Emotion regulation and the behavioral and neural correlates of adolescents with mental health disorders

by

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ABSTRACT

In this study, I looked at how a clinical group emotionally processes and regulates when presented with an emotional oddball task. A clinical group, ages 12 to 17, with mental health disorders including attention deficit hyperactivity disorder (ADHD), conduct disorder (CD) and affective disorders, was compared to a healthy control group, 12 to 17 years old, in order to assess behavioral and neuropsychophysiological differences in response to emotional information presented. Using a modified emotional oddball paradigm, which contained emotional pictures (i.e., distracter type: fear, neutral, sad) and non-emotional pictures called targets (i.e., target type: target-after-fear, target-after-neutral, target-after-sad, and target-after-target), participants were asked to respond with a right hand button press to targets (i.e., circles) and a left hand button press for all other stimuli. Reaction time (i.e., RT) was recorded for all participants. Event-related potentials (ERPs) were also recorded via a high density 256-channel recording system. Statistical comparisons were made between the two groups for behavioral (i.e., RT) data , 42 participants for the clinical group and 17 participants for the healthy control group, and ERP (i.e., P300) data, 35 participants for the clinical group and 13 participants for the healthy control group. Both clinical and control groups responded slower to fear distracters than neutral or sad distracters. There was no significant differences between the clinical and control groups for RT or ERPs (i.e., P300) for target types. We suggest that this study has the potential to elucidate emotion processing and emotion regulation information for adolescents with clinical disorders, but possibly due to the large variability of mental health disorders, the differences were not made apparent statistically.

KEYWORDS: Emotion regulation, emotional oddball paradigm, affective disorder, ADHD, CD, ERPs, P300

PREFACE

This thesis was the result of a collaboration within the Singhal Lab in the Department of Psychology at the University of Alberta. Andrea T. Shafer came up with the original idea and did the recruiting from CASA. The research project, of which this thesis is a part, received approval from the University of Alberta Research Ethics Board, [Non-drug approaches to support health in youth, Pro: 00012772, October 20, 2010]. Andrea ran the experiment and collected ERP data for all participants.

I was responsible for data cleaning, analysis and writing the present manuscript.

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CHAPTER I - Introduction

1.1 Emotion Regulation

Emotion regulation is a goal-oriented focus, that is either implicit or explicit, that recruits one or more brain processes to facilitate emotion generation in humans (Gross, Sheppes, & Urry, 2011; Gross, Sheppes, & Urry, 2011a, b; Gyurak, Gross, & Etkin, 2011; Sheppes, Suri, & Gross, 2015). Gross and Thompson (2007) suggest that emotion regulation is the "initiation, maintenance, and modification" of emotions in both unconscious and conscious processes (Webb, Gallo, Miles, Gollwitzer, & Sheeran, 2012). Gross and Jazaieri (2014) indicate that emotion regulation is also affected by the intensity, frequency and types of emotion. Emotion regulation may be intrinsic, when an individual regulates their own emotions, or extrinsic, when an individual regulates another person's emotions (Gross & Jazaieri, 2014). There are three common factors found in effective regulation: awareness, goals, and strategies (Gross & Jazaieri, 2014). Gross's process model has been highly influential in the study of emotion regulation, providing a theoretical model of how emotion regulation works practically (Gross, 1998). To emotionally regulate, individuals must be able to decide when to regulate, which strategy is best to use in a given context, and how to implement the strategy (Webb, Miles, & Sheeran, 2012). Gross's process model separates emotion regulation into five processes in temporal order, from perceiving an emotional stimulus to emotion generation (Gross, 1998). The five regulation processes are: situation selection, situation modification, attentional deployment, cognitive change, and response modulation (Gross, 1998). Emotion regulation can be further broken down into whether or not the individual can perceive the need to regulate, choose an effective strategy for the context and apply it to the given situation (Bonanno & Burton, 2013; Sheppes & Gross, 2012; Sheppes, Scheibe, Suri, Gross, 2011; Webb et al., 2012). Sheppes and Levin (2013) found

that when choosing between two emotional regulation strategies, such as distraction and reappraisal, those individuals that could pick the best strategy for the situation had better emotion regulation outcomes. They determined that regulatory choice is moderated by motivational factors, such as long-term goals, financial rewards, strategy complexity, as well as cognitive factors (Sheppes & Levin, 2013; Sheppes et al., 2011). Context plays an important role in emotion regulation. Researchers have found that the ability to perceive subtle changes in the environment determines whether the individual can match the appropriate emotion to its context (Aldao, 2013; Bonanno & Burton, 2013; Burns, Isbell, & Tyler, 2008; Carver & Connor-Smith, 2010; Dixon-Gordon, Aldao, & Reyes, 2015; Mauss & Butler, 2010; Troy, Shallcross, & Mauss, 2013). Current research suggests that the most efficacious way to regulate emotion is for the individual to be flexible, which is the ability to change strategies as the emotional context changes (Bonanno, 2005; Kashdan & Rottenberg, 2010; Opitz, Gross, & Urry, 2012; Troy & Mauss, 2011). Flexibility for healthy emotion regulation means that individuals have the ability to both up-regulate (i.e., express) and down-regulate (i.e., suppress) emotion as demanded by the situational context (Bonanno, 2005; Bonanno & Burton, 2013; Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Gross & Thompson, 2007; Kashdan & Rottenberg, 2010; Sheppes & Gross, 2012; Troy and Mauss, 2011). Even typically positive strategies, such as reappraisal, are not the best choice in each context, therefore, different strategies must be interjected based on the emotional context (Sheppes & Levin, 2013; Sheppes et al., 2014). The absence of flexibility is seen in many mental health disorders such as rumination in anxiety disorders (Aldao, Nolen-Hoeksema, & Schweizer, 2010). Emotion regulation is also affected by individual variability, personality, culture, demographic variation, emotional values and the presence of mental health

disorders (Burns et al., 2008; Kim, Cornwell, & Kim, 2012; Mauss & Butler, 2010; Rossi & Pourtois, 2012; Sheppes et al., 2015; Webb et al., 2012).

Emotion processing in the brain. Emotionally salient stimuli increase processing in pertinent brain structures and enhance attention (Vuilleumier, 2002, 2005, 2009). Emotional relevance biases perceptual and attentional processing such that attention can be drawn away from a typical task and oriented to emotional stimuli because of its inherent value to the individual (Bradley, 2009; Pourtois, Schettino, & Vuilleumier, 2013; Yiend, 2010). Selective attention is the action of directing attention to only certain stimuli in the environment (Yantis, 2008). Emotion processing uses similar neural pathways as selective attention to acknowledge important visual information in the environment (Bekhtereva, Craddock, & Muller, 2015; Lang & Davis, 2006; Okon-Singer, Lichtenstein-Vidne, Cohen, 2013; Petersen & Posner, 2012). Researchers suggest that emotions and cognitive processing influence each other and are in a continuous state of change, both by bottom-up and top-down processing (Cole, Martin, & Dennis, 2004; Hart, Green, Casp, & Belger, 2010; Pessoa, Padmala & Morland, 2005; Pessoa et al., 2002; Tamietto & de Gelder, 2010). Specifically, emotional processing is thought to occur in parallel to cognitive processing, and therefore, it is theorized that emotional processing may use similar networks either separately, together with or in competition with attentional and perceptual resources (Amaral, Behniea, & Kelly, 2003; Balconi, 2011; Krolak-Salmon, Fischer, Vighetto, & Mauguiere, 2001; Pourtois, Spinelli, Seeck, & Vuilleumier, 2010; Rossi & Pourtois, 2012; Shaw, Lien, Ruthruff, & Allen, 2011; Sutton & Altarriba, 2011; Vuilleumier & Pourtois, 2007). Humans do not have unlimited attentional resources, and so it is thought that stimuli compete for sensory processing resources (Petersen & Posner, 2012). Lang & Davis (2006) suggest that emotional stimuli can attract more attention than neutral stimuli by biasing visual

processing resources; this can be done in a conscious state of awareness or unconsciously (Anderson, Christoff, Panitz, De Rosa & Gabrieli, 2003; Erthal et al., 2005; Luo et al., 2010; Pessoa, 2010; Pessoa et al., 2002; Pourtois et al., 2010; Vuilleumier, Armony, Driver, & Dolan, 2001; Vuilleumier & Driver, 2007). Current research is suggesting that specific emotions do not elicit a particular brain response systematically differentiating each emotion from the next (Kragel & Labar, 2016). Emotion, either positive or negative, can activate similar parts of the brain but may interact with more or less pathways depending on the arousal (i.e., low to high) and valence (i.e., unpleasant to pleasant) of the emotional stimuli (Cunningham & Brosch, 2012; Murray, 2007). This information suggests that the brain does not have a specific neural pathway that represents specific emotions (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012) and that there are contributing neural systems that help elicit emotional responses in the brain. This type of research looks at the brain holistically using multivariate techniques such as representational similarity analysis and pattern classification to understand how emotion is represented in the brain (Kragel & Labar, 2016; Hamann, 2012).

Why we perceive emotion. Current theories suggest that we perceive emotion so readily because we are motivated to do so by survival (Bradley, 2009). The motivational model of emotion suggests that perceiving emotional stimuli easily promotes human survival (Armony & Dolan, 2002; Bradley, 2009; Bradley, Keil, & Lang, 2012; Lang & Bradley, 2010; Ohman, Flykt & Esteves, 2001; Vuilleumier & Schwartz, 2001). This emotional information, termed motivated attention (Bradley et al. 2003; Lang, Bradley, & Cuthbert, 1997; Schupp, Flaisch, Stockburger, & Junghofer, 2006) or emotional attention (Pourtois et al., 2013), can be either positive or negative, categorized as either the defensive or appetitive systems (Bradley et al. 2003; Lang & Bradley, 2013). The defensive system would be triggered by an aversive stimulus that impacts a

person's safety, such as a bear entering a campsite (Wiens & Syrjanen, 2013). Appetitive examples are stimuli that provide feelings of wellbeing such as ingestion and sex (Brosch, Sander, Pourtois, & Scherer, 2008). The valence level, how unpleasant or pleasant the stimuli is, gauges the level of response and the level of activation of different systems (e.g., perceptual, limbic) which results in arousal levels (Lang & Bradley, 2010). According to the motivational model of emotion, increased arousal in either the defensive or appetitive system increases emotional attention (Lang & Bradley, 2010).

Neural and behavioral responses to emotion. Historically, there has been much debate as to whether there is a negative or positive bias to emotional attention, as past research suggested that emotional processing tended to have a negativity bias (Delplanque, Silvert, Hot, & Sequeira, 2005; Foti, Hajcak & Dien, 2009; Hajcak & Olvet, 2008; Huang & Luo, 2006). For example, participants would react more readily to negative stimuli such as fear than to all other stimuli. Current research shows evidence for both a negative and a positive bias (Franken, Muris, Nijs, & van Strien, 2008; Schupp, Junghöfer, Weike, & Hamm, 2004; Schupp, Ohman et al., 2004; Schupp et al., 2007, Weinberg & Hajcak, 2010). Regardless, both positive and negative emotional stimuli elicit a greater neural response when compared to neutral stimuli (Rozenkrants & Polich, 2008; Schupp, Junghofer, Weike, & Hamm, 2003 a, b; Schupp, Junghofer et al., 2004; Schupp et al., 2006). Several researchers have claimed that it may be a combination of objective and subjective measures and differing paradigms used in emotion research that may be responsible for why emotional attention produces differing results (Hedger, Gray, Garner, & Adams, 2016; Schlossmacher, Junghofer, Straube, & Bruchmann, 2017; Sterzer, Stein, Ludwig, Rothkirch, & Hesselmann, 2014). As a whole, emotional attention typically produces larger neural responses for emotional stimuli when compared to neutral stimuli (Lindquist et al., 2012),

and faster reaction times in both the temporal and limbic brain areas when compared to non-emotional attentional responses alone (Krolak-Salmon et al., 2004; Luo, Fu, & Galvin, 2007, 2010; Pourtois et al., 2010).

