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Neuropsychological Correlates of Chronic Pain: The Influence of Anxious Arousal on Cognitive Control

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NEUROPSYCHOLOGICAL CORRELATES OF CHRONIC PAIN: THE INFLUENCE OF ANXIOUS AROUSAL ON COGNITIVE CONTROL

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This thesis is dedicated to the numerous individuals managing chronic pain, as well as those who devote their careers to helping individuals suffering from chronic pain live happy and healthy lives.

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ABSTRACT

The present set of studies assessed the relation between neural, psychological, and cognitive mechanisms that have been shown to influence the experience of chronic pain. Specifically, Studies One and Two advanced our understanding of chronic pain by illustrating its unique relationship with anxious arousal and frontal neural activity on inhibitory control difficulties. Study Three focuses on targeting how anxious arousal is distinctly associated with neural correlates of inhibitory control in order to provide a framework that can be applied to individuals experiencing chronic pain. Together, these studies aim to inform the development of evidenced-based interventions that target anxious arousal in order to enhance the effectiveness of chronic pain management. Overall, the findings from this set of studies highlighted the cross-sectional relation between EF, psychological symptoms, and neural mechanisms and specifically distinguished the importance of anxious arousal, inhibitory control, and neural mechanisms measured with EEG (i.e., frontal alpha and beta) and ERP (N200, N450, Conflict SP) methodologies.

CHAPTER ONE

INTRODUCTION

Chronic pain can be classified as recurrent or persistent physical discomfort and is one of the most costly, debilitating, and common conditions affecting individuals of all ages. Chronic pain is prevalent in childhood and adolescence with studies showing chronic pain pervasiveness ranging from 8% - 40% (King et al., 2011). Many children and adolescents with chronic pain may continue to suffer from pain well into adulthood (Fearon & Hotopf, 2001; Walker et al., 2010). Chronic pain affects over 20% of people worldwide and it accounts for 15-20% of all physician visits (Treede et al., 2015). Multiple studies have shown that chronic pain has a detrimental effect on physical and psychological health, daily activity, employment, and economic well-being (Smith et al., 2001). Chronic pain conditions are also associated with a costly societal burden. In the UK, chronic back pain alone accounts for an estimated 45 million days of work lost per year (Smith et al., 2001). In the US, chronic pain is responsible for over 600 million dollars in annual healthcare costs and lost productivity (Zale & Ditre, 2015).

Although definitions vary, chronic pain is typically defined as pain lasting more than three months (Merskey & Bogduk, 1994). It is characterized as pain that persists past indicated healing time and lacks the acute warning function of physiological nociception (i.e., the physiological process by which body tissues are protected from damage; Treede et al., 2015). Chronic pain is a significant health concern that impacts quality of life and several domains

of daily functioning (e.g., missed school days, cognitive functions, psychological health). Studies have found that chronic pain severely affects sleep duration and quality, the ability to exercise, walk, do household chores, attend social activities, and maintain an independent lifestyle (Breivik et al., 2006). Further, individuals with chronic pain reported that their pain impacted their employment status, leading some individuals to switch from full- to part-time work, or change jobs entirely due to their inability to meet job demands (Breivik et al., 2006). Low household income and unemployment are significant socioeconomic sequelae of chronic pain (Johannes et al., 2010). Chronic pain is a public health issue that poses substantial burdens on afflicted individuals.

Despite the significant societal costs, many individuals with chronic pain are not receiving adequate treatment for a variety of reasons, including adverse medication side effects, discussions with medical providers indicating nothing more can be done to alleviate pain, or deciding to live with their pain without additional interventions due to the burden of pursuing other options (Breivik et al., 2006). Medical treatment for the majority of chronic pain patients is often in the form of non-steroidal anti-inflammatory drugs (i.e., NSAIDs), or weak opioid analgesics, that show little short- or long-term relief (Breivik et al., 2006). Current treatments for chronic pain are minimally effective and need to be improved in order to provide clinically significant relief (Breivik et al., 2006).

