reflects attentional resources and the allocation of resources to focus. Latency likely is a reflection of the time it takes to process the stimuli more fully, and perhaps influences actions and motivational states down the road. If it is a harder task, or takes more time to process, we will see a longer latency. In our study, the P100 was larger in response to emotional stimuli in the health groups suggesting a more robust attention system for this part of the task. However, the P300 was not substantially different between groups. This suggests that while early attention differed and presumably was better

during early processing, the later processes were more equivalent between groups. However, there was an effect of a larger P300 in males compared to females, which could suggest a developmental difference where either sex or age impact later selection processes of emotional stimuli in our task. We cannot disentangle the effects of age and sex in this study. Moreover, in response to the non-emotional targets, P300 was larger and had longer latency for the CASA group, and for male participants overall compared to females. Taken together with the P100 results, this might suggest that the early attention processes to emotional stimuli are related to the later attention processes required for non-emotional target processing. And, that the relationship between early and late attention differs between groups in our study, and between age (and sex).

Early attention, as reflected by the P100 Distractors, showed similar results between our groups, indicating that bottom-up attention works similarly in healthy and CASA groups. For example, both CASA and control groups showed a significantly increased amplitude in response to negative emotional stimuli on both the POR and the Oz electrodes. Both groups are affected more by the emotional stimuli than the neutral stimuli. Healthy controls, however, showed an increased amplitude not only in regard to the negative emotional stimuli, but also overall compared to the CASA group. Although both groups showed the same pattern of response, the healthy controls showed larger amplitudes, which indicates that initial reactivity (assessed by the P100) to emotional stimuli is not necessarily maladaptive, but it is *how* one processes and deals with that emotional stimulus. This directing of attention is assessed by the P300, and differs between groups.

Adolescents as a whole are anecdotally known to be more emotionally reactive, but there appears to be a neural basis behind this phenomenon. At the sensitive period of puberty, there is important brain development in the areas of attention, emotion regulation, and also in the areas

of social interaction (Somerville, 2013; Blakemore et al., 2008; Ahmed et al., 2015). Being able to "fit in" and forge new peer relationships is very important, so understanding how your peers feel about you (i.e. emotional stimuli) is incredibly valuable. This importance placed on social interaction during adolescent years is suggested as one reason why greater attention is paid to emotional stimuli. This theme of more dedication of neural resources fits with the still-developing neural networks of younger participants still undergoing puberty (Spear, 2013). From synaptic pruning and myelation (Luna et al., 2001; Lewis et al., 2006), to development of the prefrontal cortex, adolescents have developmental differences (Monk et al., 2003). In our population, the sensitive period of puberty onset is towards the younger ages of our participants (11-17), hence why we often see age effects with the younger participants showing larger amplitudes. Further, the males in our study, although well age-matched between CASA and control groups, are slightly younger than the females as a whole. This, in conjunction with males and females showing different time periods for neural development, could contribute to the sex effects that we see with males showing a larger amplitude. As it is impossible to differentiate the effects of age and sex in our study, we can still note the importance of brain development on attention, with younger participants, and males, showing increased amplitude and longer latency to emotional stimuli.

It is important to note that on the P100 the CASA group also showed a larger response to negative stimuli as compared to neutral emotional stimuli, but the healthy controls' response was greater. This suggests that initial reactivity to emotional stimuli (as reflected by the P100) is not necessarily the mechanism for dysfunction, but it is the later, more top-down evaluation of these stimuli (as reflected by the P300) that is the mechanism for dysfunction in adolescents at-risk for psychological disorders. Although the healthy controls may be initially dedicating more

resources to emotional stimuli, they are able to later regulate their responses when it comes to the top-down, controlled attention as reflected by the P300.

Late attention, reflected by the P300, showed results in our target stimuli. Both groups showed an increased amplitude and a longer latency to the negative emotion stimuli. Although we saw similar results in the P100, they mean something entirely different in the P300. Initial, immediate responses to emotion were increased as seen in the P100, but in the P300 we see the same pattern. This means that in a more conscious, top-down process of attention, adolescent brains are choosing to dedicate more time to processing negative emotional stimuli.

This effect is larger in males, where in addition to showing increased activation to negative emotional stimuli, they are showing more time spent processing these stimuli than their female counterparts, as shown by longer latency. This longer processing of emotional stimuli indicates that there are slower attentional processes in males as compared to females. It is difficult to elucidate whether the males' neural systems are less developed due to lack of brain development and being slightly younger than the females (Blakemore, 2008; Gur & Gur, 2016), or whether this is a sex effect due to the male hormones' influence on neurotransmitters (Barenbaum & Beltz, 2011; Ernst, Romeo, & Anderson, 2009; Nelson et al., 2005; Sisk & Zehr, 2005), or something else entirely. However, it is clear that there are other factors affecting how the brain processes emotional stimuli, most notably: age, sex, and brain development, in addition to the Axis 1 psychological disorders we are studying.

Both CASA and control groups took longer (longer latency) to analyze and respond to the emotional stimuli, and are showing more neural resources dedicated to the processing of the emotional stimuli (larger amplitude). To some extent this is normal; as previously discussed, adolescents have reason for attributing more importance to emotional stimuli (Somerville, 2013).

Yet, when too much attention is paid to negative emotional stimuli, this can be a cause of attention and emotion regulation disorders, such as those we see in our at-risk CASA group (Shafer et al., 2012; Oliveira et al., 2013). Thus, our group effect, where we see a significant effect of latency and borderline significant effects overall, shows the clinical CASA group with a significantly longer latency and slightly larger amplitude on the P100 and P300. Because the CASA group is *over-focussing* on emotional stimuli, as reflected by our ERP results, this can draw vital attentional resources away from other competing parts of the brain. As the neural generators of the P100 and P300 include areas responsible for visual attention (Shigeto et al., 1998; Mulert et al., 2004), deficits in these areas could be a mechanism behind dysfunction in adolescents with ADHD, depression and anxiety.

Interestingly, the younger participants and the males showed similar patterns to the CASA group on the P300, with longer latency and larger amplitude. Thus, perhaps some of the same mechanisms of a lack of brain development could be owing to the dysfunction in attention. Although this may mean that participants with affective disorders are effectively analyzing stimuli with a less developed brain, this would be good news for treatment, as adolescent brains are malleable. In adolescents where the normal attenuation to emotional stimuli is accentuated, such as we saw in our P300 CASA group, they may be using too many of their attentional resources to process that stimulus, leaving little attentional resources left for the rest of the brain's functioning.