1.2 Event-Related Potentials

To study emotion, an imaging device called an electroencephalography records electrical activity, through electrodes placed on the scalp, that is generated in different brain structures (Hajcak, MacNamara, & Olvet, 2010; Teplan, 2002). Electrical activity is generated by neurons in the brain when an action potential is elicited by a neurophysiological response (i.e., cognitive or motor), such as making a decision or completing a task. Many action potentials from large populations of neurons produce positive and negative flows of current (Kappenman & Luck, 2011). Large populations of activated neurons are necessary to get recordable information, which is called an electroencephalogram (EEG; Teplan , 2002). Data from an EEG can be broken down further into more useable information called event-related potentials (ERPs; Fabiani, Gratton, & Federmeier, 2007; Luck, 2005; Teplan, 2002). ERPs are extracted from raw EEG recordings by averaging several recording periods (i.e., epochs) that are time-locked for specific events (i.e., tasks in emotional oddball designs; Kappenman & Luck, 2011; Teplan, 2002). ERPs are scalp-recorded voltage change elicited by an external or internal stimulus that signifies a mental process in the brain (i.e., attention or decision-making; Kappenman & Luck, 2011; Luck, 2005). Fabiani and colleagues (2007) suggest that there are psychophysiological inferences that can be made if we make the assumption that ERPs are a sign of brain activity produced by a psychological process such as attention. First, that an EEG can measure differences in conditions such as an attention task compared to a working memory task. Second, psychological processes can be invoked by a stimulus, and therefore, psychological processes can be studied for

differences (Fabiani, Gratton, & Federmeier, 2007). ERPs are analyzed for differences in latency (i.e., reaction time), amplitude (size) and scalp topography (Kappenman & Luck, 2011; Luck, 2005; Olofsson, Nordin, Sequeira, & Polich, 2008; Rugg & Coles, 1995). Amplitude and latency provide information about the strength and time course of neural processes. Research suggests that ERPs are useful to study the time course of different cognitive processes in the brain (Barbas, Zikopoulos, & Timbie, 2010; Luck, 2014; Nieuwenhuis, Aston-Jones & Cohen, 2005; Phillips, Ladouceur, & Drevets, 2008a, b; Polich, 2007; Rugg & Coles, 1995; Vrticka, Sander, & Vuilleumier, 2011). ERPs can either be positive or negative deflections (i.e., P or N), and are typically given a numerical value to identify the approximate time that the ERP value occurs at (i.e., P300 starts approximately 300 milliseconds after a stimulus; Kappenman & Luck, 2011). The advantages of EEG research are that it is non-invasive, as the electrodes sit on top of the scalp, and it has excellent temporal resolution, the ability to record complex patterns of neural activity all over the brain, within fractions of a second (Kappenman & Luck, 2011; Rugg & Coles, 1995). The main disadvantage of EEG research is that the spatial resolution, determining the specific brain structures that generate the EEG information, is poor. There are other neuroimaging techniques that are used to look at the function and structure of the brain. Typically, fMRI is used for studying the location of signals that are distributed around the brain and diffusion tensor imaging (DTI) looks at the structural components of the brain (Campanella & Philippot, 2006).

How ERPs are elicited in emotion research. ERPs are typically affected by emotional pictures, and therefore, an abundance of research have used pictures in different paradigms to investigate affective and cognitive processing (Lang & Bradley, 2010). ERPs can be elicited by different emotional stimuli such as emotional scenes or faces (Pourtois, Grandjean, Sander, &

Vuilleumier, 2004; Schupp et al., 2006; Stolarova, Keil, & Moratti, 2006; West, Anderson, Ferber, & Pratt, 2011) and depending on the type of emotional paradigm used, early and late neural responses can be stimulated (Hajcak et al, 2010; Olofsson et al., 2008; Sabatinelli et al., 2007; Schupp et al., 2006). Typically used in emotional research, International Affective Picture System (IAPS) are a standardized set of pictures that have been reviewed for their valence (unpleasant to pleasant) and arousal (low to high) levels (Lang et al., 1997). IAPS are rated on a nine-point-scale for both valence and arousal by mainly female and male university aged participants. Research has found that IAPS with emotional content bias attention compared with neutral content (Codispoti, Ferrari, & Bradley, 2007; Olofsson & Polich, 2007). Emotional oddball paradigms, where participants are required to differentiate between frequent (i.e., neutral) and deviant (i.e., emotional) stimuli, are regularly used to look at emotionality in both normal and clinical populations (Wang, McCarthy, Song, & Labar, 2005). Differences in amplitude and latency in these populations can provide information of how emotional processing is occurring in normal and pathological brains (Campanella & Philippot, 2006; Kok, 2001).

Emotion produces greater ERPs and faster RTs. Emotional research has established that both pleasant and unpleasant stimuli are viewed longer than neutral stimuli (Bradley, Codispoti, Cuthbert, & Lang, 2001) and have larger ERP components following an emotional stimulus compared to a neutral one (Carretie, Hinojosa, Albert, & Mercado, 2006; Carretie, Hinojosa, Martin-Loeches, Mercado, & Tapia, 2004a; Delplanque, Lavoie, Hot, Silvert, & Sequeira, 2004; Keil et al., 2002; Schupp, Junghöfer, Weike, & Hamm, 2003b, 2004; Schupp, Cuthbert et al., 2004; Schupp, Ohman et al., 2004). Jiang and colleagues (2018) found that fearful faces produced larger ERPs even when the participants were not conscious of the stimuli. Since emotional stimuli evokes more attention and visual perception than neutral stimuli

(Dominguez-Borras & Vuilleumier, 2013; Pourtois et al., 2013), this typically results in faster RTs and greater accuracy in tasks (Anderson, 2005; Anderson & Phelps, 2001; Brosch, Pourtois, Sander, Vuilleumier, 2011; Frischen, Eastwood, & Smilek, 2008; Pourtois et al., 2004). Neuroimaging studies have found greater fMRI responses in visual regions in response to emotional material, including face stimuli (Junghofer et al., 2006; Phan et al., 2004; Sabatinelli et al., 2005, 2007, 2011). ERP studies have revealed emotional amplification as face processing occurs, from initial perception of the stimulus to higher cognitive processing, suggesting that emotional information continues to be processed as long as the stimulus is present (Eimer & Holmes, 2007; George, 2013).

The history of the P300 ERP. In 1965, the P300 was discovered by four men, Sutton, Braren, Zubin, & Jon, and has since been studied for over four decades. The P300 is a positive ERP component that is recorded at parietal sites, which occurs approximately between 250 to 600 milliseconds (ms) after a stimulus occurs (Bledowski et al., 2004; Campanella & Phillippot, 2006; Hajcak et al., 2010; Kok, 2001; Polich, 2007; Ranganath & Rainer, 2003; Rotshtein et al., 2010; Singhal et al., 2012). The P300 is elicited by a person attending to a stimulus, storing the information in short term memory, and making a decision about the stimulus (Hajcak et al., 2010; Polich, 2007; Rotshtein et al., 2010). The cognitive process it measures is the conscious awareness to make decisions and to access memories to add experiential information to the decision-making process (Campanella & Phillippot, 2006; Kok, 2001; Ranganath & Rainer, 2003). Recent research suggests that not only are the ventral regions of the brain (i.e., the amygdala, etc.), involved in emotion processing, responsible for eliciting the P300, but the dorsal executive neural system (i.e., the dorsolateral prefrontal cortex and lateral parietal cortex) contribute to the P300 as well; this area is largely responsible for executive cognitive

functioning, of which attention and working memory are involved (Moore, Shafer, Bakhtiari, Dolcos, & Singhal, 2019). Research suggests that the P300 is processed by top-down attentional resources and influenced by the extrinsic and/or intrinsic motivation of the individual in conjunction with the effects of the stimulus (Hajcak et al., 2010). P300 responses can be elicited by emotional stimuli (Carretie, Iglesias, & Garcia, 1997; Krolak-Salmon et al., 2001; Luck, 2014; Olofsson et al., 2008). Variations in amplitude and latency (i.e., RT) are seen in the P300 typically when the complexity, vigilance, arousal level, task-relevance or motivational significance is altered (Campanella et al., 2002; Hansenne, 2000). P300 latency is said to reflect the timing of mental processes (i.e., reaction time) and P300 amplitude is said to be related to the intensity of the processing (Kok, 2001). One consistent finding is that emotionally intense or highly arousing stimuli produces a larger P300 (Briggs & Martin, 2009; Delplanque et al., 2004, 2005; Delplanque, Silvert, Hot, Rigoulot, & Sequeira, 2006; Keil et al., 2002; Olofsson and Polich, 2007; Polich, 2007; Rozenkrants & Polich, 2008). For example, a recent study done with emotionally healthy participants, found larger P300 ERPs for target stimuli in an emotional oddball task (Moore et al., 2019).

1.3 Brain structures involved in Emotion Processing

The amygdala monitors emotional information in the environment, and determines the involvement of other cognitive processes (i.e., whether to up-regulate or down-regulate), based on the salience of the emotional stimuli, and the appropriate behavioral response (Pourtois et al., 2013; Vuilleumier, 2005; Vuilleumier & Huang, 2009). This interplay between the amygdala and cognitive resources (i.e., attention, perception) is continually modulated by a feedback system that acts additively or diminutively (Etkin, Prater, Menon, & Schatzberg, 2010; Sabatinelli et al., 2009). Pourtois and colleagues (2013) have called this “gain control system” the Multiple

Attention Gain Control (MAGiC) model. Despite emotion and attention having distinct neural pathways, emotions can either gain more or less attention depending on the emotional information and its impact on the individual (Brosch et al., 2011; Pourtois et al., 2013; Rossi & Pourtois, 2012). It is suggested that the brain has a "common perceptual pathway" that emotional information can use to compete for awareness (Pourtois et al., 2013). The MAGiC model suggests that the perceptual and attentional networks are continually being modulated by information received from multiple sources throughout the brain and that this influx of information operates in parallel to elucidate sensory information and determine an appropriate response (McMains & Kastner, 2011; Vuilleumier, 2005; Vuilleumier & Driver, 2007).

There are several other brain structures that also provide support to emotional processing, such as various parts of the prefrontal cortex (PFC), limbic regions such as the anterior cingulate cortex (ACC) and the hypothalamus, and the visual cortex (Dolcos, Iordan, & Dolcos, 2011; Dolcos & McCarthy, 2006; Fichtenholtz et al., 2004; Fitzgerald et al., 2004; Goldin, McRae, Ramel, & Gross, 2008; Kim & Hamann, 2007; Phan, Taylor et al., 2004; Phan, Wager, Taylor, & Liberzon, 2004; Phillips, Drevets, Rauch, & Lane, 2003; Sabatinelli et al., 2007, 2011; Wang et al., 2005). Research has distinguished the brain structures responsible for emotional processing as the "ventral neural circuitry," running from the front to the back of the brain, cognitive to visual control centres, respectively, and orchestrated by the amygdala, the emotion control centre (Dolcos & McCarthy, 2006; Shafer et al., 2019). The "dorsal neural circuitry" runs parallel to the emotional processing circuitry but is differentiated by overseeing the attentional control of the brain (Petersen & Posner, 2012). Emotion processing occurs when the amygdala receives the initial sensory information and then enhances feedback systems to and from the PFC and visual cortex via cortical and subcortical structures (Grabowska et al., 2011; McGaugh, 2005). It is

suggested that there is a two-stage model of emotional attention where the amygdala perceives emotion as early as 40 ms, and then the amygdala triggers a gain control response to the visual cortex (Garrido, Barnes, Sahani, & Dolan, 2012; Luo et al., 2010; Pourtois et al., 2010; Vuilleumier et al., 2001). Research suggests that the amygdala is initially activated without any conscious awareness, where sensory information is relayed without any attentional control (Tamietto & Gelder, 2010). Other research also suggests that the amygdala detects emotions the earliest in the processing stream, and attention and perception additively effect this processing through sensory feedback that continues well after the initial recognition of the stimulus (Pourtois & Vuilleumier, 2006; Rudrauf et al., 2008; Vuilleumier, 2005). For example, the amygdala responded more to fearful faces compared to neutral stimuli initially but this response was modulated depending on task load during later processing (Costafreda, Brammer, David, & Fu, 2008; Erthal et al., 2005; Luo et al., 2010).

The amygdala and visual processing of emotion. It is suggested that the feedback networks from the amygdala to the visual cortex are responsible for the perceptual enhancement of emotional stimuli (Sabatinelli et al., 2005). There are currently two theories: the two-pathway hypothesis (via subcortical or direct occipital inputs) and the two stage hypothesis (via fast cortical-to-cortical pathways; Pessoa and Adolphs, 2010; Pourtois et al., 2010; Vuilleumier, 2005). This gain control on the perceptual processing system appears to be largely due to the amygdala, as an amgydalar response is typically elicited before visual processing occurs and there is evidence in primates and DTI studies in humans of bidirectional connections to sensory brain areas (Amaral et al., 2003; Catani, Jones, Donato, & ffytche, 2003; Lang & Davis, 2006; Pourtois & Vuilleumier, 2006; Veilleumier, 2005; Vuilleumier & Driver, 2007). It is suggested that it is this connection that provides perceptual bias towards emotional stimuli (bottom-up;

Grandjean et al., 2005; Peelen, Atkinson, Andersson, & Vuilleumier, 2007; Vuilleumier et al., 2001) as lesions of the amygdala abolish this bias (Rotshtein et al., 2010; Vuilleumier, Richardson, Armony, Driver, & Dolan, 2004). Specifically, primate studies show projections from the visual nuclei to the lateral amygdala (Day-Brown et al., 2010). Amygdala and visual cortex connectivity was also inferred in fMRI studies in humans, as increased BOLD signals were seen in both areas as rated picture arousal increased (Sabatinelli et al., 2005). Schettino and colleagues (2011) suggest that rapid processing occurs through subcortical pathways from the visual centre (i.e., magnocellular nuclei) to the amygdala and provides course visual information (i.e., low-spatial, non-specific information; Alorda, Serrano-Pedraza, Campos-Bueno, Sierra-Vazquez, & Montoya, 2007; Carretie, Hinojosa, Lopez-Martin, & Tapia, 2007). More specific information is provided through slower cortical to cortical pathways from the visual centre (i.e., parvocellular nuclei) to the amygdala. Bocanegra & Zeelenberg (2011) and Mermillod and colleagues (2010) also found that the fear response was transported through the fast responding, low-spatial information subcortical pathways. Face and object processing require the slow cortical-to-cortical pathways that provide detailed information (Schettino et al., 2011; Vlamings, Goffaux, & Kemner, 2009). These findings together suggest that fear detection uses the fast, non-conscious processing route (Méndez-Bértolo et al., 2016) whereas further processing of emotional stimuli is boosted by inputs from the amygdala and other cortical and subcortical areas (Vuilleumier, 2005).