Theoretical Models of Chronic Pain

Biomedical models of chronic pain have been around for thousands of years (Asmundson et al., 2004), though a grasp on the underlying mechanisms remains elusive. In the early 2000s, researchers primarily focused on the physiological components of chronic pain (Asmundson et

al., 2004), but this biological perspective has fallen short in accounting for the vast individual differences in pain presentation, pain perception, and the propensity to develop chronic pain. More recently, research has focused on understanding the experience of pain through theoretical models that integrate biological and psychosocial factors associated with chronic pain. For example, the fear-avoidance model of chronic pain theorizes that anxiety is the intervening variable between fear and avoidance behavior, such that when an individual is anxious due to anticipated pain or injury, the cognitive (i.e., the narrowing of attention to threat cues), physiological (i.e., autonomic arousal), and motivational (i.e., the desire to prevent anticipated pain or injury) components of anxiety are evoked (Asmundson et al., 2004). Expectancies and attentional shifts may promote hypervigilance for evidence of potential pain or injury, pain-related memories, and pain-relevant schemas. These schemas may be activated to filter and appraise the evidence of pain, and autonomic activation is primed to avoid or protect against the potential threat (Asmundson et al., 2004). Pain-related fear can be negatively reinforced by avoidance behaviors (i.e., avoidance of fearful stimuli may reduce fear in the short-term, but strengthens fear response in the long-term; Zale & Ditre, 2015). By accounting for the components of anxiety (i.e., the cognitive, physiological, and motivational aspects), this model provides an explanation for levels of pain intensity that may not be congruous with actual injury. It further provides explanation for pain that occurs in the absence of identifiable injury or pathology. The fear-avoidance model posits that pain-related fear activates psychological mechanisms that lead to the avoidance of movement and activity (Zale & Ditre, 2015). Although this behavior may be adaptive in the context of acute pain, long-term avoidance of physical

activity may impair functioning, reduce participation in occupational and recreational activities, increase negative mood, and contrive to greater levels of disability (Zale & Ditre, 2015).

While this model has contributed to advancing theories regarding how anxiety contributes to the maintenance of chronic pain through possible pain or injury anticipation contexts, it fails to disentangle the neuropsychological mechanisms by which anxiety perpetuates chronic pain. Further, studies examining the role that other psychological symptoms, such as depression, play in chronic pain have shown variable results (Romano & Turner, 1985). By understanding what factors sustain an individual's experience of living in a body that chronically interprets exteroceptive and interoceptive stimuli as uncomfortable and noxious, we can aim to develop more effective treatments for chronic pain. The three studies included in this dissertation seek to advance our understanding regarding the interrelated nature of the neural, psychological, and cognitive mechanisms associated with chronic pain in order to ultimately inform the development of evidenced-based interventions.

The Role of Anxious (Somatic) Arousal and Cognitive Control in Chronic Pain

Although multiple psychological symptoms are associated with chronic pain, somatic symptoms are an important focus of chronic pain research due to their high rate of co-occurrence with chronic pain (Bettendorf et al., 2008; Latthe et al., 2006). Somatic symptoms have traditionally been defined as physical symptoms of unknown pathology and measured as individual symptoms or as symptom constellations (Dhossche et al., 2001; Steinhausen, 2006). However, this term has been highly criticized due to its diagnostic inflation, or the high probability of misdiagnosing a medical illness, and its inefficacy in capturing the true pain experience those afflicted are facing, despite unknown pathology (Katz et al., 2015). A more