4.4 Limitations

Some of the limitations of this study could be addressed in future research, and other limitations were unfortunately outside of the control of the researchers on this study. Starting with the latter, this data was collected several years ago, and due to the constraints of the

COVID-19 pandemic, I could not collect more data. Since human data collection was suspended for the duration of the pandemic, we had to work with the data that we had. This, unfortunately, left us with a limited sample size.

One limitation is, then, sample size. There were 50 participants in total, and after removing those with excessive noise or missing data for the necessary electrodes, we had only 30-46 participants per ERP analysis. Although it was enough to run analysis, it was not enough participants to have the power to run all of the statistical analyses together (while separating the P100 and P300 of course). Thus, we had to run each of the 3 electrodes on the P100 individually, and each of the 3 electrodes on the P300 individually. This was accounted for in our adjusted p-value of significance of $p=0.017$ instead of $p=0.05$. Although we found significant results, it would have been beneficial to have more participants to increase power and reduce the possibility of Type 2 Error.

Next, this experiment was designed as an EEG and fMRI study. This style of experiment resulted in a limited of number of trials per participant, further limiting the amount of data we had to work with. This is primarily the reason that participants were excluded from analysis, as they were only removed if they had no usable data for the ERP of interest. Because the fMRI requires significant spacing between trials, due to simply taking longer to collect data as opposed to EEG, we could not have participants complete hundreds of trials. fMRI works by measuring the blood flow in the brain following a neural response, instead of measuring the electrical activity of the brain directly as is done with EEG. This blood flow that is measured is called the hemodynamic response. Because it takes time for the blood to travel to the area of the brain that is being activated, the hemodynamic response is much more delayed than the ERPs response we see on EEG. The task had to be identical in pacing for both the EEG and the fMRI, and since

fMRI takes longer, we had to slow down the spacing of the trials to accommodate. This means we cannot do as many trials as we could on an EEG-only task.

In addition, the groups had to be well-matched for age and sex, making data collection difficult again. Thankfully, our dataset was well matched for age and sex between the clinical CASA and control groups (CASA Females mean age 15.47, Males 14.65; Control Females mean age 15.11, males 14.2). However, unfortunately the males and females were not well matched. As you can see above, the males were slightly less than 1 year younger than the females on average, for both the CASA and control groups. So, although we could accurately compare the CASA and control groups, we had to deal with the confounding variable of sex. Further, due to the brain development patterns of males and females, the effect of sex was confounded with age. As females' brains develop earlier in some areas, perhaps due to hormonal puberty differences (Barenbaum & Beltz 2011; Somerville, 2013), the 1-year gap between males and females is effectively akin to multiple years of brain development. On the other hand, this age difference allowed us to find more evidence for the developmental differences between males and females at different ages, which has been interesting. Also, for this study we were not able to follow the participants over a long period of time. This means that we could not assess the effect age has within participants, and how they would perform on the same task at later stages of development.

Furthermore, it would have been interesting to assess the level of pubertal development of the participants to analyze the correlations between age and sex. For example, our dataset spanned from age 11 to 17, so there are likely some adolescents who have not started puberty, and some who are nearly finished. This has a large effect on brain development that does not necessarily correlate perfectly with age. Our effects of age indicate that there likely would be an effect of pubertal stage, but this is purely speculative as we did not collect that information. As

we saw in our study, there are significant effects of age where the older participants are more accurate on the task, as are the females. Most of the females are older than the males in this study, so they are also at a later stage of pubertal development, which can have a significant effect on the brain. We also saw younger participants, and males, show a larger amplitude on the P100 in response to emotional distractors. On the P300, we saw males with a longer latency and larger amplitude in response to the targets, indicating they both took more time to process the stimulus, as well as more neural resources (larger amplitude). These results are in line with a less developed brain in younger, male participants. Shafer et al. (2020) conducted a neuroimaging study with Diffusion Tensor Imaging (DTI) showing the differences between healthy controls and the CASA group. Many white matter tracts don't fully develop until long after puberty (Shafer 2020), so these psychological disorders could be having an impact on normal pubertal brain development. For example, they found that there is decreased white matter efficiency, or fractional anisotropy, in the CASA group, and impaired white matter structure overall (Shafer 2020). The Shafer et al. group (2020) found strong findings to support white matter structures being affected in adolescents with psychological disorders. As these white matter structures don't develop fully until well into adulthood, the effects of disorder during puberty could wreak disastrous consequences.

This data was also collected with the previous edition of the DSM, the DSM-IV. Although most things have remained the same in the current DSM, there are potentially some diagnoses that would have changed if the data were collected today. For example, there are increasing trends of females being diagnosed with ADHD (Davidovich et al., 2017) because the diagnostic criteria have changed to accurately describe the way it presents in females, and not just males. Similarly, depression and anxiety have historically been under-reported in males.

Since I did not do a diagnostic analysis, this limitation is not directly affecting my results. However, it is pertinent to note so that future studies can ensure that their participant pool is representative of the population and does not have any sex-related diagnostic bias.

4.5 Future Directions

As discussed above, the obvious next step would be to integrate this ERP data with fMRI data on attention. From previous research we know that the ventral and dorsal frontoparietal networks are involved in the P100 and P300, respectively, but it would be completely different to simultaneously image it in adolescents with EEG and fMRI. Similarly to Moore et al.'s 2019 paper, which was done on adults, using simultaneous EEG and fMRI with the same oddball paradigm would give us more information about how the adolescent brain functions. Being able to link certain areas of the brain, such as the ventrolateral prefrontal cortex (Moore et al., 2019) on fMRI to the same temporally-accurate regions using EEG enables a time-intensity-location relationship as opposed to simply the time-intensity (and less accurate location) relationship that is assessed with EEG latency and amplitude. Further, to expand on this previous research, doing simultaneous EEG-fMRI on clinical patients would be extremely insightful. There is a lot of research that needs to be done on adolescents, who have less research in certain fields as compared to adults. Unfortunately, a lot of this possible neuroimaging research has likely been disregarded due to prohibitive costs. fMRI is expensive and time consuming (which is also expensive), so not every researcher or lab is able to conduct fMRI neuroimaging studies, even though there are many practical applications such as described here. fMRI, however, can justify its use in many situations. For example, imaging adolescent brains over time is a practical use, as EEG cannot show structure, only function. fMRI can do both. Although we know P100 and P300 waveforms are different in adolescents with and without Axis 1 psychological disorders, we

cannot determine what brain structures are affected without imaging such as fMRI. Conducting a longitudinal EEG-fMRI study on adolescents would allow us to see the differences between a typically developing adolescent brain, an adolescent brain with a psychological disorder, as well as the physiological effects of certain treatments. Understanding the structural and functional neural circuits that underpin the P100 and P300 responses seen on EEG could allow for even more targeted treatment. In a field such as adolescent mental health, where research is drastically needed and has a potent effect, fMRI research is unquestionably worth the time and effort.