1.4 Mental Health Disorders and Emotion Processing

Emotion regulation is a multi-process event, that involves multiple brain structures, which starts with emotional stimuli, uses one or more processing systems, and ends with how emotion is generated in an individual (Bonanno & Burton, 2013; Gross et al., 2011a, b, 2015;

Sheppes et al., 2015; Webb, Gallo et al., 2012). It is suggested that many mental health disorders involve emotion processing and/or emotion regulation problems (Gross & Jazaieri, 2014; Jazaieri, Urry, & Gross, 2013; Kring, 2010; Werner & Gross, 2010), such as conduct disorder (Euler, Sterzer, & Stadler, 2014; Sterzer, Stadler, Krebs, Kleinschmidt, & Poustka, 2005), attention deficit hyperactivity disorder (ADHD; Walcott & Landau, 2004), and mood and anxiety disorders (Aldao et al., 2010; Campbell-Sills & Barlow, 2007; Carthy, Horesh, Apter, & Gross, 2010; Hannesdottir & Ollendick, 2007; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007). Mental health disorders are thought to be associated with impairment in the brain structures that either generate emotion or facilitate emotion regulation (Sheppes et al., 2015). For example, Shafer and colleagues (2019) found a reduction in the amount of white matter found in the corpus callosum, a white matter tract that shares information between the right and left sides of the brain. This reduction in white matter was associated with psychopathology in adolescents (Shafer et al., 2019). Emotion dysregulation can occur at any one of the following stages: awareness of stimulus, determining the importance of the stimulus (i.e., valence) and response to the stimulus (Gross, 2002). An emotionally dysregulated individual can either fail to emotionally regulate, or use emotional regulation strategies that are mismatched to the current emotional context (Bonanno & Burton, 2013; Gross, 2013; Gross & Jazaieri, 2014). Many studies have shown deficits in emotion recognition by individuals with mental health disorders, and ERP studies have linked this deficit with P300 alterations (Alexander, Hermens, Keage, & Gordon, 2008; Dawel, Kearney, McKone, & Palermo, 2012; Hansenne, 2000). Most emotion research is done with participants that are emotionally and physically healthy. For example, a recent study done with healthy participants found positive correlations between emotional arousability and neural response to negative stimuli, and concentration difficulties and emotional distractibility

(Moore et al., 2019). Less research is done with participants with mental health diagnoses or patients that have incurred damage to parts of their brain. For example, patients that have amygdala damage have emotional and social deficits such as the inability to recognize fear in facial expressions or other threat stimuli (Pourtois et al., 2013). Kennedy and Adolphs (2010) research suggests that a possible reason for these deficits is the inability for these patients to attend to the appropriate stimulus. For example, the amygdala-damaged individuals lacked the ability to look at the eyes of emotional faces and when directed to do so were able to elicit a normal fear response. This suggests that the deficit lies in the inability for the amygdala to provide appropriate feedback to the attentional system to get an appropriate response, rather than solely a perceptual deficit problem (Barbas et al., 2010; Vuilleumier & Huang, 2009).

The effects of emotion on cognitive processing. The interplay between cognition and emotion processing can either enhance or exacerbate the processing of emotional stimuli. For example, cognitive-behavioural therapy (Butler, Chapman, Forman, & Beck, 2006) is used to treat many mental health disorders, and one of the central focuses of this treatment is how to identify and improve emotion regulation skills (Campell-Sills & Barlow, 2007; Hofmann, Schmeichel, & Baddeley, 2012). Emotional research demonstrates that cognitive processing can be impacted both positively and negatively. Emotional stimuli can negatively impact working memory performance, produce slow reaction times during perceptual tasks, and reduce task accuracy, suggesting cognitive control impairments (Blair et al., 2007; Dolcos & McCarthy, 2006; Gupta & Deak, 2015; Hart et al., 2010; Uher, Brooks, Bartholdy, Tchanturia, & Campbell, 2014). Alternatively, emotions presented both visually and auditorily can also have positive influences on cognition, such as improving reaction times and accuracy (Kanske & Kotz, 2011; Schupp et al., 2007; Zinchenko, Kanske, Obermeier, Schroger, & Kotz, 2015). There seem to be

several factors that determine whether the interaction between emotion and cognitive processes produces positive or negative effects, they include: level of threat, cognitive load, individual differences, and the availability of conflict solving cognitive resources (Cohen & Henik, 2012; Gupta, Hur, & Lavie, 2016; Gupta & Raymond, 2012; Kanske, 2012; Kim, Cornwell, & Kim, 2012; Okon-Singer et al, 2013; Pessoa, 2009). Moore and colleagues (2019) found that individual differences played a role in how emotion was processed and how much attention was directed to tasks, as they found a positive correlation between participants' sensitivity to negative emotion and emotional distraction. It is suggested that the PFC, where cognitive processing such as attention takes place, may be responsible for the reactivity to emotional stimuli seen in some mental health disorders. This reactivity to emotional stimuli affects the individual's ability to identify distinct differences between emotional stimuli, making the individual more vulnerable to negative affect (Bishop, 2007). For example, aversive stimuli can subvert processing from the attentional load to the amygdala so that the brain pays attention to threatening environmental cues instead (Cornwell et al., 2011). Amygdala activity can be increased or decreased depending on emotional regulation strategies, personality factors, genetic makeup, and mental health (Canli, Sivers, Whitfield, Gotlib, & Gabrieli, 2002; Cornwell et al., 2011; Drevets, 2003; Etkin et al., 2010; Hariri, Tessitore, Mattay, Fera, & Weinberger, 2002; Ochsner & Gross, 2005). In turn, amygdala changes alter the functioning of the prefrontal areas of the brain. Research found that the amygdala had increased reactivity to emotional stimuli for those individuals with mental health disorders (Davey, Allen, Harrison, & Yucel, 2011; Kim, Loucks, Palmer, & Brown, 2011). The amygdala's reactivity to emotional stimuli are also influenced by an individual's emotional state and the emotional context they are in (Bishop, 2007; Cornwell et al., 2011). Further, this reactivity to emotional stimuli in mental health disorders is not just elicited by

negative stimuli but also positive stimuli, tending towards the current theory that emotional attention is stimulated by all salient emotional stimuli (Somerville, Kim, Johnstone, Alexander, & Whalen, 2004; Straube, Mentzel, & Miltner, 2005). For example, hypervigilance towards emotional stimuli can be induced in anxious individuals by positive emotions as well as negative (Rossignol, Philippot, Bissot, Rigoulot, & Campanella, 2012). Attentional biases for negative information are typically seen in individuals that are either anxious or depressed (Denkova et al., 2010). This may be a result of the typical emotion regulation strategy that is used. Three emotion-regulation strategies that are potentially protective against mental health disorders are reappraisal, problem solving, and acceptance (Aldao et al., 2010). Whereas, three emotion regulation strategies that are considered risk factors for mental health disorders are suppression, avoidance, and rumination (Aldao et al., 2010). Overall, research suggests that flexibility to implement the best context-specific emotion regulation strategy provides the most adaptive results (Aldao et al., 2010).

Anxiety disorders and emotion regulation. Emotion regulation problems are suggested to be a key factor in the genesis of anxiety, causing a chronic inability to supress negative emotion (Jackson, Malmstadt, Larson, & Davidson, 2000). It is suggested that anxiety disorders tend to have an increase in emotional sensitivity, distractibility and impairments in cognitive and perceptual functioning (Bishop, 2009; Dolcos, Iordan, & Dolcos, 2011; Eysenck, Derakshan, Santos, & Calvo, 2007; Pujol et al., 2009). Some studies have shown that anxiety interferes with normal emotion processing and tends to increase attention bias towards threat or negative stimuli (Armstrong & Olatunji, 2012; Bar-Haim et al., 2007; Campanella & Philippot, 2006; Carretie, Mercado, Hinojosa, Marten-Loeches, & Sotillo, 2004b; Cisler & Koster, 2010; Frewen, Dozois, Neufeld, & Lanius, 2008; Mogg & Bradley, 2002; Rossignol et al., 2013; Williams, 2006; Yiend,

2010). fMRI studies show an abnormal amygdala response to threatening stimuli in anxious children (McClure et al., 2007; Thomas et al., 2001a). Anxiety not only increases attention towards threat and makes it difficult for the individual to disengage attention, but it also modifies how fear is perceived (Armstrong & Olatunji, 2012, Bar-Haim et al., 2007). Studies show anxiety can increase amygdala and sensory cortices responses, resulting in a faster perceptual processing and reaction time in tasks (Bishop, 2007). At the same time, anxiety can diminish the individual's ability to detect threat appropriately because the amygdala is overly reactive to stimuli (Cornwell et al., 2011; Rossi & Pourtois, 2012; van Marle et al., 2009). Research suggest that anxiety increases processing for decision-making but diminishes the ability to make discriminations between emotional content (Bishop, 2007). In one study, socially phobic participants showed an attention bias towards threatening faces, whereas another study with participants of a similar diagnosis tended to direct their attention away from emotional faces (Mogg, Philippot, & Bradley, 2004; Pishyar, Harris, & Menzies, 2008). Research suggests that socially anxious people are hypervigilant to recognize emotion in people's faces for the initial second of processing, but then tend to avoid consistent eye contact with people for any further duration of processing (Miskovic & Schmidt, 2012; Mogg & Bradley, 2002, 2004). Moriya & Tanno (2011) also found that highly anxious participants exhibited this behaviour of looking longer at emotional faces for the first second of stimulus exposure but then avoided further processing at later time points. It has also been suggested socially anxious participants have difficulty disengaging attention from a stimulus (Klumpp & Amir, 2009; Rossignol et al., 2012; Sheppes, Luria, Fukuda, & Gross, 2013). This may be consistent with the symptoms of rumination typically seen in anxiety. Anxiety in children have some consistent similarities with adults as they also demonstrate greater frequency and intensity of negative emotional responses

and deficits in using appropriate emotion regulation strategies in different contexts (Carthy et al., 2010). They also report greater negative emotional ratings in response to emotional stimuli and subjective experience of emotion (Goldin et al., 2008; McLaughlin, Hatzenbuehler, Mennin, & Nolen-Hoeksema, 2011; Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). Thomas and colleagues (2001) suggest that hyper-reactivity in children may be responsible for the greater activation of the amygdala in response to fearful faces. Anxious children show greater experiential emotional reactivity to emotional stories and to threatening images compared to controls (Carthy et al., 2010; Suveg & Zeman, 2004). They also show less flexible control of attention, a cognitive ability necessary for emotion regulation (Muris, Roelofs, Rassin, Franken, & Mayer, 2005). Anxious children reported lower self-efficacy in their ability to regulate emotion and more dysregulated expressions of emotion compared to non-anxious children due to their inability to effectively regulate their emotions (Suveg & Zeman, 2004). Kim & Cicchetti (2010) suggest that children with anxiety struggle with emotional awareness, which ultimately affects their ability to emotion regulate.

Affective disorders and emotion regulation. Research suggests that attentional resources are impacted by negative affective states (DeRaedt & Koster, 2010; Deveney & Pizzagalli, 2008). Kemp and colleagues (2005) suggest that there is a negativity bias in depression, which involved prolonged processing of emotional stimuli that is predominantly negative or aversive. Other research suggests that depressed individuals process all emotional stimuli, and evaluate positive, neutral or ambiguous facial expressions as more sad or less happy when compared to a control group (Bourke, Douglas, & Porter, 2010). Brain imaging in individuals with depression and bipolar disorders have found abnormal functional asymmetries in the frontal cortex, which effect their ability to emotionally regulate (Herrington et al., 2010;

Philips, Ladouceur, & Drevets, 2008). Reviews of EEG depression studies have found more activity on the right side of the PFC, which is consistent with findings for unpleasant emotions (Davidson et al., 2002). Research has also suggested that depression is related to impaired emotion regulation (Bourke et al., 2010; Campbell-Sill, Barlow, Brown, & Hofmann, 2006; Erk et al., 2010; Joormann & Gotlib, 2010; Kovacs, Joormann, & Gotlib, 2008; Lui & Thompson, 2017). For example, depressed individuals have difficulties with cognitive control and inhibiting negative thoughts. Research suggests that depressed individuals are more likely to suppress emotion than to use other emotion regulation strategies such as reappraisal (Joorman & Gotlib, 2010). Depressed individuals are also less likely to use effective strategies or have the motivation to implement strategies when needed (Campbell-Sills & Barlow, 2007). In individuals with major depressive disorder, fMRI research suggest reduced activation of the PFC during emotion regulation (Erk et al., 2010). The ability to read the emotional context of each situation is particularly difficult for those individuals with depression (Coifman & Bonanno, 2010; Larson, Nitschke, & Davidson, 2007). This may be largely due to the lack of flexibility when responding to emotional stimuli. Rottenberg and colleagues (2002, 2005) noticed that depressed participants had similar responses (i.e., sad) while watching different emotional scenes (i.e., amusing, neutral, and sad) whereas healthy controls showed variable emotions dependant on the scene shown. Insensitivity to emotional context was also seen in bereaved individuals (Bullock & Bonanno, 2013). Long-term studies revealed that insensitivity to emotional context predicted continued depressive symptoms, whereas those individuals who were sensitive to emotional context had a reduction in their depressive symptoms over an 18 month period (Coifman & Bonanno, 2010).