encompassing term, somatic arousal, is synonymous with “anxious arousal” and consists of an enduring pattern of hypervigilance, sympathetic nervous system hyperarousal to mild stressors, and state fear (Sharp et al., 2015). Anxious arousal has historically been conceptualized within the psychopathology literature as a distinct dimension or subtype of anxiety per the framework of the tripartite model of anxiety and depression (Clark & Watson, 1991). The tripartite model, which aimed to explain the co-occurrence between anxiety and depression syndromes, suggests that although anxiety and depression share a substantial component of general affective distress (i.e., negative affect), they can be differentiated on the basis of factors specific to each syndrome (Clark & Watson, 1991). Clark & Watson (1991) theorized that anhedonia, or a reduced ability to experience pleasure and/or diminished interest in engaging in pleasurable activities, was uniquely related to depression, and anxious arousal was hypothesized to be distinct to anxiety. While some studies have used descriptors such as ‘worry,’ ‘panic,’ or ‘fear’ to characterize anxious arousal, these terms are theorized to better represent state anxiety in response to an acutely threatening situation and are distinguished as another subtype of anxiety known as anxious apprehension (Sharp et al., 2015). Anxious arousal has been differentiated as a trait-based construct referring to the propensity to experience state fear more often and more easily with a temporally stable pattern of hypervigilance between acute fear responses (Sharp et al., 2015). The fear-anxiety-avoidance model has suggested explanations for individual differences in pain perception such that anxiety may be the mediating factor between fear and avoidance behavior that is often observed in chronic pain. However, the mechanism by which anxiety perpetuates chronic pain is not yet understood. Additionally, as depression frequently co-occurs with anxiety (Gorman, 1996), understanding the specific differences and synergistic

characteristics of co-occurring symptoms is imperative to understanding how psychological symptoms are associated with chronic pain (Sartorius et al., 1996; Zahn–Waxler et al., 2000). Applying dimensional approaches to psychological symptoms (i.e., affectivity, anxious arousal, anxious apprehension) that transcend traditional categorical classification (i.e., depression/anxiety; (Blanchard-Fields, 2005; Blanchette & Richards, 2010; Mitchell & Phillips, 2007; Yiend, 2004) may characterize dimensions of psychological function that are associated with chronic pain more accurately.

Not only are psychological symptoms associated with chronic pain, but those suffering from chronic pain have also endorsed difficulties with cognitive control, which may impair the ability to control thoughts, feelings, and emotions (Muraven et al., 1998; Richards & Gross, 2000). Cognitive control, or executive functions (EFs), are interrelated high-level cognitive domains including inhibition, shifting, and updating, that enable individuals to successfully navigate day-to-day experiences. EFs are implemented by prefrontal cortex (PFC) and represent a collection of interrelated domains that enable individuals to modify their thoughts and behaviors (Solberg et al., 2009). Individuals with chronic pain have reported significantly impaired overall EF compared to those not in chronic pain (Baker et al., 2016; Berryman et al., 2014). EF impairments, specifically impairment in inhibitory control function, have also been experimentally observed in individuals who experience chronic pain (Buhle & Wager, 2010; Glass et al., 2011). Inhibitory control is defined as the capacity to inhibit or regulate prepotent responses, or the ability to stop a planned or ongoing thought or action (Williams et al., 1999). Inhibitory control is required in many activities of daily living in which an individual needs to tune out stimuli that are irrelevant to a task or process at hand. Beyond cognitive capacities

needed for the regulation of emotional distress, the demands of chronic pain also include managing the pain itself through redirecting or shifting attention and inhibiting or suppressing ruminative thoughts about pain (Solberg et al., 2009). Because similar brain regions, particularly anterior cingulate cortex (ACC), have been implicated in pain, executive function, and emotional regulation, difficulties regulating emotional distress may be due to "cognitive overload," whereby the added challenges of chronic pain may fatigue or tax self-regulatory strength (Glass et al., 2011; Shackman et al., 2011; Solberg et al., 2009). Understanding how chronic pain is associated with psychological symptoms (particularly anxious arousal) and inhibitory control abilities may aid in finding effective treatments aimed at targeting these synergistic constructs.

The present set of studies assesses the relation between neural, psychological, and cognitive mechanisms that have been shown to influence the experience of chronic pain. Studies One and Two advanced our understanding regarding the interrelated nature of these mechanisms. Study Three focuses on targeting how these mechanisms may relate to each other in order to provide a framework that can be applied to individuals experiencing chronic pain.