It would also be interesting to separate data into pre- and post- puberty adolescents (perhaps age 9-12 and 13-17, depending on the adolescent). This could give further insight into the effects of puberty on both brain development and mental health. Many psychological disorders appear in adolescence (Asselman et al., 2015), during or shortly after puberty, so there may be some interesting effects from a developmental neuropsychology standpoint.

Next, future research should encompass the pre- and post-effects of various treatment strategies for Axis 1 disorders. For example, the data collected for the present study included Mindfulness-Based Stress Reduction Therapy (MBSR). This therapy teaches adolescents meditation strategies as a way of coping with stress, and is thought to help with emotion regulation. This, in turn, could effectively help these adolescents with attention and impulsivity difficulties, as they would theoretically be less overwhelmed by the emotional stimuli. Being less affected by stressful, or emotional stimuli, would leave more neural resources for other important processes like sustained attention and decision making. Thus, doing the same ERP and behavioural analysis on this group of adolescents after they have completed MBSR training could show results hopefully resulting in patterns more similar to those of the control group. A study on the same participants used here showed that subjective measures of participants' mood,

self-control, inter-personal relationships and other measures all improved post-MBSR training, which is certainly in line with brain development to mirror that of the control group (Van Vliet et al., 2017). Further, a clinical trial done on the same participants used in this study showed that MBSR training resulted in significantly shorter times before adolescents were discharged, as well as improvements in adaptive skills and teacher ratings (Vohra et al., 2019). As the researchers mention, larger, more long-term studies of this sort would be beneficial. It would also be interesting to compare the outcome of this treatment alone with other treatments such as the typical pharmacological treatments, or perhaps traditional talk therapy.

Further, as research like the present study can be used to elucidate neural patterns that differ between clinical and healthy controls, this research could lead to better diagnostic and treatment practices for those with psychological disorders. ERPs provide a significant amount of information about brain development, and have "potential clinical utility for detecting early emerging vulnerabilities for...psychopathology" (Dickey et al., 2021). For example, if reliable patterns can be found on EEG and/or fMRI that differ between those with ADHD and healthy controls, it would incredibly easy to diagnose people with ADHD using neuroimaging, as opposed to our current method of surveys and talk therapy, which is subjective, and very slow and exhausting, and where people usually only come in to a psychologist when their life has been completely disrupted by said disorder. For depression and anxiety disorders, which are essentially exaggerated patterns of normal emotional reactions, neuroimaging could act as an objective metric, hopefully allowing for easier diagnosis and treatment. Nearly every health condition has an objective metric that is used to diagnose it, except for psychological disorders. There may be people who would show significant neural deficits on EEG or fMRI, but because of the subjectivity of the current diagnostic process, do not have a diagnosis and thus do not have

adequate treatment. An objective metric of diagnosis could give more validity, and perhaps dignity, to patients struggling with the symptoms of psychological conditions. The worry of whether your experience of symptoms would be “believed” is mitigated, and may result in more people seeking diagnosis when necessary, drastically improving patients’ quality of life. An interesting comment by the Dickey et al. group (2021) suggests that due to the abundance of new EEG research, it has "yet to be integrated" into current research and clinical practices, even though it could be incredibly useful. Similarly to diagnosis, neuroimaging could allow us to see improvements in patient mental health treatment from an objective neuroimaging standpoint. Just like how patients with epilepsy or other neurological disorders go in for regular EEGs to assess neural function and severity of the disorder, the same thing could potentially be done for patients with psychological disorders. Instead of solely monitoring patients with surveys and questionnaires that they may be unwilling to answer (i.e. “are you suicidal”), neuroimaging could give a much more accurate picture of the people who are struggling severely with depression, anxiety, or ADHD. These neuroimaging tests could be done before and after new treatments or medication are applied, or after dosing changes, and we would likely get more precise and objective information than simply asking patients how they feel. We could even use neuroimaging on people at high risk of depressive disorders, such as those where a parent committed suicide, as a pre-emptive screening method. Finally, every single person with a psychological disorder presents slightly differently. This leaves the healthcare system with the enormous challenge of helping those who need it – but not missing anybody, with potentially fatal consequences. Hopefully, the use of neuroimaging in diagnosis and treatment of psychological disorders would result in better analysis of the effectiveness of treatment, and less people slipping through the cracks.

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Appendix

ERP Analysis in Response to Distractors

This appendix section contains a selected statistical output of a repeated measures MANCOVA test on the P100 at the POR electrode, for the Distractors. Both the amplitude and latency were included in the analysis, with age and sex included as covariates. Box's m-test for equality of covariance and Levene's test of equality of error variance were included to test the assumption of equal variance, and to bolster the assumption of normality, along with Q-Q plots (not pictured). Mahalanobis Distance was calculated for outliers, to which there were no significant values. Groups were not random sampled, as they were specifically age- and sex-matched. Some descriptive statistics on number of participants in each group were also included. Of specific note were the significant effects of age as a covariate at $p=0.001$, and effects of experimental group with $p=0.005$. There were also significant effects of experimental condition, with $p<0.001$.