Behavioral disorders and emotion regulation. Other mental health disorders that struggle to implement emotion regulation strategies appropriately are ADHD and conduct

disorder (CD; Euler et al., 2014; Gross & Jazaieri, 2014; Sterzer et al., 2005). Children that exhibit ADHD and CD are typically more prone to cognitive impairments and low emotional regulation (i.e., undercontrolled; Eisenberg et al., 2001; Euler, et al., 2014; Williams et al., 2008). Research suggests that ADHD and CD are linked to poor inhibitory control, which affects their ability to control attention, behaviour and emotions (Eisenberg et al., 2001; Euler et al., 2014; Kim & Cicchetti, 2010). In adolescent youth with ADHD, lower amounts of white matter in the superior longitudinal fasciculus was associated with deficits in attention, working memory, and inhibitory control (Chiang, Chen, Lo, Tseng, & Gau, 2015; Shafer et al., 2019). Research suggests that there is an inverse relationship with the emotion and attentional processing systems (i.e., MAGiC model), suggesting that the use of one system takes away the full impact of the other (Bradley, 2009; Pourtois et al., 2013; Yiend, 2010). With this logic, adolescents that struggle cognitively (i.e., with attention, working memory, inhibitory control) may also struggle to emotionally process information (Dolcos et al., 2011; Iordan, Dolcos, & Dolcos, 2013). Steiben and colleagues (2007) believe that aggressive behaviour may be linked to impaired emotion regulation and may reflect limitations in executive function. Recent research suggests that aggressive behaviour may be linked to self-control efficacy (Denson, DeWall, & Finkel, 2012). Euler and colleagues (2014) found evidence of impairment in cognitive control in aggressive CD participants when presented with distressing emotional stimuli compared to healthy controls.

Mental health disorders impact behavioral responses to emotion. Individual diagnosed with mental health disorders tend to have divergent behavioural responses to emotional stimuli due to their difficulties with cognitive control and emotional regulation. For example, boys, ages 8 to 13, diagnosed with ADHD had longer reaction times to emotional

stimuli than to neutral stimuli when compared to controls (Lopez-Martin, Albert, Fernandez-Jaen, & Carretie, 2013). Research shows that highly anxious participants have faster reaction times for emotionally deviant faces when compared to controls (Campanella & Phillippot, 2006; Rossignol, Phillippot, Crommelinck, & Campanella, 2008; Rossignol, Philippot, Douilliez, Crommelinck, & Campanella, 2005). A clinical group made up of multiple diagnoses, such as ADHD and affective disorders, showed longer RT's to fearful pictures compared to unemotional pictures (Singhal et al, 2012). Altogether, participants with mental health disorders provide different behavioral responses in their ability to emotionally regulate in emotion research.

Mental health disorders impact neural responses to emotion. Individuals diagnosed with mental health disorders, in addition to behavioural differences, have different ERP patterns in the brain when compared to healthy controls. ERP studies found that the P300 appeared significantly earlier in anxious individuals compared to controls, meaning that they are detecting and responding faster to emotional stimuli (Bar-haim, Lamy, & Glickman, 2005). Meta-analyses of neuroimaging results show some similar results for individuals with anxiety (Etkin & Wager, 2007). For example, an early P300 was elicited for masked fearful faces (Liddell, Williams, Rathjen, Shevrin, & Gordon, 2004) and earlier and larger ERPs resulted from seeing fearful faces (Bar-Haim et al., 2005). Rossignol and colleagues (2005) found that highly anxious participants had earlier latency and larger amplitude for the P300 component in response to fear trials than to happy ones when compared to controls. Campanella & Phillippot (2006) found that anxious participants elicited a significantly earlier P300 latency when viewing infrequent emotional stimuli (i.e., fearful and happy faces) when compared to controls. Rossignol and colleagues (2007) found that socially anxious people elicited an earlier and larger P300 component when they viewed deviant faces of different emotions when compared with controls.

Depressed university students showed an attentional bias to negative stimuli and a larger P300 amplitude compared to controls (Ilardi et al., 2007). Conversely, Cavanagh and Geisler (2006) found that university students with depression symptoms had significantly smaller and delayed P300 amplitudes and latencies, respectively, for happy faces and significantly smaller P300 latencies for happy faces. A visual continuous performance task and an auditory oddball task was used to measure ERPs in children and adolescents, ages 6 to 18. They found that this task elicited a smaller P300 wave when compared to controls (Alexander et al., 2008). The P300 amplitude was also found to be smaller among adolescent boys shown to exhibit ADHD and CD symptoms (Bauer & Hesselbrock, 2003; Kim, Kim, & Kwon, 2001; Williams et al., 2008). A smaller P300 amplitude was also seen in adolescents with early problem behavior (i.e., early illicit drug use, etc.) and was associated with future diagnoses of externalizing mental health disorders (Iacono & McGue, 2006).

Implications for this study. The value of this study is to elucidate how adolescents with mental health disorders process and regulate emotion, providing a window into participant's neural and behavioural responses to emotional stimuli. This type of research has the potential to build upon what is known so that current treatments and interventions can evolve to provide the most efficacious help for adolescents wrestling with emotional problems. This study also may be a piece in the puzzle of future innovation in treatments and interventions as the information adds and challenges future research.

Behavioral and neural hypotheses for the current study. For the present study, I would like to identify whether there are differences between a group of adolescents with different mental health disorders (i.e., clinical group) and a mentally healthy control group (i.e., control group) in regards to how they process and regulate emotional information. We elucidated

this information using an emotional oddball paradigm that uses emotional (i.e., distracters) and non-emotional pictures (i.e., circles) to measure behavioural (e.g. RT) and neurophysiological (e.g. P300 ERP's) information. The goal was to see how each group will respond to the emotional information during and after an emotional stimulus has been presented. Studies show that affective disorders such as anxiety and depression can cause faster perceptual processing and reaction time in tasks (Bishop, 2007; Ilardi et al., 2007), and less flexible control of cognition, prolonged processing of emotional stimuli and a harder time disengaging from an emotional stimulus (Armstrong & Olatunji, 2012; Bar-Haim et al., 2007; Klump & Amir, 2009; Muris et al., 2005; Rossignol et al., 2012). My first hypothesis is that the clinical group will respond faster behaviourally to emotional stimuli in comparison to the control group. My second hypothesis is that the clinical group will find it difficult to disengage from and appropriately regulate emotional information as fast as the controls so reaction time for stimuli after emotional pictures will be longer. For my third hypothesis, I predict that the clinical group will have larger overall P300 ERPs in response to emotional content when compared to the healthy control group. I believe that the larger ERP response will be due to the clinical groups reactivity to the emotional information compared to the control group. My final prediction is that the clinical group will show a different ERP morphology overall, with earlier and larger P300's, when compared to the control group.

CHAPTER II - Methods

2.1. Participants

Fifty-five participants, ages 12 to 17 (average age = 14.87; SD = 1.38), were recruited from CASA, a local mental health provider in Edmonton, AB, Canada. These participants (i.e.,

the clinical group) were diagnosed with variable mental health disorders, such as depression and anxiety disorders, ADHD, CD, learning disorder (LD), fetal alcohol spectrum disorder (FASD), oppositional defiant disorder (ODD), intermittent explosive disorder (IED), and reactive attachment disorder (RAD), according to the *Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition* (DSM-5), Axis I Disorders. See **Table 1** for the full summary of all the clinical diagnoses included in the study. Twenty healthy controls, age 12 to 17 (average age = 14.89; SD = 1.74), were recruited from Edmonton, AB. All participants were checked for normal vision or corrected vision to complete the task appropriately. All participants and parental guardians signed both the informed consent and assent before taking part in the study. The study was approved by the Health Research Ethics Board at the University of Alberta for the ethical treatment of human participants. For behavioral data, statistical analysis was done with 42 participants in the clinical group (female = 18; male = 24), ages 12 to 17 (average age = 15.06; SD = 1.36), and 17 healthy controls (female = 11; male = 6), ages 12 to 17 (average age = 14.75; SD = 1.74). In the behavioral analysis, participants that responded in less than 200 ms were excluded from the study, due to the research that suggests that making a decision in regards to a stimulus occurs later than this time window (Campanella & Phillippot, 2006; Polich, 2007; Singhal et al., 2012). ERP data was assessed for 35 participants in the clinical group (female = 13; male = 22), ages 12 to 17 (average age = 15.04; SD = 1.46), and 13 healthy control participants were assessed (female = 10; male = 3), ages 12 to 17 (average age = 15.21; SD = 1.78). These participants were chosen because they had the best ERP signal-to-noise ratio and were free from any outlying data points as determined during data cleaning through visual inspection. The participants were the same in both the behavioral and ERP analysis, with fewer participants being included in the ERP data due to behavioral artifacts (i.e., eye blinks).

2.2 Task and Stimuli

Participants executed a modified version of the emotional oddball paradigm (Wang et al., 2005) which consisted of oddball targets and infrequent distracters, constituting 21% of the stimuli (124 trials), and frequent stimuli that act as the baseline, making up 79% (465 trials) of the stimuli (scrambled pictures). Infrequent distracters form the emotional aspect of the task, consisting of neutral pictures (26 trials), sad and fearful pictures (13 trials each), and positive pictures (4 trials). The oddball targets (i.e., circles) were grouped according to the infrequent distracter that came before the target, such as target-after-neutral (22 trials), target-after-sad (11 trials), target-after-fear (11 trials), and target-after-target (24 trials). The emotional pictures (the infrequent distracter = sad, fearful, positive, and neutral pictures) were selected from IAPS and from in-house pictures used in previous studies (Wang et al., 2005; Wang, Huettel, & De Bellis, 2008). Originally, each sad and fearful picture were paired with a neutral picture that had similar qualities (i.e., sad picture = man sitting while crying; neutral picture = man sitting with no expression on his face). For statistical analyses, these neutral subgroupings were collapsed into one neutral picture and one target-after-neutral category. Positive pictures served as emotional anchors, to provide emotional context, and were not analyzed statistically. Each target was unique as the infrequent circle targets varied in size and colour. The frequent distracter stimuli were digitally scrambled versions of the pictures therefore the scrambled pictures were similar to the emotional and neutral pictures in spatial frequency and luminance. Participants were asked to press one button for all target stimuli and the other button for all other stimuli, frequent (scrambled) and infrequent (sad, fearful, and neutral pictures) stimuli.

2.3 Event-Related Potentials (ERP) Recordings & Analyses

ERPs were recorded using a high-density 256-channel Geodesic Sensor Net (Electrical Geodesics Inc., Eugene, OR), amplified at a gain of 1000 and recorded at a sampling rate of 250 Hz [impedance <50 KΩ and initially referenced to the vertex electrode (Cz)]. Using Netstation (Version 4.4.2, Electrical Geodesics, Inc., Eugene, OR) data were bandpass filtered from 0.1 to 30 Hz, grand average re-referenced offline, and segments were constructed around events of interest from 300 ms pre-stimulus to 1500 ms post-stimulus. Data were baseline corrected (-300 to 0 ms), and eye-movement artifacts were removed. A minimum of five epochs per condition were required for the participants data to be considered in ERP analyses. Individual waveforms were reviewed and the P300 was identified for each participants at one midline electrode (i.e., Cz) and three parietal electrode sites (i.e., Pz, PCz, & POz), the area that maximally elicits the P300 amplitude (Campanella & Phillippot, 2006; Campanella et al., 2013; Polich, 2007; Singhal et al., 2012). See **Figure 1** for a diagram of electrode placements for the ERP data that was recorded and used in the statistical analysis. The time window for the P300 was determined by visual inspection as falling between 250 ms and 550 ms. The visual inspection was also in alignment with previous research that determined the P300 was within this time window for oddball tasks (Campanella & Phillippot, 2006; Campanella et al., 2013; Polich, 2007; Singhal et al., 2012). The clinical group was compared to the healthy control group for differences in P300 amplitude. This difference was compared for both groups at the same midline and parietal electrode sites. Statistically, mean and peak amplitudes for the P300 were extracted from the four electrodes mentioned earlier (i.e., Cz, Pz, PCz, & POz) and used to compare the clinical and control groups for four target types (i.e., target-after-fear, target-after-sad, target-after-neutral, & target-after-target). Specifically, an amplitude value was taken at intervals of 4 ms for each epoch (i.e., time window) for all variables between the time window of 250 and 550 ms. An

overall mean and peak amplitude was calculated from the combined epochs for each target (i.e., target-after-fear, target-after-sad, target-after-neutral, target-after-target) at each electrode site (i.e., Cz, PCz, Pz, POz). RT was recorded for distracter (i.e., fear, sad, & neutral) and target type (i.e., target-after-fear, target-after-sad, target-after-neutral, target-after-target). Accuracy, the percentage of times each participant accurately pressed the correct button that matched the correct stimulus, and false alarms, the percentage of times each participant pressed either button before the stimulus was presented, was calculated for each participant.