Overview of Study One: Somatic Symptoms Distinctly Influence Visceral Nociception in Primary Dysmenorrhea

The first study, "Somatic Symptoms Distinctly Influence Visceral Nociceptive Mechanisms in Primary Dysmenorrhea," was expanded from my Master's thesis, which investigated the psychological and neural correlates associated with increased sensitivity to experimental pain in a sample of women with dysmenorrhea (i.e., painful menstrual cramps) compared to women without dysmenorrhea. This study focused on the role of psychological symptoms and neural mechanisms that impact visceral (i.e., relating to the internal organs of the

body) pain sensitivity to better elucidate the mechanisms that influence the experience of noxious stimuli. Women with dysmenorrhea (n = 106) and healthy controls (n = 24) participated in the study. Psychological symptoms (i.e., somatic symptoms, depression, anxiety) were assessed with questionnaire measures and visceral pain sensitivity was evaluated via experimentally induced bladder pain. Resting state PFC activity was studied with electroencephalography (EEG).

Overall, the findings from this study indicated that dysmenorrhea diagnosis and high levels of somatic symptoms, or somatic arousal, were predictive of increased pain perception via an experimental pain task. Depression and anxiety were associated with experimental bladder pain. Further, resting state brain activity in the alpha and beta oscillatory bands were associated with experimental pain and anxiety, respectively. Together, these findings provide evidence that for individuals experiencing chronic pain, somatic (i.e., anxious) arousal and related neural mechanisms (e.g., frontal alpha and beta activity) are associated with increased pain perception in the presence of noxious stimuli.

This study shows associations between frontal alpha and beta activity with experimental bladder pain, and abnormalities in PFC function have also been implicated in somatic symptoms (Hakala et al., 2002) as well as depression and anxiety disorders (Latthe et al., 2006). Frontal alpha activity has been associated with anticipatory processes, attentional demands, and cognitive control (Klimesch et al., 1998; Klimesch, 1999; Michels et al., 2008) which play a key role in pain perception and modulation (Moont et al., 2010; Quiton & Greenspan, 2007). Frontal beta power is also linked to chronic pain and it has been localized to cortical networks involved

in both cognitive control functions and pain response, such as anterior cingulate, prefrontal, and somatosensory cortices (Stern et al., 2006; Vanneste et al., 2017).

Since chronic pain and somatic symptoms are both associated with abnormalities in frontocingulate networks, and given the evidence supporting an association between affect and pain, understanding the multidimensionality of chronic pain-related symptoms is imperative in developing effective treatment. While Study One elucidated the importance of psychological symptoms, particularly somatic (i.e., anxious) arousal, and frontal resting-state activity on pain perception for individuals experiencing chronic pain, it did not account for the role of inhibitory control, which is implicated in pain perception and modulation per the fear-anxiety-avoidance model (Asmundson et al., 2004). Study Two was conducted to further understand the relations between chronic pain and psychological symptoms by studying their effects on inhibitory control.

Overview of Study Two: Chronic Pain and Inhibitory Control in Emerging Adults: The Role of Psychological Symptoms

In order to further investigate the relation between psychological symptoms (i.e., affectivity, anxious (i.e., somatic) arousal, and anxious apprehension/worry) and inhibitory control in the context of chronic pain, Study Two was implemented to identify the effects of chronic pain and the unique and synergistic effects of psychological symptomatology on inhibitory control. Specifically, to extend the findings from Study One, this study tested the hypothesis that emerging adults with chronic pain would have a symptom profile primarily characterized by anxious (i.e., somatic) arousal. Further, I hypothesized that anxious arousal would distinctly influence the relation between chronic pain and inhibitory control function. This

study used self-report measures of pain, anxious arousal, anxious apprehension (i.e., worry), affectivity, and inhibitory control abilities to study a sample of emerging adults (N = 2345, ages 18 - 27). This age range has been identified as a critical developmental window for executive function optimization, particularly inhibitory control, and those experiencing chronic pain may be especially vulnerable to inhibitory control deficits (Blyth et al., 2004; McCracken & Turk, 2002; Stinson et al., 2014).