Within-Subjects Factors

Measure	ExperimentalCondition	Dependent Variable
Amplitude	1	PORAmplitudeNEGDistr
	2	PORAmplitudeNeu
Latency	1	PORLatencyNEGDistr
	2	PORLatencyNeu

Between-Subjects Factors

		N
Sex	F	22
	M	24
Exp. Group	CASA	30
	HC	16

Multivariate Tests[a]

Effect			Value	F	Hypothesis df	Error df	Sig.
Between Subjects	Intercept	Pillai's Trace	.329	9.818[b]	2.000	40.000	<.001
		Wilks' Lambda	.671	9.818[b]	2.000	40.000	<.001
		Hotelling's Trace	.491	9.818[b]	2.000	40.000	<.001
		Roy's Largest Root	.491	9.818[b]	2.000	40.000	<.001
	Age	Pillai's Trace	.280	7.771[b]	2.000	40.000	.001
		Wilks' Lambda	.720	7.771[b]	2.000	40.000	.001
		Hotelling's Trace	.389	7.771[b]	2.000	40.000	.001
		Roy's Largest Root	.389	7.771[b]	2.000	40.000	.001
	Sex	Pillai's Trace	.137	3.172[b]	2.000	40.000	.053
		Wilks' Lambda	.863	3.172[b]	2.000	40.000	.053
		Hotelling's Trace	.159	3.172[b]	2.000	40.000	.053
		Roy's Largest Root	.159	3.172[b]	2.000	40.000	.053
	Exp.Group	Pillai's Trace	.234	6.118[b]	2.000	40.000	.005
		Wilks' Lambda	.766	6.118[b]	2.000	40.000	.005
		Hotelling's Trace	.306	6.118[b]	2.000	40.000	.005
		Roy's Largest Root	.306	6.118[b]	2.000	40.000	.005
	Sex * Exp.Group	Pillai's Trace	.003	.067[b]	2.000	40.000	.935
		Wilks' Lambda	.997	.067[b]	2.000	40.000	.935
		Hotelling's Trace	.003	.067[b]	2.000	40.000	.935
		Roy's Largest Root	.003	.067[b]	2.000	40.000	.935
Within Subjects	ExperimentalCondition	Pillai's Trace	.357	11.118[b]	2.000	40.000	<.001
		Wilks' Lambda	.643	11.118[b]	2.000	40.000	<.001
		Hotelling's Trace	.556	11.118[b]	2.000	40.000	<.001
		Roy's Largest Root	.556	11.118[b]	2.000	40.000	<.001
	ExperimentalCondition * Age	Pillai's Trace	.307	8.866[b]	2.000	40.000	<.001
		Wilks' Lambda	.693	8.866[b]	2.000	40.000	<.001
		Hotelling's Trace	.443	8.866[b]	2.000	40.000	<.001
		Roy's Largest Root	.443	8.866[b]	2.000	40.000	<.001
	ExperimentalCondition * Sex	Pillai's Trace	.058	1.236[b]	2.000	40.000	.301
		Wilks' Lambda	.942	1.236[b]	2.000	40.000	.301
		Hotelling's Trace	.062	1.236[b]	2.000	40.000	.301
		Roy's Largest Root	.062	1.236[b]	2.000	40.000	.301
	ExperimentalCondition * Exp.Group	Pillai's Trace	.046	.968[b]	2.000	40.000	.389
		Wilks' Lambda	.954	.968[b]	2.000	40.000	.389
		Hotelling's Trace	.048	.968[b]	2.000	40.000	.389
		Roy's Largest Root	.048	.968[b]	2.000	40.000	.389
	ExperimentalCondition * Sex * Exp.Group	Pillai's Trace	.010	.193[b]	2.000	40.000	.825
		Wilks' Lambda	.990	.193[b]	2.000	40.000	.825
		Hotelling's Trace	.010	.193[b]	2.000	40.000	.825
		Roy's Largest Root	.010	.193[b]	2.000	40.000	.825

a. Design: Intercept + Age + Sex + Exp.Group + Sex * Exp.Group
Within Subjects Design: ExperimentalCondition

b. Exact statistic

Levene's Test of Equality of Error Variances[a]

	F	df1	df2	Sig.
PORAmplitudeNEGDistr	1.231	3	42	.311
POR Amplitude (Neu)	.651	3	42	.587
PORLatencyNEGDistr	1.746	3	42	.172
POR Latency (Neu)	2.131	3	42	.111

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Age + Sex + Exp.Group + Sex * Exp.Group
Within Subjects Design: ExperimentalCondition

Box's Test of Equality of Covariance Matrices[a]

Box's M	72.492
F	1.867
df1	30
df2	1607.731
Sig.	.003

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Age + Sex + Exp.Group + Sex * Exp. Group
Within Subjects Design: Experimental Condition

P100 Oz ERP Analysis in Response to Distractors

This appendix contains a selected statistical output of a repeated measures MANCOVA test on the P100 Oz electrode, for the Distractors condition. Both amplitude and latency were included in the analysis, with age and sex included as covariates. Box's m-test for equality of covariance and Levene's test of equality of error variance were included to test the assumption of equal variance, and to bolster the assumption of normality, along with Q-Q plots (not pictured). Mahalanobis Distance was calculated for outliers, to which there were no significant values. Groups were not random sampled, as they were specifically age- and sex-matched. Some descriptive statistics on number of participants in each group were also included. Of specific note were the significant effects of age as a covariate with p=0.005, effects of sex with p=0.15 and borderline effects of experimental group with p=0.037. There were also significant effects of experimental condition, with p=0.006.

Within-Subjects Factors

Measure	ExperimentalCondition	Dependent Variable
Amplitude	1	OzAmplitudeNEGDistr
	2	OzAmplitudeNeu
Latency	1	OzLatencyNEGDistr
	2	OzLatencyNeu

Between-Subjects Factors

		N
Sex	F	22
	M	23
Exp. Group	CASA	29
	HC	16

Multivariate Tests[a]