2.4 Experimental Procedures

The oddball trials, the infrequent distracters and oddball target stimuli, were divided into 4 sets of 25 trials and 1 set of 24 trials. For each trial, the negative distracter oddball trials were pseudorandomized, meaning that no more than two trials of the same valence type were seen successively, to avoid eliciting mood states. Each trial involved the presentation of one stimulus (i.e., infrequent or target) for 750 ms and a fixation screen for 1250 ms. The inter-trial interval was 2 seconds. To prevent an anticipatory effect in participants, the interval between the infrequent stimuli (i.e., infrequent distracters and target) was randomized on an exponential distribution with a median of 8 seconds and a range between 6 and 10 seconds. The task for each participant was to determine whether the stimulus was a target or non-target by pressing a right or left button. For each target (i.e., circle), participants were directed to make a right hand button press and for all other stimuli (i.e., frequent and infrequent distracters), participants were instructed to press a left hand button press. Participants were told to press either button as soon as an image was visually presented and to be as accurate as possible, and to experience any feelings and thoughts that the pictures may elicit. See **Figure 2** for a depiction of the task design.

2.5 Statistical Analyses

Clinical and healthy controls were analyzed using Statistical Package for Social Sciences (SPSS) for behavioral RT & ERP (i.e., P300) mean and peak amplitude for distracters and targets using four separate within-subjects design analysis of variance (ANOVA). The between subjects variable for each ANOVA was group, clinical and healthy control. The within subjects variable for the target ANOVA was target type (i.e., target-after-sad, target-after-fear, target-after-neutral, target-after-target). The within subjects variable for distracter ANOVA's was distracter type (i.e., sad, fear, and neutral). A one-way repeated measures ANOVA was run for within group comparisons for distracter type for RT, and three one-way repeated measures ANOVA were run for within group comparisons for target data for RT and ERP (i.e., for mean and peak amplitude). The distracter and target ANOVA's evaluated responses for the infrequent distracter (i.e., sad, fearful, and neutral) and the target and preceding infrequent distracter compilations (i.e., target-after-sad, target-after-fear, target-after-neutral, and target-after-target), respectively. The Greenhouse-Geisser correction was reported for all p-values that did not meet the sphericity assumption, meaning that the Mauchly's Test of Sphericity was significant; this means that the levels of within-subjects variable did not have equal variance and the different levels of within-subjects variables were not correlated to the same extent, and therefore, required an Epsilon correction such as the Greenhouse-Geisser correction (Gamst, Meyers, & Guarino, 2008).

CHAPTER III – Results

3.1 RT for Distracter (Hypothesis 1) and Target types (Hypothesis 2)

Hypothesis one: There was a significant increase in processing time for fearful distracters, with longer RTs in both clinical and healthy control groups. A one-way repeated

measures ANOVA was run for RT for the within groups comparison of Distracter type. The Distracter type was statistically significant under the Greenhouse-Geisser correction, $F_{(1.420, 80.912)} = 21.537$, E = .710, $p < .05$, $\eta^2 = .274$. Pairwise comparisons were done using a Bonferroni correction, with an alpha level of .05, which showed significantly longer RTs for fear, $p < .05$, and neutral, $p < .05$, distractors than sad distracters (i.e., fear > neutral > sad). There were no differences between the sad and neutral distracters, p = .299. There were no differences between clinical (n=42) and control (n=17) groups for RT distracter, $F_{(1, 57)} = .141$, p = .709 or Distracter type x Group interaction, $F_{(1.420, 80.912)} = .445$, p = .575. See **Table 2** for the RT mean and standard error for both clinical and healthy control groups.

Hypothesis two: A one-way repeated measures ANOVA was run for RT for the within groups comparison of Target type. There were no differences in RT between clinical (n=42) and control (n=17) groups for Target type, $F_{(1,57)} = .736$, p = .395. There were no differences in RT for Target type (i.e., target-after-fear, target-after-neutral, target-after-sad, and target-after-target), $F_{(3, 171)} = .592$, p = .621 or the Target type x Group interaction, $F_{(3, 171)} = .190$, p = .903.

3.2 Accuracy and False Alarms

Participants had a high overall average of accuracy for both the Target and Distracter types and a low overall average of false alarms. The overall mean for Target type accuracy was 87.32% (SD = 12.46%). Separately, the mean and standard deviation for Target type accuracy were target-after-fear, 89.3% (12.44%), target-after-neutral, 87.22% (12.66%), target-after-sad, 88.14% (13.03%), and target-after-target, 84.51% (11.73%). The overall mean for Distracter type was 97.93% (SD = 5.22%), and specifically, fear distracters, M = 98.24%, SD = 5.55%; neutral distracters, M = 97.69%, SD = 5.06%; sad distracters, M = 97.86%, SD = 5.06%. The overall mean and standard deviation for false alarms was M = 1.80% (SD = 4.67%); false alarms mean

and standard deviation for each condition were similar (i.e., fear distracters, M = 1.30%, SD = 4.55%; sad distracters, M = 1.96%, SD = 4.65; and neutral distracters, M = 2.14%, SD = 4.80%). Overall, the high mean accuracy and low false alarm results suggest that the data is a fair depiction of the participants ability.

3.3 Mean Amplitude ERP (P300) Results for Target type (Hypothesis 3 & 4)

Hypothesis three and four: A one-way repeated measures ANOVA was run for mean amplitude, of the P300 ERP, for the within groups comparison for Target type. There were no differences in the mean amplitude ERP data for Target type for the midline placement, Cz, $F_{(1.562, 71.869)} = .217$, E = .521, p = .750 or for the Target type x Group interaction, $F_{(1.562, 71.869)} = .337$, p = .661. Nor were there any statistical differences in the parietal ERP data for Target type, the parietal-central (**PCz**), $F_{(1.482, 68.184)} = .502$, E = .494, p = .552, parietal **(Pz)**, $F_{(2.296, 105.608)} = .764$, E = .765, p = .485, or parietal-occipital **(POz)** areas, $F_{(2.303, 105.946)} = .616$, E = .768, p = .564. There were also no differences in the Target type x Group interactions for all parietal data: the parietal-central **(PCz)**, $F_{(1.482, 68.184)} = .437$, p = .588, the parietal **(Pz)**, $F_{(2.296, 105.608)} = .085$, p = .939, or the parietal-occipital **(POz)** sites, $F_{(2.303, 105.946)} = .155$, p = .883. See **Table 3** for the mean ERP amplitude and standard error for the P300. See **Figure 3** to **Figure 6** for graphical representations of the mean amplitude for all electrode sites.

There were no differences between the clinical (n=35) or control (n=13) groups for all the ERP data for all electrode sites, midline **(Cz)**, $F_{(1, 46)} = 2.609$, p = .113, parietal-central **(PCz)**, $F_{(1, 46)} = 1.641$, p = .207, parietal **(Pz)**, $F_{(1, 46)} = 2.174$, p = .147, or parietal-occipital **(POz)** sites, $F_{(1, 46)} = .582$, p = .449.

3.4 Peak Amplitude ERP (P300) Results for Target type (Hypothesis 3 & 4)

Hypothesis three and four: A one-way repeated measures ANOVA was run for peak amplitude, of the P300 ERP, for the within groups comparison for Target type. There was no main effect of Target type for peak amplitude ERP data at the midline, **Cz,** $F(_{3, 138}) = .327$, $p = .806$, nor a significant interaction between Target type x Group for the midline, **Cz**, $F(_{3, 138}) = .724$, $p = .539$. Also, all parietal electrodes provided insignificant results for the peak amplitude ERP data for all Target types (ie., target-after-fear, target-after-sad, target-after-neutral, and target-after-target): the parietal-central electrode, **PCz**, $F(_{3, 138}) = .621$, $p = .603$, parietal electrode, **Pz**, $F(_{3, 138}) = .564$, $p = .640$, and the parietal-occipital electrode, **POz**, $F(_{3, 138}) = .377$, $p = .770$. The Target type x Group interactions for all parietal electrodes were also not significant: parietal-central electrode, **PCz**, $F(_{3, 138}) = .870$, $p = .459$, parietal electrode, **Pz**, $F(_{3,138}) = .193$, $p = .901$, and parietal-occipital, **POz**, $F(_{3, 138}) = .189$, $p = .904$. See **Table 4** for the mean peak amplitude results and standard error for the P300 ERP.

There were no differences between the clinical (n=35) or control (n=13) groups for all electrode sites, midline **(Cz)**, $F(_{1, 46}) = 2.147$, $p = .150$, parietal-central electrode **(PCz)**, $F(_{1, 46}) = 2.102$, $p = .154$, parietal electrode **(Pz)**, $F(_{1, 46}) = 2.508$, $p = .120$, and parietal-occipital electrode **(POz)**, $F(_{1,46}) = .718$, $p = .401$.

3.5 Summary of Results

Overall, this current study provided information into how adolescents processed emotional stimuli, as fear was processed significantly slower than sad and neutral stimuli in both the clinical and control groups. There were no significant differences in behavior between the clinical and control groups, meaning that emotion was processed similarly in both groups. The target type behavioral data showed no significant differences between the target type conditions (i.e., target-after-fear, target-after-neutral, target-after-sad & target-after-target). This suggests

that emotion played a similar role in how each group responded to the target after each emotional stimulus. For the ERP data, there were no significant differences in the mean or peak amplitude for target type conditions or between the groups. This result suggests that the neural behavior of all participants varied to a similar extent in both the clinical and control groups.

CHAPTER IV – Discussion

The main purpose of this study was to elucidate the behavioural and neurophysiological markers of certain emotions in a clinical population with DSM-V, Axis-l disorders such as affective disorders, ADHD, and CD. We used a modified emotional oddball paradigm to gain RT and ERP data for both clinical and healthy control groups. The participants were asked to respond to emotional stimuli (i.e., fearful and sad distracters) and non-emotional pictures (i.e., circles) that was presented after the emotional stimuli (i.e., target-after-sad, target-after-fear, target-after-neutral, and target-after-target). ERP data and RT was recorded for all responses to emotional or non-emotional pictures. Overall, four results were found in the current study. First, we found that the overall mean of all participants, both clinical and control groups, showed slower RTs to fearful distracters compared to neutral and sad distracters. This result was similar to a prior study done in the same lab (Singhal et al., 2012). Second, we also found that there were no statistical differences in both the clinical and control groups behavioral response to target types. Third, the ERP data provided non-significant results for both groups in all brain regions tested. Fourth, there appeared to be a difference in the ERP morphology of the clinical and control groups, suggesting a potential difference in processing emotion. Overall, the ERP data did not provide the expected differences between emotion processing and emotion regulation in

either the clinical or control groups, as there were no significant differences found. For future research, looking at the potential limitations of the current study may allow any confounds to be alleviated and possible differences to be divulged.

4.1 Significant RT for Distracter Type (Hypothesis 1)

Emotion regulation is affected by the intensity, frequency and types of emotion being processed (Gross and Jazaieri, 2014). Regulatory choice is decided by motivational factors and regulatory strategy complexity which are affected by prior experience, personality and cognitive ability (Burns et al., 2008; Mauss & Butler, 2010; Rossi & Pourtois, 2011; Sheppes et al., 2012, 2013, 2015; Webb et al., 2012). Research suggests that emotions and cognitive processing influence each other and can have positive or negative effects (Cole et al., 2004; Hart et al., 2010; Pessoa et al., 2002, 2005; Tamietto & de Gelder, 2010). There are several factors that determine whether the interaction between cognitive and emotional processing produces positive or negative effects; these factors are: individual differences, cognitive load, level of threat, and the availability of cognitive resources (Cohen & Henik, 2012; Gupta & Raymond, 2012; Gupta et al., 2016; Kanske, 2012; Kim et al., 2012; Okon-Singer et al, 2013; Pessoa, 2009). For example, emotional stimuli can impact cognitive processing in a negative manner such that it can slow reaction time for perceptual tasks, diminish the capability of working memory performance, and reduce accuracy in tasks (Blair et al., 2007; Dolcos & McCarthy, 2006; Denkova et al., 2010; Gupta & Deak, 2015; Hart et al., 2010; Uher et al., 2014). My hypothesis, based on current research, was that the RT to emotional stimuli would be faster for the clinical group in comparison to the control group. In the present study, both the clinical and control group responded slower to fearful stimuli than to all other stimuli. Therefore, the distracter pictures that portrayed fear stimuli may have impacted the participants cognitive processes in a negative

manner. The slower RT could be due to more attention being gained for the fearful stimuli and an inability to disengage attention as fast as both groups did to all other stimuli. Or the slow RT in both groups was due to needing more time to appropriately regulate their emotion to the fear stimuli in comparison to all other distracters. Behaviorally, since both groups responded similarly to the fearful distracter stimuli, this suggests that overall both groups took longer to process the fearful stimuli and make a decision as to what category the stimuli fell under (i.e., which button to press). Similar to the present study, another study in the same lab also found that clinical and controls groups had longer RTs to fearful pictures compared to sad and neutral stimuli (Singhal et al., 2012).