Emerging adults with chronic pain represent a unique population to study inhibitory control as they face many challenging transitions with respect to developmental tasks (e.g., organizational skills, independence from caregivers), social development (e.g., sexuality, peer relationships), and healthcare systems (i.e., transition from pediatric to adult services). Thus, emerging adulthood may be a critical window of opportunity to better understand the relationship between chronic pain and inhibitory control via psychological symptoms (particularly anxious arousal), in order to optimize and solidify positive health behaviors to prevent long-term trajectories of severe pain and disability. Psychosocial interventions that specifically target psychological symptoms affecting inhibitory control may improve function in young adults with chronic pain, which could lead to more effective treatment of chronic pain symptoms into adulthood.

Overview of Study Three: Anxious Arousal and Anxious Apprehension are Distinctly Related to the Temporal Course of Response Inhibition

Building on findings from Study One and Study Two regarding the critical role of anxious arousal symptoms on increasing experimental pain discomfort and decreasing inhibitory control function in individuals with chronic pain, Study Three was conducted in an exploratory

manner to evaluate how anxious arousal and anxious apprehension differentially influence the implementation of the temporal course of neural activity during response inhibition. Previous research investigating these anxiety subtypes has been conducted mostly within the context of experimental tasks involving affective stimuli using functional Magnetic Resonance Imaging (fMRI), which has poor temporal resolution. Behavioral (i.e., response reaction time and task accuracy) and the temporal course of neural activity through event-related potentials (ERP) components (N200, N450, and conflict SP) were analyzed. This study used the Color-Word Stroop Test (Stroop, 1935), a neuropsychological test that assesses prepotent response inhibition. This study used a sample of undergraduate students (N = 92) to elucidate the distinct contributions of anxious arousal and anxious apprehension on inhibitory control. The study used both categorical and dimensional approaches to examining anxiety symptoms to probe the relations among distinct dimensions of anxiety along with the behavioral and neural correlates of response inhibition. Using an experimental psychophysiological measure of cognitive control to study the temporal course of response inhibition contributed to advancing the understanding of anxious arousal on inhibitory control. This contributed to the broader goal of informing research intervention efforts for individuals suffering from chronic pain.

CHAPTER TWO

SOMATIC SYMPTOMS DISTINCTLY INFLUENCE VISCERAL NOCICEPTION IN PRIMARY DYSMENORRHEA

Chronic pelvic pain conditions are difficult to treat but are often associated with potentially reversible risk factors such as anxiety, depression, somatic symptoms, and dysmenorrhea (Dawood, 1985; Westling et al., 2013; Zondervan et al., 2001). Dysmenorrhea (painful periods) affects between 25-75% of menstruating women. Dysmenorrhea is characterized by painful menstrual cramps and often occurs in the absence of pelvic pathology in young women (Dawood, 1985; Harel, 2006; Westling et al., 2013; Zondervan et al., 2001). Although prostaglandin mediated uterine contractions likely cause cramping pain, recent work has suggested that neurological changes also occur that could contribute to increased visceral (internal) organ sensitivity (Dawood, 2006; Hellman et al., 2017; Iacovides et al., 2015; Milsom et al., 1994; Vincent et al., 2011). Improving our understanding of the neural mechanisms that contribute to dysmenorrhea and co-occurring visceral pain could lead to the development of better preventative strategies. Visceral pain refers to pain originating from the internal organs, most notably the thoracic, abdominal, and pelvic structures (Gebhart, 2000). Visceral pain is often diffuse and difficult to localize, making it challenging to treat. Among other types of visceral pain, one of the most debilitating endured by women across the world is dysmenorrhea.