Effect			Value	F	Hypothesis df	Error df	Sig.
Between Subjects	Intercept	Pillai's Trace	.457	16.392[b]	2.000	39.000	<.001
		Wilks' Lambda	.543	16.392[b]	2.000	39.000	<.001
		Hotelling's Trace	.841	16.392[b]	2.000	39.000	<.001
		Roy's Largest Root	.841	16.392[b]	2.000	39.000	<.001
	Age	Pillai's Trace	.237	6.061[b]	2.000	39.000	.005
		Wilks' Lambda	.763	6.061[b]	2.000	39.000	.005
		Hotelling's Trace	.311	6.061[b]	2.000	39.000	.005
		Roy's Largest Root	.311	6.061[b]	2.000	39.000	.005
	Sex	Pillai's Trace	.193	4.663[b]	2.000	39.000	.015
		Wilks' Lambda	.807	4.663[b]	2.000	39.000	.015
		Hotelling's Trace	.239	4.663[b]	2.000	39.000	.015
		Roy's Largest Root	.239	4.663[b]	2.000	39.000	.015
	Exp.Group	Pillai's Trace	.156	3.600[b]	2.000	39.000	.037
		Wilks' Lambda	.844	3.600[b]	2.000	39.000	.037
		Hotelling's Trace	.185	3.600[b]	2.000	39.000	.037
		Roy's Largest Root	.185	3.600[b]	2.000	39.000	.037
	Sex * Exp.Group	Pillai's Trace	.043	.874[b]	2.000	39.000	.425
		Wilks' Lambda	.957	.874[b]	2.000	39.000	.425
		Hotelling's Trace	.045	.874[b]	2.000	39.000	.425
		Roy's Largest Root	.045	.874[b]	2.000	39.000	.425
Within Subjects	ExperimentalCondition	Pillai's Trace	.230	5.816[b]	2.000	39.000	.006
		Wilks' Lambda	.770	5.816[b]	2.000	39.000	.006
		Hotelling's Trace	.298	5.816[b]	2.000	39.000	.006
		Roy's Largest Root	.298	5.816[b]	2.000	39.000	.006
	ExperimentalCondition * Age	Pillai's Trace	.206	5.065[b]	2.000	39.000	.011
		Wilks' Lambda	.794	5.065[b]	2.000	39.000	.011
		Hotelling's Trace	.260	5.065[b]	2.000	39.000	.011
		Roy's Largest Root	.260	5.065[b]	2.000	39.000	.011
	ExperimentalCondition * Sex	Pillai's Trace	.023	.469[b]	2.000	39.000	.629
		Wilks' Lambda	.977	.469[b]	2.000	39.000	.629
		Hotelling's Trace	.024	.469[b]	2.000	39.000	.629
		Roy's Largest Root	.024	.469[b]	2.000	39.000	.629
	ExperimentalCondition * Exp.Group	Pillai's Trace	.022	.444[b]	2.000	39.000	.645
		Wilks' Lambda	.978	.444[b]	2.000	39.000	.645
		Hotelling's Trace	.023	.444[b]	2.000	39.000	.645
		Roy's Largest Root	.023	.444[b]	2.000	39.000	.645
	ExperimentalCondition * Sex * Exp.Group	Pillai's Trace	.000	.004[b]	2.000	39.000	.996
		Wilks' Lambda	1.000	.004[b]	2.000	39.000	.996
		Hotelling's Trace	.000	.004[b]	2.000	39.000	.996
		Roy's Largest Root	.000	.004[b]	2.000	39.000	.996

a. Design: Intercept + Age + Sex + Exp.Group + Sex * Exp.Group
Within Subjects Design: ExperimentalCondition

b. Exact statistic

Levene's Test of Equality of Error Variances[a]

	F	df1	df2	Sig.
OzAmplitudeNEGDistr	.400	3	41	.754
Oz Amplitude (Neu)	.402	3	41	.752
OzLatencyNEGDistr	.217	3	41	.884
Oz Latency (Neu)	2.338	3	41	.088

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Age + Sex + Exp.Group + Sex * Exp. Group
Within Subjects Design: ExperimentalCondition

Box's Test of Equality of Covariance Matrices[a]

Box's M	81.865
F	2.105
df1	30
df2	1624.199
Sig.	<.001

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Age + Sex + Exp.Group + Sex * Exp. Group
Within Subjects Design: Experimental Condition

P300 PR ERP Analysis in Response to Distractors

This appendix contains a selected statistical output of a repeated measures MANCOVA test on the P300 PR electrode, for the Distractors condition. Both amplitude and latency were included in the analysis, with age and sex included as covariates. Age was removed because it had no significant effects; analysis was re-run without it. Box's m-test for equality of covariance and Levene's test of equality of error variance are included to test the assumption of equal variance, and to bolster the assumption of normality, along with Q-Q plots (not pictured). Mahalanobis Distance was calculated for outliers, to which there were no significant values. Groups were not random sampled, as they were specifically age- and sex-matched. Some descriptive statistics on number of participants in each group were also included. Of specific note were the significant effects of sex as a covariate with $p=0.003$. There were also significant effects of experimental condition with $p<0.001$.

Within-Subjects Factors

Measure	ExperimentalCondition	Dependent Variable
Amplitude	1	PRAmplitudeNEGDistr
	2	PRAmplitudeNeu
Latency	1	PRLatencyNEGDistr
	2	PRLatencyNeu

Between-Subjects Factors

		N
Exp. Group	CASA	27
	HC	14
Sex	F	19
	M	22

Multivariate Tests[a]