Statistically, there were no differences between the clinical and control groups for RT accuracy and false alarms for both distracter and target types. Accuracy measures how often the participants match the button press with the correct stimuli. False alarms measure how often participants press the button before the stimuli is presented. When splitting the groups and analyzing their data separately, the clinical group was significantly different for target type accuracy, $p = .041$. Participants were less accurate for target-after-target stimuli than the two emotional target types, target-after-sad and target-after-fear (i.e., target-after-target<target-after-neutral<target-after-sad<target-after-fear). This result suggests that targets after emotional pictures (i.e., fear and sad) did not negatively affect the participants accuracy.

4.2 No Significant Results for RT or ERP for Target Type (Hypothesis 2 and 3)

The P300 is a positive ERP component that is recorded at parietal sites, occurring between 250 - 600ms after a stimulus occurs; it is elicited by a decision that is influenced by prior experiential memories (Campanella & Phillippot, 2006; Hajcak et al., 2010; Kok, 2001; Polich, 2007; Ranganath & Rainer, 2003; Rotshtein et al., 2010; Singhal et al., 2012). P300's can

be elicited by an emotional stimulus and is influenced by extrinsic and/or intrinsic motivation of the individual in conjunction with the effects of the stimulus (Carretie et al., 1997; Hajcak et al., 2010; Krolak-Salmon et al., 2001; Luck, 2014; Olofsson et al., 2008). Amplitude and latency (RT) differences are seen in the P300 typically when the complexity, vigilance, arousal level, task-relevance or motivational significance is altered (Campanella et al., 2002; Hansenne, 2000). P300 latency is generally thought to be the result of mental processes or decisions made in the emotional oddball paradigm (i.e., reaction time) whereas the P300 amplitude is thought to be related to the intensity of the emotional processing (Kok, 2001). Emotion research typically find similar results with emotionally intense or highly arousing stimuli producing larger P300 ERPs (Briggs & Martin, 2009; Delplanque et al., 2004, 2005, 2006; Keil et al., 2002; Moore et al., 2019; Olofsson & Polich, 2007; Polich, 2007; Rozenkrants & Polich, 2008). The greater neural response and faster reaction time is typically exhibited to both positive and negative emotional stimuli when compared to neutral stimuli (Krolak-Salmon et al., 2004; Lindquist et al., 2012; Luo et al., 2010; Pourtois et al., 2010; Rozenkrants & Polich, 2008; Schupp et al., 2003a, b; Schupp, Cuthburt et al., 2004; Schupp et al., 2006; Weinberg & Hajcak, 2010). Prior research seems to suggest that the clinical population typically responds differently when compared to healthy control groups. In regards to RT, anxious and depressed groups have faster RTs to negative stimuli (Campanella & Phillippot, 2006; Rossignol et al., 2005, 2008) and groups with ADHD diagnoses have longer RTs to emotional stimuli (Lopez-Martin et al., 2013). ERPs also tend to show differences in a clinical group when compared to healthy control groups. In healthy controls, P300 ERPs are larger for target type in emotional oddball tasks (Moore et al., 2019). ERPs (i.e., P300) appear earlier and tend to be larger in anxious participants when they are confronted with emotional stimuli in comparison to controls (Bar-haim et al., 2005; Campanella

& Philippot, 2006; Liddell et al., 2004; Rossignol et al., 2005, 2008). Clinical groups with depression diagnoses also showed a similar result to negative stimuli, with larger P300 amplitude than healthy controls (Ilardi et al., 2007). Child and adolescents with ADHD and CD show smaller P300 amplitude compared to healthy controls for emotional stimuli (Alexander et al., 2008; Bauer & Hesselbrock, 2003; Kim et al., 2001). My second hypothesis was that the clinical group will find it difficult to disengage from and appropriately regulate emotional information as fast as the controls, suggesting that the reaction time for stimuli after emotional pictures will be longer. In the present study, differing RT in clinical and healthy controls were not seen as there were no significant differences in RT for target types or between the groups. This suggests that both the clinical and healthy control group responded similarly to each emotional stimuli as there were no differences to the targets after emotional stimuli or between the groups. My third hypothesis was that the clinical group would elicit a larger overall P300 in response to the emotional content when compared to the control group. I suggested that the ERP would be larger due to the clinical groups reactivity to the emotional stimuli. For all ERP data, there was no statistical differences in response to target type nor were there differences seen between the clinical and healthy control groups. This suggests that there was no significant variability in how each group emotionally responded to the target after emotional stimuli. A similar result was also seen for RT and ERP (i.e., P300) for target type in a previous study done in the same lab (Singhal et al., 2012). The implications that both the mean and peak ERP amplitude produced non-significant results is indicative of possible limitations in the study, as the variability in how the mean and peak amplitude is measured should have unveiled differences in at least one of the measures. See the limitations section below for possible reasons.

4.3 ERP Morphology (Hypothesis 4)

Research suggests that people with mental health disorders such as affective disorders, ADHD, and CD, struggle with emotion processing and/or emotion regulation (Aldao et al., 2010; Euler et al., 2014; Gross, & Jazaieri, 2014; Jazaieri et al., 2013; Kring, 2010; Walcott & Landeau, 2004; Werner & Gross, 2010). Emotion processing problems or dysregulation can occur either in the awareness of stimuli or the response to the stimulus (Gross, 2002). ERP studies have linked this emotional processing/regulation deficit with P300 alterations (Alexander et al., 2008; Dawel et al., 2012; Hansenne, 2000). Individuals with affective disorders show increase attention towards negative information (Armstrong & Olatunji, 2012; Bar-Haim et al., 2007; Campanella & Philippot, 2006; Carretie et al., 2004b; Cisler & Koster, 2010; Denkova et al., 2010; De Raedt & Koster, 2010; Frewen et al., 2008; Mogg & Bradley, 2002; Rossignol et al., 2013; Williams, 2006; Yiend, 2010). Individuals with ADHD and/or CD show undercontrolled emotion regulation, seen in poor inhibitory control for attention, behavior and emotions (Eisenberg et al., 2001; Euler et al., 2014; Kim & Cicchetti, 2010; Sterzer et al., 2005). Shafer and colleagues (2019) found differences in the white matter tracts of adolescents with mental health disorders when compared to healthy controls, associating structural differences in the brain to psychopathology. This suggests simply that structural differences in the brain affect the functional ability of the brain. My fourth hypothesis is that the clinical group would show a different ERP morphology overall, with earlier and larger P300's, when compared to the control group. The research stated above suggested that we would see a difference in behavior and neural responses to emotional tasks between mental health disorders and healthy controls, but we did not see this replicated in our present study. Looking at statistical trends and the mean amplitude in graphical form (**see Figure 3-6**), there is a general trend that suggests potential differences between the groups. For example, the difference between the clinical and control

groups for the mean amplitude for target type had generally low p-values: midline (Cz), p = .113; parietal-central (PCz), p = .207; parietal (Pz), = .147, and parietal-occipital (POz), p = .449. The reasons we may not have seen any differences may be due to the potential limitations of the study which are noted below.

4.4 Limitations

Our study provided some similar results to a previous study (Singhal et al., 2012), which offers some information about adolescent emotional processing and regulation, but at the same time, there are some results that appear incongruous with past research suggesting there may be limitations that need to be considered for future research in this area. I have suggested in the limitations section that fewer diagnoses, more ERP epochs per condition, an understanding of medications and their possible confounds, alternate electrode placements, more participants for healthy controls, a normal distribution of gender, and a smaller age range to study adolescents' behavior and neural responses may help with potential confounds in future studies.

The clinical group had multiple diagnoses which may be a possible reason for the lack of significant results seen between the two groups. In the research, there tended to be differing behavioral and neural results seen from affective and behavioral disorders (see discussion above, section 4.2), and the clinical group in our study had multiple diagnoses. For example, one participant had multiple diagnoses of both affective and behavioral disorders, such as MDD, ADHD inattentive type and PTSD, and there were other participants with multiple diagnoses representing both affective and behavioral disorders. Multiple diagnoses among the participants may be a potential limitation for this present study.

Providing more epochs per condition may have provided more statistical power to the present study. When data cleaning, there were many epochs that could not be included because

of artifacts such as eye blinks and body movements, therefore, several participants could not be used in the statistical analysis because they did not have the required number of epochs (i.e., 5 or more) to be included. This could be mitigated by making the emotional oddball paradigm longer, comprised of more opportunities to respond to both distracters and target types. Providing more pictures for participant's to respond to may raise the probability of having clean epochs without eye blinks or body movements and add to the overall epoch count; more epochs provide more information for statistical analysis and more statistical power to the overall study. The contention in deciding how many epochs per condition is ultimately determined by what is required for the study and then how long to make the task overall to get the best results, as making the task too long tires or bores the participants so that they are unable to give their best response. Only requiring 5 epochs per condition may have contributed to the lack of results seen in the current study as there is the potential with less epochs to have more noise compared to the ERP signal, thus biasing the results, therefore, requiring more epochs increases the probability of capturing the appropriate ERP signal by averaging out the noise (Luck, 2005).

The clinical group was on different medications such as stimulants and anti-depressants which may alter the typical emotion processing and/or emotion regulation that would be present in participants with Axis-I diagnoses. Recent research suggests that psychopathology is linked to reductions in white matter in the brain, but there is also the potential that drugs used to treat mental health disorders may impact the structure of the brain as well (Chiang et al., 2015; Shafer et al., 2019). For example, Marrus and colleagues (2014) reviewed the effects of different psychotropic drugs on brain matter. They found that some drugs increased cell genesis, but many drugs decreased volumes of both grey and white matter in the brain. Medications may potentially

confound the information collected for these individuals and may be another reason why there were no statistical differences in the ERP results for this group.

Electrode placements to pick up maximal P300 coverage may be different for adolescents when compared to adults. For this study, I chose to look at 4 electrodes (Cz, midline, PCz, parietal-central, Pz, parietal, and POz, parietal-occipital; **see Figure 1**), using earlier research to guide where the P300 is typically elicited, which has been typically done on adults (Campanella et al., 2013; Polich, 2007; Singhal et al., 2012). Also, in recent fMRI-EEG research, looking at the ventral and dorsal neural circuitry responsible for emotional and attention processing, respectively, they found that the P300 also had contributions from the dorsal neural circuitry. This information is not only consistent with prior research on the P300, as it is known to play a role in attention and working memory, but it can also provide alternative placements for where the P300 is maximally picked up from electrodes across the brain. In short, more ERP research needs to be done for adolescents, as they are going through structural changes in their brain throughout their adolescent years, and electrode placements that maximally elicit the P300 may be different when compared to adult electrode placements.

To provide more statistical power to the study, all groups should meet the recommended group size. According to the Central Limit Theorem, the behavioural (n=42) and ERP (n=35) data that resulted from the clinical group was significantly large enough to be a normally distributed sample that is representative of the larger clinical population as it was over the recommended size of 30 participants. The control group did not meet this criteria, as the sample size was considerably less for behavioral (n=17) and ERP (n=13) data, due to eliminating participants from analysis because they did not meet expected requirements, and may not be

representative of the larger mentally healthy population. This could be a consideration of why the ERP data was not statistically different for the clinical and control groups.

There was a large difference in the amount of males and females in both the clinical and control group for the ERP analysis which may have contributed to the null result. Specifically, there were 35 participants in the clinical group, 13 females and 22 males, and 13 participants in the control group, 10 females and 3 males. The reason gender may play a role in the results is because females and males may respond differently, either neurally and/or behaviorally, to emotional stimuli (Campanella et al., 2004). The research suggests that females may have greater P300 responses compared to males (Steffensen et al., 2008), and males may have quicker reaction times (Tsolaki, Kosmidou, Hadjileontiadis, Kompatsiaris, Tsolaki, 2015). Li and colleagues (2008) found that females are more sensitive in identifying negative facial emotion in comparison to males. Adolescent twin studies also showed gender differences in the P300, with larger P300 amplitudes for adolescent females up to 17 years of age (van Beijsterveldt, Molenaar, Geus, & Boomsma, 1998). These results switched for the ages of 17 and 18, as the P300 was larger for adolescent males at these age points. Bilalpur and colleagues (2017) found differential cognitive processing for negative emotions between the sexes, as females showed a higher sensitivity to negative emotions than males, which was replicated in stronger P300's for females compared to males. These results suggest that gender differences may impact ERP results, and therefore, may need to be considered for future emotional research.