Susceptibility to pain sensitivity may be a primary risk factor for developing chronic pelvic pain for women with dysmenorrhea. Women with dysmenorrhea have reported higher sensitivity to experimental pain, even during phases of the menstrual cycle when pain is not experienced (Bajaj et al., 2002; Iacovides et al., 2015). Even in the absence of chronic pelvic pain, studies have shown that women with dysmenorrhea have increased visceral sensitivity during spontaneous bladder fullness (Tu et al., 2013).

Neurobiological Correlates of Visceral Pain

Activity in brain regions associated with increased risk for chronic visceral pain has not yet been characterized. However, it has been shown that women with primary dysmenorrhea have alterations in frontal connectivity (Gu et al., 2010), descending pain modulatory circuitry, and entorhinal cortex (Vincent et al., 2011). Previous studies have not specifically examined women while they are simultaneously experiencing menstrual cramps, although studies have examined what occurs during experimentally evoked visceral pain. Experiments such as colorectal distension evokes an increase in anterior cingulate cortex (ACC) activity, with increased ACC activity in participants with chronic visceral pain (Mayer et al., 2005; Silverman et al., 1997). Conversely, lesioning (or inhibiting) ACC can eliminate perceived aversiveness to pain (Gu et al., 2010; Qu et al., 2011). ACC is a critical junction box for affective and cognitive processing, and it has been implemented in affective, social, and physical pain perception (Eisenberg et al., 2010; Fuchs et al., 2014; Kondo et al., 2004; Mayer et al., 2005; Rotge et al., 2014; Silverman et al., 1997). Innovatively designed studies using hypnosis have shown that ACC is primarily involved in the affective processes related to pain response, while

somatosensory cortex is primarily associated with the bottom-up detection of noxious input (Rainville et al., 1997).

ACC is part of a network of frontal brain structures involved in top-down attentional control functions (Silton et al., 2010). Activation of the frontal-limbic network has been theorized to support effective coping strategies during pain anticipation (Berman et al., 2008). During the anticipation of a painful stimulus, patients with visceral pain are less likely to engage frontal mechanisms to down-regulate limbic activity (Berman et al., 2008). Related, frontal cortical hyperactivity has been observed in patients with chronic pain. Frontal hyperactivity was shown to modulate cognitive appraisal of pain-relevant emotional signals from the limbic system (Bernstein et al., 2002). Frontal activity during pain has been linked to affective processing, with bilateral dorsolateral prefrontal cortices (DLPFC) negatively correlated with intensity perception and unpleasantness (Lorenz et al., 2003; Lorenz & Casey, 2005). The inter-regional association between midbrain and medial thalamic activity was significantly reduced during increased left DLPFC activity (Lorenz & Casey, 2005), which suggests that pain affect may reduce connectivity within the midbrain-medial thalamic pathway. Right DLPFC was associated with a weakened relationship with the anterior insula with regard to pain intensity and affect, suggesting that the DLPFC actively regulates pain perception by modulating cortical pathways (Lorenz et al., 2003).

However, most visceral pain research has focused on gastrointestinal disorders, largely overlooking pain mechanisms in women with dysmenorrhea. Heightened pain sensitivity in women with dysmenorrhea might be associated with disinhibition of thalamo-orbitiofrontal-prefrontal networks that contribute to maintain thalamic sensitization (Tu et al., 2009, 2010).

This could induce compensatory inhibitory mechanisms in somatic sensorimotor regions, as well as dysfunctional DLPFC mechanisms which could be associated with increased negative affect (Berkley, 2013; Tu et al., 2010). One of the only other studies on dysmenorrhea and pain (but not during menses), showed increased limbic activity in women with dysmenorrhea that was linked to increased duration of dysmenorrhea (Vincent et al., 2011).