Effect			Value	F	Hypothesis df	Error df	Sig.
Between Subjects	Intercept	Pillai's Trace	.980	877.196[b]	2.000	36.000	<.001
		Wilks' Lambda	.020	877.196[b]	2.000	36.000	<.001
		Hotelling's Trace	48.733	877.196[b]	2.000	36.000	<.001
		Roy's Largest Root	48.733	877.196[b]	2.000	36.000	<.001
	Exp.Group	Pillai's Trace	.051	.960[b]	2.000	36.000	.393
		Wilks' Lambda	.949	.960[b]	2.000	36.000	.393
		Hotelling's Trace	.053	.960[b]	2.000	36.000	.393
		Roy's Largest Root	.053	.960[b]	2.000	36.000	.393
	Sex	Pillai's Trace	.279	6.974[b]	2.000	36.000	.003
		Wilks' Lambda	.721	6.974[b]	2.000	36.000	.003
		Hotelling's Trace	.387	6.974[b]	2.000	36.000	.003
		Roy's Largest Root	.387	6.974[b]	2.000	36.000	.003
	Exp.Group * Sex	Pillai's Trace	.038	.708[b]	2.000	36.000	.499
		Wilks' Lambda	.962	.708[b]	2.000	36.000	.499
		Hotelling's Trace	.039	.708[b]	2.000	36.000	.499
		Roy's Largest Root	.039	.708[b]	2.000	36.000	.499
Within Subjects	ExperimentalCondition	Pillai's Trace	.907	176.006[b]	2.000	36.000	<.001
		Wilks' Lambda	.093	176.006[b]	2.000	36.000	<.001
		Hotelling's Trace	9.778	176.006[b]	2.000	36.000	<.001
		Roy's Largest Root	9.778	176.006[b]	2.000	36.000	<.001
	ExperimentalCondition * Exp.Group	Pillai's Trace	.047	.894[b]	2.000	36.000	.418
		Wilks' Lambda	.953	.894[b]	2.000	36.000	.418
		Hotelling's Trace	.050	.894[b]	2.000	36.000	.418
		Roy's Largest Root	.050	.894[b]	2.000	36.000	.418
	ExperimentalCondition * Sex	Pillai's Trace	.202	4.570[b]	2.000	36.000	.017
		Wilks' Lambda	.798	4.570[b]	2.000	36.000	.017
		Hotelling's Trace	.254	4.570[b]	2.000	36.000	.017
		Roy's Largest Root	.254	4.570[b]	2.000	36.000	.017
	ExperimentalCondition * Exp.Group * Sex	Pillai's Trace	.014	.257[b]	2.000	36.000	.775
		Wilks' Lambda	.986	.257[b]	2.000	36.000	.775
		Hotelling's Trace	.014	.257[b]	2.000	36.000	.775
		Roy's Largest Root	.014	.257[b]	2.000	36.000	.775

a. Design: Intercept + Exp.Group + Sex + Exp.Group * Sex
Within Subjects Design: ExperimentalCondition

b. Exact statistic

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
PRAmplitudeNEGDistr	Based on Mean	2.365	3	37	.087
	Based on Median	2.033	3	37	.126
	Based on Median and with adjusted df	2.033	3	30.226	.130
	Based on trimmed mean	2.311	3	37	.092
PR Amplitude (Neu)	Based on Mean	5.525	3	37	.003
	Based on Median	2.420	3	37	.081
	Based on Median and with adjusted df	2.420	3	18.728	.098
	Based on trimmed mean	4.448	3	37	.009
PRLatencyNEGDistr	Based on Mean	5.710	3	37	.003
	Based on Median	2.408	3	37	.083
	Based on Median and with adjusted df	2.408	3	26.756	.089
	Based on trimmed mean	5.439	3	37	.003
PR Latency (Neu)	Based on Mean	2.568	3	37	.069
	Based on Median	1.679	3	37	.188
	Based on Median and with adjusted df	1.679	3	26.147	.196
	Based on trimmed mean	2.536	3	37	.072

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Exp.Group + Sex + Exp.Group * Sex
Within Subjects Design: ExperimentalCondition

Box's Test of Equality of Covariance Matrices[a]

Box's M	60.497
F	1.512
df1	30
df2	1525.277
Sig.	.038

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Exp.Group + Sex + Exp. Group * Sex Within Subjects Design: Experimental Condition

P300 PR ERP Analysis in Response to Targets

This appendix contains a selected statistical output of a repeated measures MANCOVA test on the P300 PR electrode, for the Targets condition. Both amplitude and latency were included in the analysis, with age and sex included as covariates. Age and Sex were removed as covariates because they had no significant effects; analysis was re-run without them. Box's m-test for equality of covariance and Levene's test of equality of error variance were included to test the assumption of equal variance, and to bolster the assumption of normality, along with Q-Q plots (not pictured). Mahalanobis Distance was calculated for outliers, to which there were no significant values. Groups were not random sampled, as they were specifically age- and sex-matched. Some descriptive statistics on number of participants in each group are also included. Of specific note were the borderline significant effects of Experimental Group with $p=0.033$. There were also significant effects of experimental condition, with $p<0.001$.

Within-Subjects Factors

Measure	ExperimentalCondition	Dependent Variable
Amplitude	1	PRAmplitudeTATarget
	2	PRAmplitudeTANeg
Latency	1	PRLatencyTATarget
	2	PRLatencyTANeg

Between-Subjects Factors

		N
Exp. Group	CASA	20
	HC	10

Multivariate Tests[a]

Effect			Value	F	Hypothesis df	Error df	Sig.
Between Subjects	Intercept	Pillai's Trace	.993	2050.015[b]	2.000	27.000	<.001
		Wilks' Lambda	.007	2050.015[b]	2.000	27.000	<.001
		Hotelling's Trace	151.853	2050.015[b]	2.000	27.000	<.001
		Roy's Largest Root	151.853	2050.015[b]	2.000	27.000	<.001
	Exp.Group	Pillai's Trace	.224	3.896[b]	2.000	27.000	.033
		Wilks' Lambda	.776	3.896[b]	2.000	27.000	.033
		Hotelling's Trace	.289	3.896[b]	2.000	27.000	.033
		Roy's Largest Root	.289	3.896[b]	2.000	27.000	.033
Within Subjects	ExperimentalCondition	Pillai's Trace	.930	178.436[b]	2.000	27.000	<.001
		Wilks' Lambda	.070	178.436[b]	2.000	27.000	<.001
		Hotelling's Trace	13.217	178.436[b]	2.000	27.000	<.001
		Roy's Largest Root	13.217	178.436[b]	2.000	27.000	<.001
	ExperimentalCondition * Exp.Group	Pillai's Trace	.021	.294[b]	2.000	27.000	.748
		Wilks' Lambda	.979	.294[b]	2.000	27.000	.748
		Hotelling's Trace	.022	.294[b]	2.000	27.000	.748
		Roy's Largest Root	.022	.294[b]	2.000	27.000	.748

a. Design: Intercept + Exp.Group
Within Subjects Design: ExperimentalCondition

b. Exact statistic

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
PR Amplitude (TATarget)	Based on Mean	3.368	1	28	.077
	Based on Median	2.707	1	28	.111
	Based on Median and with adjusted df	2.707	1	26.710	.112
	Based on trimmed mean	3.370	1	28	.077
PRAmplitudeTANeg	Based on Mean	1.683	1	28	.205
	Based on Median	1.739	1	28	.198
	Based on Median and with adjusted df	1.739	1	26.662	.199
	Based on trimmed mean	1.719	1	28	.201
PR Latency (TATarget)	Based on Mean	.474	1	28	.497
	Based on Median	.449	1	28	.508
	Based on Median and with adjusted df	.449	1	27.037	.508
	Based on trimmed mean	.511	1	28	.481
PRLatencyTANeg	Based on Mean	.238	1	28	.630
	Based on Median	.234	1	28	.632
	Based on Median and with adjusted df	.234	1	23.445	.633
	Based on trimmed mean	.235	1	28	.632