The large age differences, 12-17 years, and the dynamic changes (i.e., pubescent and neural) that occur in adolescents through this time period may have contributed to the null ERP results in the present study (Blakemore, Burnett, & Dahl, 2010; Somerville, Jones, & Casey, 2010). During adolescence, the brain structurally and functionally changes, by increasing the

volume of white matter and learning to inhibit impulsive behaviours, respectively (Shafer et al., 2019; Paus, 2005). Adolescence is also a period of time where there is large changes in emotional development. As adolescents age, their ability to emotionally regulate appropriately increases (Kesek, Zelazo, & Lewis, 2008). This suggests that emotion regulation may not be a static variable in this study, and therefore, may be a limitation because of the many changes that occur during adolescence. Keeping the age range smaller for future studies may potentially provide a clearer picture of emotional processing and regulation in adolescents.

CHAPTER V - Conclusion

This study provided some additional information about emotion regulation and processing in adolescents, ages 12-17, with DSM V Axis-I disorders such as ADHD, CD, and affective disorders. The RT for fearful stimuli (i.e., distracter types) was slower compared to neutral and sad stimuli for both clinical and control groups, suggesting that both groups processed fearful information slower than all other stimuli. There were no differences in RT time for target types (i.e., non-emotional stimuli after emotional information) suggesting that both groups were able to regulate emotional information to the same extent. There was also no difference in the P300 mean and peak amplitudes amongst the clinical and control groups, suggesting that they processed emotional information similarly. The lack of reactivity to emotional stimuli in our clinical group was atypical as previous research has suggested that mental disorders such as anxiety and depression have reactive amygdala responses compared to healthy controls. Since some of the same results were seen earlier in a similar study done in the same lab (Singhal et al., 2012), it may suggest that there are some limitations in our present and past study or there are no differences in emotional modulation of attentional processing for

adolescents, 12-17 years of age, with DSM V Axis-1 disorders. Either way, more research needs to be done to elucidate emotional processing and regulatory information in adolescents. This study and succeeding studies like it, can provide valuable insights into what emotion processing and regulation is like for adolescents with mental health disorders. Additive information to this field may provide practitioners with more efficacious treatments and interventions to help adolescents with their emotional problems.

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APPENDIX

Table 1. Diagnoses and Medications

		Medication (number of patients)			
Diagnosis:	**Number (male/female)**	**Unknown**	**Stimulants**	**Anti-depressants**	**Other**
ADHD CO-MORBID WITH ONE OR MORE FOLLOWING DISORDERS:					
OCD, PCRP, ODD, SRC, RAD, MDD, GAD, CD, learning disorders, tourette's disorder, mood disorder, anxiety disorder, nocturnal enuresis, substance abuse, social phobia, bipolar disorder, developmental coordination disorder, borderline personality disorder	(15/6)	3/3	6	SSRI-2 NRI	SNRI Antipsychotics -1 Atypical Antipsychotics -2
ONE OR MORE AFFECTIVE DISORDERS (major depression, dysthymia, anxiety (GAD, PTSD, social phobia) CO-MORBID WITH ONE OR MORE OF THE FOLLOWING DISORDERS:					
PCRP, SCR, RAD, ODD, CD, FASD, sexual abuse, expressive language disorder, central auditory processing deficit, gender identity disorder	(5/11)	2/3	3	SSRI-2	Atypical Antipsychotics -2 Antipsychotics -1
AFFECTIVE DISORDERS:					
Major depression, PTSD	(1/1)	0/0	2	SSRI-2	-
ADHD:					
	(1/0)	0/0	-	-	-
OTHERS: TWO OR MORE FOLLOWING DISORDERS:					
ODD, PCRP, CD, adjustment disorder	(2/4)	1/0	1		SNRI SSRI-2
DIAGNOSIS UNKNOWN:	(7/2)	3/2	1	SSRI-2	Atypcial antipsychotic -2
TOTAL:	55 (31/24)	9/8	13	SSRI-2 NRI	SSRI-2 SNRI Antipsychotics -1 Atypcial Antipsychotics -2

ADHD, attention-deficit/hyperactivity disorder; OCD, obsessive-compulsive disorder; PCRP, parent-child relation problem; ODD, oppositional defiant disorder; SRC, sibling-relational conflict; RAD, reactive attachment disorder; CD, conduct disorder; FASD, fetal alcohol spectrum disorder; PTSD, post-traumatic spectrum disorder; MDD, major depressive disorder; GAD, generalized anxiety disorder; SSRI, selective serotonin reuptake inhibitors; NRI, norepinephrine reuptake inhibitors.

Table 2: Mean reaction time (RT) and standard error (SE) to distractors and targets

Distractor Type	Group	Fear	Sad	Neutral
RT Mean (SE)	Clinical n = 42	618. 59 (24.73)	565.75 (18.35)	566.67 (18.92)
	Healthy Control n = 17	604.31 (44.42)	541.01 (40.51)	559.70 (41.67)

Target Type	Group	Target-after-fear	Target-after-sad	Target-after-neutral	Target-after-target
RT Mean (SE)	Clinical n=42	482.28 (14.03)	492.41 (14.40)	485.71 (14.36)	482.00 (13.70)
	Control n=17	465.36 (21.44)	466.26 (22.24)	467.16 (17.86)	459.63 (20.85)

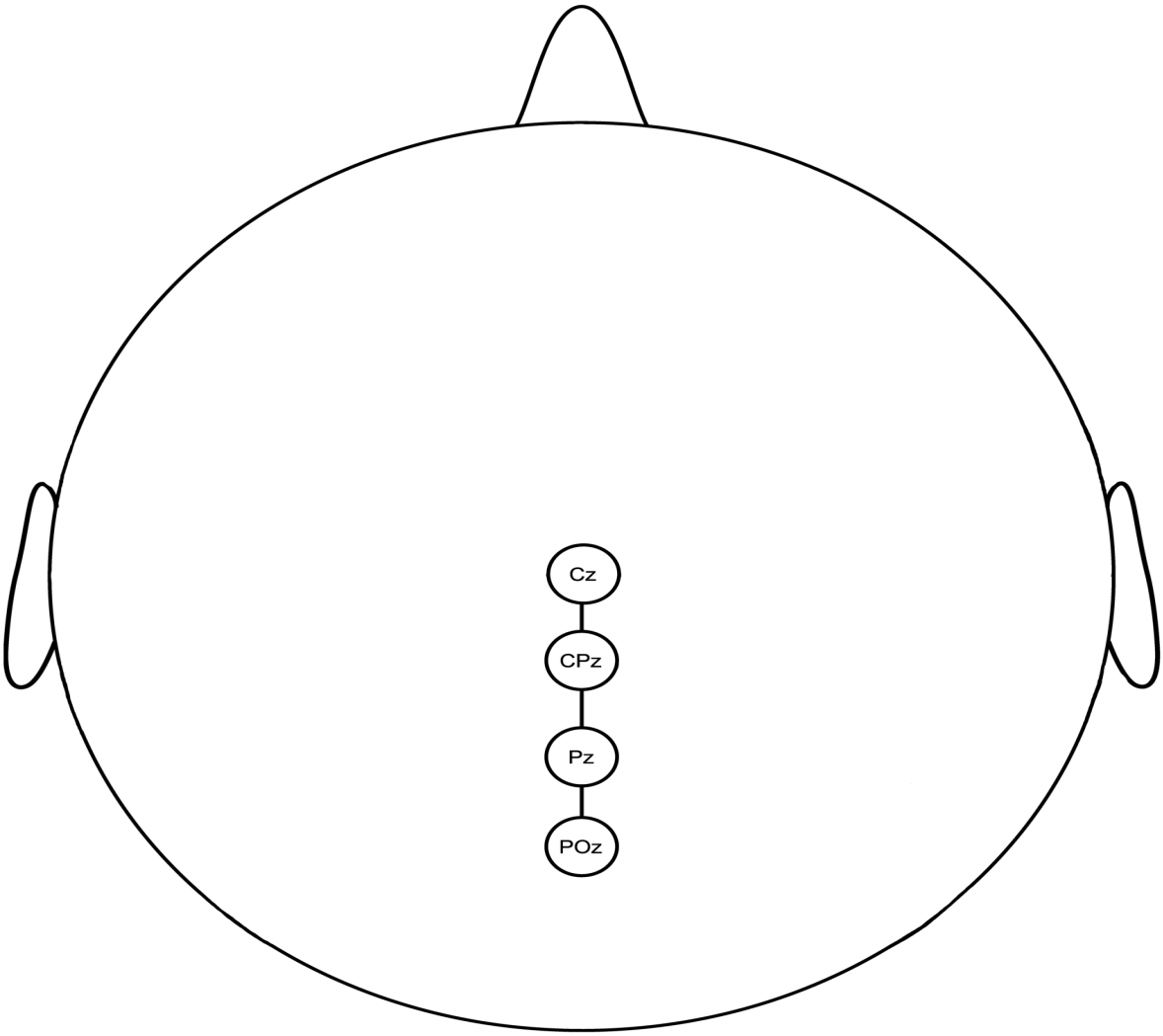
Table 3: Mean ERP amplitudes and standard error (SE) for P300

Target Type	Group	Target-after-fear	Target-after-sad	Target-after-neutral	Target-after-target
P300 midline Cz (SE)	Clinical n = 35	3.20 (.89)	4.82 (1.41)	3.92 (.82)	3.93 (.81)
	Control n = 13	6.33 (2.07)	6.17 (1.49)	6.78 (1.57)	6.54 (1.32)
P300 parietal PCz (SE)	Clinical n = 35	2.75 (2.07)	5.22 (.87)	4.85 (.71)	3.36 (.85)
	Control n = 13	5.76 (1.34)	6.01 (1.40)	6.15 (1.11)	6.42 (1.37)
P300 parietal Pz (SE)	Clinical n = 35	5.64 (1.60)	6.80 (.94)	6.98 (1.06)	6.37 (1.24)
	Control n = 13	8.73 (1.85)	9.26 (1.78)	10.34 (1.99)	9.00 (1.60)
P300 parietal POz (SE)	Clinical n = 35	6.25 (1.52)	6.06 (.90)	6.82 (1.14)	5.96 (1.22)
	Control n = 13	8.29 (2.17)	6.81 (1.99)	8.58 (2.38)	7.56 (1.71)

Table 4. Mean peak ERP amplitudes and standard error (SE) for P300

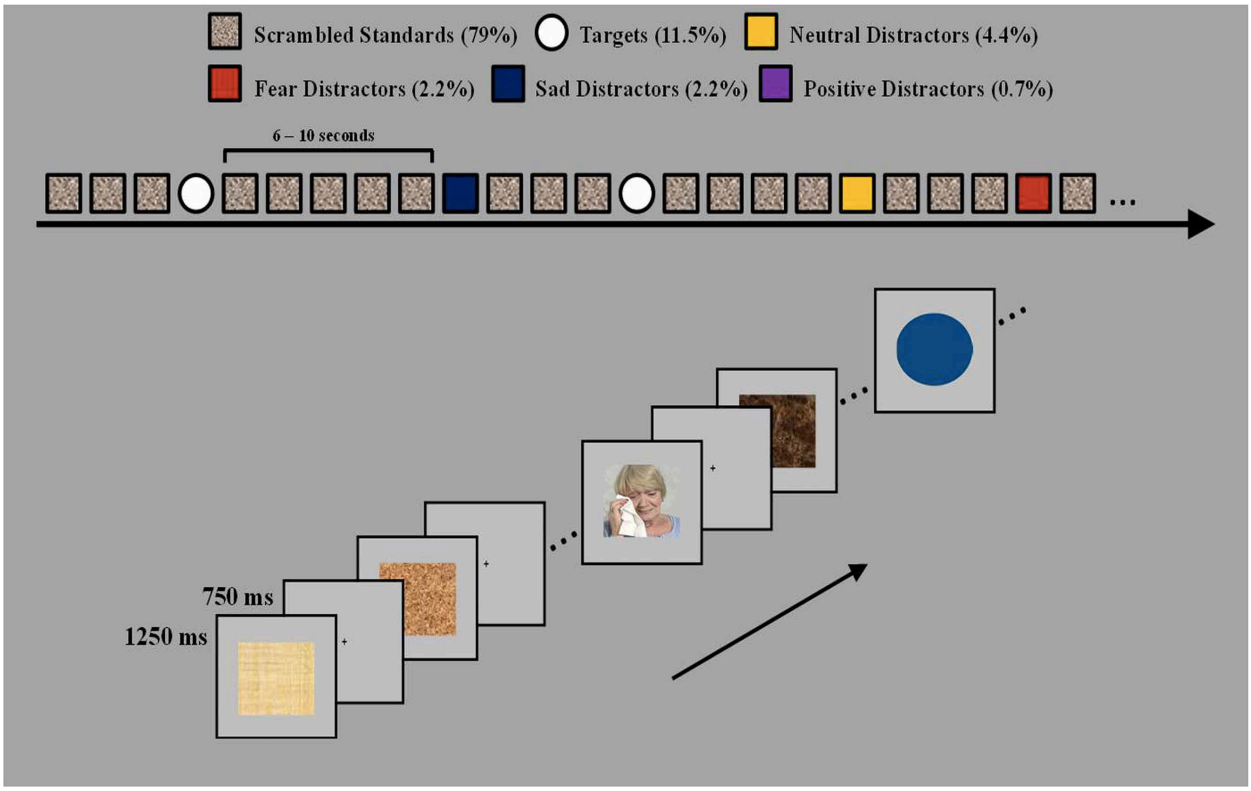
Target Type	Group	Target-after-fear	Target-after-sad	Target-after-neutral	Target-after-target
P300 midline Cz (SE)	Clinical n = 35	8.62 (1.17)	10.42 (1.82)	8.91 (1.12)	8.51 (1.00)
	Control n = 13	13.20 (2.28)	11.57 (1.55)	11.69 (1.79)	11.54 (1.57)
P300 parietal PCz (SE)	Clinical n = 35	6.88 (1.93)	10.04 (1.11)	8.76 (.88)	6.91 (.77)
	Control n = 13	10.74 (1.51)	10.71 (1.56)	9.48 (1.32)	10.64 (1.72)
P300 parietal Pz (SE)	Clinical n = 35	11.29 (1.40)	12.41 (1.20)	11.78 (1.25)	11.17 (1.08)
	Control n = 13	15.58 (2.71)	15.59 (2.30)	14.64 (2.33)	14.08 (1.82)
P300 parietal POz (SE)	Clinical n = 35	11.76 (1.30)	11.46 (1.19)	11.50 (1.21)	10.72 (1.14)
	Control n = 13	14.02 (2.54)	12.49 (2.38)	12.99 (2.63)	13.09 (2.00)