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Exp.Group
Within Subjects Design: ExperimentalCondition

Box's Test of Equality of Covariance Matrices[a]

Box's M	19.392
F	1.571
df1	10
df2	1529.481
Sig.	.110

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Exp.Group Within Subjects Design: Experimental Condition

P300 PL ERP Analysis in Response to Targets

This appendix contains a selected statistical output of a repeated measures MANCOVA test on the P300 PL electrode, for the Targets condition. Both amplitude and latency were included in the analysis, with age and sex included as covariates. Age was removed as a covariate because it had no significant effects; analysis was re-run without it. Box's m-test for equality of covariance and Levene's test of equality of error variance were included to test the assumption of equal variance, and to bolster the assumption of normality, along with Q-Q plots (not pictured). Mahalanobis Distance was calculated for outliers, to which there were no significant values. Groups were not random sampled, as they were specifically age- and sex-matched. Some descriptive statistics on number of participants in each group were also included. Of specific note were the borderline significant effects of Sex, when separate analyses were run on Latency only, with $p=0.031$. There were also significant effects of experimental condition, with $p<0.001$.

Within-Subjects Factors

Measure	ExperimentalCondition	Dependent Variable
Amplitude	1	PLAmplitudeTATarget
	2	PLAmplitudeTANeg
Latency	1	PLLatencyTATarget
	2	PLLatencyTANeg

Between-Subjects Factors

		N
Exp. Group	CASA	19
	HC	12
Sex	F	15
	M	16

Multivariate Tests[a]

Effect			Value	F	Hypothesis df	Error df	Sig.
Between Subjects	Intercept	Pillai's Trace	.991	1492.519[b]	2.000	26.000	<.001
		Wilks' Lambda	.009	1492.519[b]	2.000	26.000	<.001
		Hotelling's Trace	114.809	1492.519[b]	2.000	26.000	<.001
		Roy's Largest Root	114.809	1492.519[b]	2.000	26.000	<.001
	Exp.Group	Pillai's Trace	.046	.622[b]	2.000	26.000	.545
		Wilks' Lambda	.954	.622[b]	2.000	26.000	.545
		Hotelling's Trace	.048	.622[b]	2.000	26.000	.545
		Roy's Largest Root	.048	.622[b]	2.000	26.000	.545
	Sex	Pillai's Trace	.162	2.519[b]	2.000	26.000	.100
		Wilks' Lambda	.838	2.519[b]	2.000	26.000	.100
		Hotelling's Trace	.194	2.519[b]	2.000	26.000	.100
		Roy's Largest Root	.194	2.519[b]	2.000	26.000	.100
	Exp.Group * Sex	Pillai's Trace	.071	.986[b]	2.000	26.000	.387
		Wilks' Lambda	.929	.986[b]	2.000	26.000	.387
		Hotelling's Trace	.076	.986[b]	2.000	26.000	.387
		Roy's Largest Root	.076	.986[b]	2.000	26.000	.387
Within Subjects	ExperimentalCondition	Pillai's Trace	.768	43.080[b]	2.000	26.000	<.001
		Wilks' Lambda	.232	43.080[b]	2.000	26.000	<.001
		Hotelling's Trace	3.314	43.080[b]	2.000	26.000	<.001
		Roy's Largest Root	3.314	43.080[b]	2.000	26.000	<.001
	ExperimentalCondition * Exp.Group	Pillai's Trace	.113	1.648[b]	2.000	26.000	.212
		Wilks' Lambda	.887	1.648[b]	2.000	26.000	.212
		Hotelling's Trace	.127	1.648[b]	2.000	26.000	.212
		Roy's Largest Root	.127	1.648[b]	2.000	26.000	.212
	ExperimentalCondition * Sex	Pillai's Trace	.014	.178[b]	2.000	26.000	.838
		Wilks' Lambda	.986	.178[b]	2.000	26.000	.838
		Hotelling's Trace	.014	.178[b]	2.000	26.000	.838
		Roy's Largest Root	.014	.178[b]	2.000	26.000	.838
	ExperimentalCondition * Exp.Group * Sex	Pillai's Trace	.053	.720[b]	2.000	26.000	.496
		Wilks' Lambda	.947	.720[b]	2.000	26.000	.496
		Hotelling's Trace	.055	.720[b]	2.000	26.000	.496
		Roy's Largest Root	.055	.720[b]	2.000	26.000	.496

a. Design: Intercept + Exp.Group + Sex + Exp.Group * Sex
Within Subjects Design: ExperimentalCondition

b. Exact statistic

Tests of Between-Subjects Effects

Transformed Variable: Average

Source	Measure	Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Amplitude	5392.251	1	5392.251	110.338	<.001
	Latency	15145419.7	1	15145419.7	3094.976	<.001
Exp.Group	Amplitude	12.767	1	12.767	.261	.613
	Latency	4207.999	1	4207.999	.860	.362
Sex	Amplitude	1.760	1	1.760	.036	.851
	Latency	25489.291	1	25489.291	5.209	.031
Exp.Group * Sex	Amplitude	.084	1	.084	.002	.967
	Latency	9704.347	1	9704.347	1.983	.170
Error	Amplitude	1319.503	27	48.870		
	Latency	132125.847	27	4893.550		

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
PL Amplitude (TATarget)	Based on Mean	.541	3	27	.658
	Based on Median	.323	3	27	.809
	Based on Median and with adjusted df	.323	3	21.039	.809
	Based on trimmed mean	.510	3	27	.679
PLAmplitudeTANeg	Based on Mean	.994	3	27	.411
	Based on Median	.645	3	27	.593
	Based on Median and with adjusted df	.645	3	13.443	.599
	Based on trimmed mean	.942	3	27	.434
PL Latency (TATarget)	Based on Mean	1.063	3	27	.381
	Based on Median	.987	3	27	.413
	Based on Median and with adjusted df	.987	3	26.487	.414
	Based on trimmed mean	1.067	3	27	.380
PLLatencyTANeg	Based on Mean	.906	3	27	.451
	Based on Median	.475	3	27	.702
	Based on Median and with adjusted df	.475	3	22.116	.703
	Based on trimmed mean	.834	3	27	.487

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Exp.Group + Sex + Exp.Group * Sex
Within Subjects Design: ExperimentalCondition

Box's Test of Equality of Covariance Matrices[a]

Box's M	39.584
F	.889
df1	30
df2	1036.898
Sig.	.640

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Exp.Group + Sex + Exp. Group * Sex Within Subjects Design: Experimental Condition

P300 Pz ERP Analysis in Response to Targets

This appendix contains a selected statistical output of a repeated measures MANCOVA test on the P300 Pz electrode, for the Targets condition. Both amplitude and latency were included in the analysis, with age and sex included as covariates. Age was removed as a covariate because it had no significant effects; analysis was re-run without it. Box's m-test for equality of covariance and Levene's test of equality of error variance were included to test the assumption of equal variance, and to bolster the assumption of normality, along with Q-Q plots (not pictured). Mahalanobis Distance was calculated for outliers, to which there were no significant values. Groups were not random sampled, as they were specifically age- and sex-matched. Some descriptive statistics on number of participants in each group were also included. Of specific note were the significant effects of Sex, with $p=0.002$. There were also significant effects of experimental condition with $p<0.001$.

Within-Subjects Factors

Measure	ExperimentalCondition	Dependent Variable
Amplitude	1	PzAmplitudeTATarget
	2	PzAmplitudeTANeg
Latency	1	PzLatencyTATarget
	2	PzLatencyTANeg

Between-Subjects Factors

		N
Exp. Group	CASA	23
	HC	14
Sex	F	18
	M	19

Multivariate Tests[a]

Effect			Value	F	Hypothesis df	Error df	Sig.
Between Subjects	Intercept	Pillai's Trace	.988	1313.108[b]	2.000	32.000	<.001
		Wilks' Lambda	.012	1313.108[b]	2.000	32.000	<.001
		Hotelling's Trace	82.069	1313.108[b]	2.000	32.000	<.001
		Roy's Largest Root	82.069	1313.108[b]	2.000	32.000	<.001
	Exp.Group	Pillai's Trace	.001	.016[b]	2.000	32.000	.984
		Wilks' Lambda	.999	.016[b]	2.000	32.000	.984
		Hotelling's Trace	.001	.016[b]	2.000	32.000	.984
		Roy's Largest Root	.001	.016[b]	2.000	32.000	.984
	Sex	Pillai's Trace	.315	7.355[b]	2.000	32.000	.002
		Wilks' Lambda	.685	7.355[b]	2.000	32.000	.002
		Hotelling's Trace	.460	7.355[b]	2.000	32.000	.002
		Roy's Largest Root	.460	7.355[b]	2.000	32.000	.002
	Exp.Group * Sex	Pillai's Trace	.058	.991[b]	2.000	32.000	.382
		Wilks' Lambda	.942	.991[b]	2.000	32.000	.382
		Hotelling's Trace	.062	.991[b]	2.000	32.000	.382
		Roy's Largest Root	.062	.991[b]	2.000	32.000	.382
Within Subjects	ExperimentalCondition	Pillai's Trace	.838	83.022[b]	2.000	32.000	<.001
		Wilks' Lambda	.162	83.022[b]	2.000	32.000	<.001
		Hotelling's Trace	5.189	83.022[b]	2.000	32.000	<.001
		Roy's Largest Root	5.189	83.022[b]	2.000	32.000	<.001
	ExperimentalCondition * Exp.Group	Pillai's Trace	.049	.820[b]	2.000	32.000	.449
		Wilks' Lambda	.951	.820[b]	2.000	32.000	.449
		Hotelling's Trace	.051	.820[b]	2.000	32.000	.449
		Roy's Largest Root	.051	.820[b]	2.000	32.000	.449
	ExperimentalCondition * Sex	Pillai's Trace	.139	2.573[b]	2.000	32.000	.092
		Wilks' Lambda	.861	2.573[b]	2.000	32.000	.092
		Hotelling's Trace	.161	2.573[b]	2.000	32.000	.092
		Roy's Largest Root	.161	2.573[b]	2.000	32.000	.092
	ExperimentalCondition * Exp.Group * Sex	Pillai's Trace	.173	3.352[b]	2.000	32.000	.048
		Wilks' Lambda	.827	3.352[b]	2.000	32.000	.048
		Hotelling's Trace	.209	3.352[b]	2.000	32.000	.048
		Roy's Largest Root	.209	3.352[b]	2.000	32.000	.048

Levene's Test of Equality of Error Variances[a]

		Levene Statistic	df1	df2	Sig.
Pz Amplitude (TATarget)	Based on Mean	.957	3	33	.425
	Based on Median	.657	3	33	.585
	Based on Median and with adjusted df	.657	3	30.895	.585
	Based on trimmed mean	.929	3	33	.438
PzAmplitudeTANeg	Based on Mean	.501	3	33	.685
	Based on Median	.123	3	33	.946
	Based on Median and with adjusted df	.123	3	27.449	.946
	Based on trimmed mean	.414	3	33	.744
Pz Latency (TATarget)	Based on Mean	1.605	3	33	.207
	Based on Median	.870	3	33	.467
	Based on Median and with adjusted df	.870	3	25.078	.470
	Based on trimmed mean	1.617	3	33	.204
PzLatencyTANeg	Based on Mean	.603	3	33	.617
	Based on Median	.355	3	33	.786
	Based on Median and with adjusted df	.355	3	25.344	.786
	Based on trimmed mean	.546	3	33	.655

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.

a. Design: Intercept + Exp.Group + Sex + Exp.Group * Sex
Within Subjects Design: ExperimentalCondition

Box's Test of Equality of Covariance Matrices[a]

Box's M	49.929
F	1.182
df1	30
df2	1047.709
Sig.	.231

Tests the null hypothesis that the observed covariance matrices of the dependent variables are equal across groups.

a. Design: Intercept + Exp.Group + Sex + Exp. Group * Sex
Within Subjects Design: Experimental Condition