Figure 1. Electrode Placements



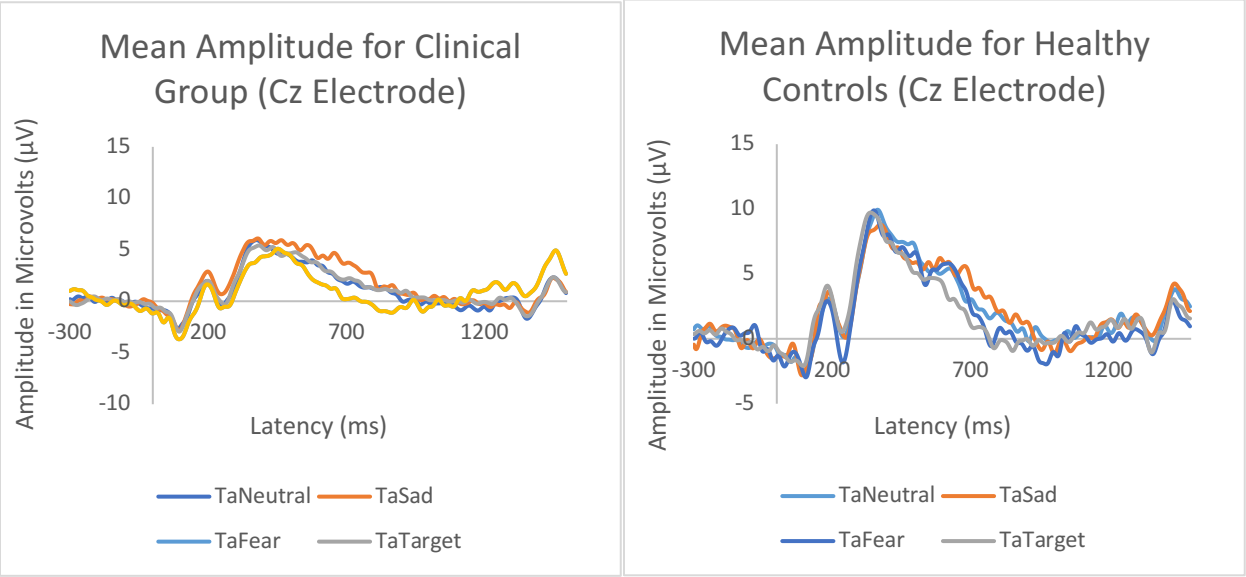
Electrodes: **Cz**, midline; **CPz**, Central-Parietal; **Pz**, Parietal, & **POz**, Parietal-Occipital.

Figure 2. Task Design



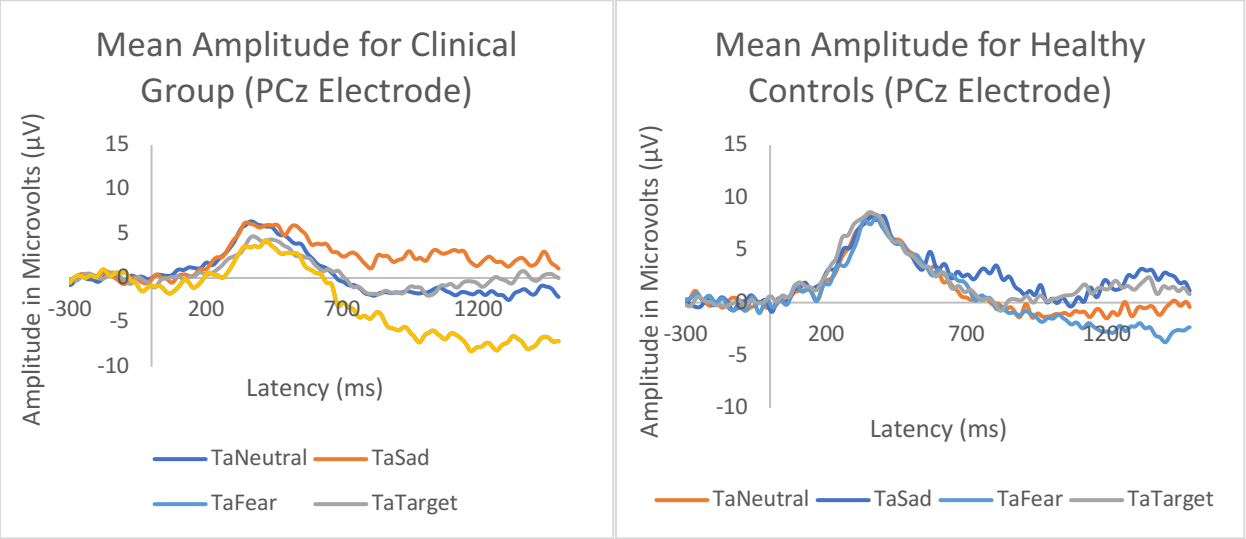
The task used three infrequent stimuli and an oddball target: fear, sad, and neutral distracters and target circles of varying size and color. These stimuli were presented pseudorandomly between pictures that were scrambled, at intervals of 6-10 s. Participants were asked to make a right hand button press for all target stimuli and a left hand button press for all other stimuli.

Figure 3. Mean Amplitude for Clinical and Control groups at the Midline electrodes (Cz)



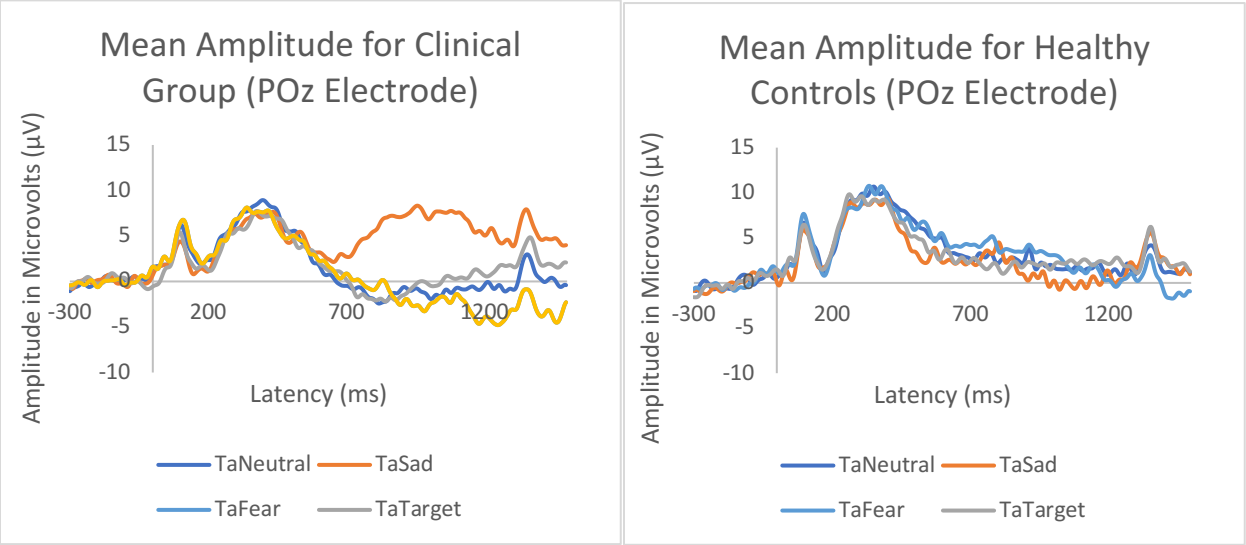
Grand-average waveforms for clinical and control groups for target stimuli at the midline electrode (Cz) showing no significant differences between the groups or target types. *TaNeutral, Target-after-Neutral; TaSad, Target-after-Sad; TaFear, Target-after-Fear; TaTarget, Target-after-Target*

Figure 4. Mean Amplitude for Clinical and Control Groups at Parietal-Central Electrodes (PCz)



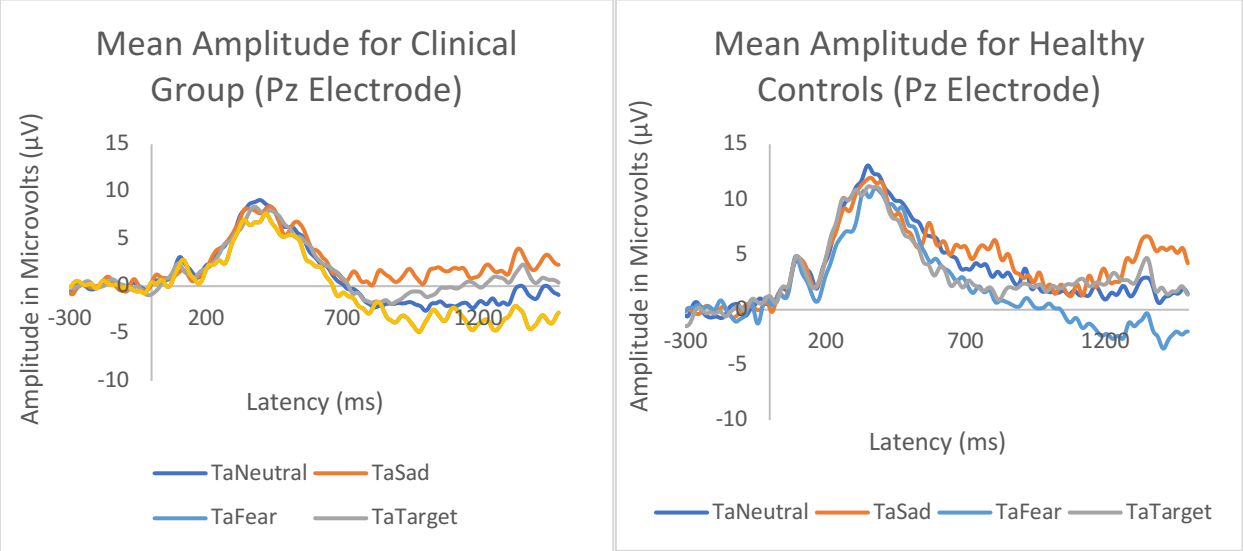
Grand-average waveforms for clinical and control groups for target stimuli at the parietal-central electrodes (PCz) showing no significant differences between the groups or target types. *TaNeutral, Target-after-Neutral; TaSad, Target-after-Sad; TaFear, Target-after-Fear; TaTarget, Target-after-Target*

Figure 5. Mean Amplitude for Clinical and Control Groups at Parietal-Occipital Electrodes (POz)



Grand-average waveforms for clinical and control groups for target stimuli at the parietal-occipital electrodes (POz) showing no significant differences between the groups or target types. *TaNeutral, Target-after-Neutral; TaSad, Target-after-Sad; TaFear, Target-after-Fear; TaTarget, Target-after-Target*

Figure 6. Mean Amplitude for Clinical and Control Groups at Parietal Electrodes (Pz)



Grand-average waveforms for clinical and control groups for target stimuli at the parietal electrodes (Pz) showing no significant differences between the groups or target types. *TaNeutral, Target-after-Neutral; TaSad, Target-after-Sad; TaFear, Target-after-Fear; TaTarget, Target-after-Target*