Frontal cortical brain activity is also often indexed using electroencephalography (EEG) methods to study theta (4 -7 Hz), alpha (8 – 13 Hz), and beta (14 – 30 Hz) oscillatory activity. Beta power has also been linked to chronic pain, such that beta power has been localized to cortical networks involved in both cognitive control functions and pain response, such as anterior cingulate, prefrontal, and somatosensory cortices in patients with fibromyalgia (Stern et al., 2006; Vanneste et al., 2017). Studies on pain have reported that both the beta and alpha frequencies are correlated with both transient and tonic noxious painful experiences and may reflect individuals' tonic pain responsiveness (Vanneste et al., 2017). Gonzalez-Roldan et al. (2016) found that patients with fibromyalgia had enhanced beta activity during resting state that was localized to right precentral gyrus, right middle frontal gyrus, superior frontal gyrus, midcingulate cortex, and right medial frontal gyrus. Further, studies have found a strong association with pgACC activity in the beta frequency range and pain vigilance and awareness (Vanneste et al., 2017). Enhanced frontal beta power may be a critical indicator of risk for chronic pain. Beta activity has been associated with the continuation of a cognitive set that overrides the effect of potentially unexpected external events (Engel & Fries, 2010). Beta is theorized to increase during maintenance of a cognitive set, particularly if a change or interruption is anticipated (Engel & Fries, 2010). Thus, enhanced beta activity is hypothesized to

be associated with perseveration, or a reduction in cognitive control and flexibility (Engel & Fries, 2010). Individuals who live with chronic pain may experience poor cognitive control (Shackman et al., 2011), which could be related to frequent and perseverative rumination about pain, or repetitively thinking about the causes and consequences of negative pain-related experiences (Sullivan et al., 2001). Persistent beta activity may result in abnormal persistence of a cognitive set and inhibit flexible cognitive control (Vanneste et al., 2017).

Reduced alpha power has been repeatedly associated with the administration of noxious stimuli (Nir et al., 2012) and has also shown to have a strong association between increased pregenual anterior cingulate cortex (pgACC) activity in the alpha frequency range and pain modulation (Vanneste et al., 2017). Increased dACC and sgACC in the alpha frequency range has been shown to be involved in the affective component of pain, which also correlates with depression (Vanneste et al., 2017). Within the alpha band, two to three sub-bands have been defined across various studies, each with its own pattern of desynchronization: lower alpha-1 (7-10 Hz) in considered to reflect anticipatory processes and attentional demands (Klimesch et al., 1998; Klimesch, 1999; Michels et al., 2008) which have a key role in pain perception and modulation (Moont et al., 2010; Quiton & Greenspan, 2007). Upper alpha (approximately 10-12 Hz) desynchronization represents task specific requirements (e.g., semantic memory functions, visuo-motor processes; Nir et al., 2012). Some studies have called for two lower alpha bands that reflect attentional demands such as alertness and expectancy (i.e., lower alpha-1: approximately 6-8 Hz and lower alpha-2: approximately 8-10 Hz), with lower alpha-1 representing phasic alertness and lower alpha-2 reflecting expectancy (Klimesch et al., 1998).

Less is known regarding theta frequency bands in relation to chronic pain, although increased dorsal ACC (dACC) and subgenual ACC (sgACC) as well as medial prefrontal cortex (mPFC) and DLPFC activity has been correlated with fatigue in individuals diagnosed with fibromyalgia and experiencing chronic pain (Vanneste et al., 2017).

Co-occurrence of Psychological Symptoms in Dysmenorrhea

Although multiple psychological symptoms may be associated with visceral pain, its relationship to somatic symptomatology has been an area of focus (Bettendorf et al., 2008; Latthe et al., 2006). Somatic symptoms have traditionally been defined as physical symptoms of unknown pathology and measured as individual symptoms or as symptom constellations (Dhossche et al., 2001; Steinhausen, 2006). However, this term has been highly criticized due to its diagnostic inflation, or the high probability of misdiagnosing a medical illness, and its inefficacy in capturing the true pain experience those afflicted are facing, despite unknown pathology (Katz et al., 2015). A more encompassing term, somatic arousal, is synonymous with "anxious arousal" and consists of an enduring pattern of hypervigilance, sympathetic nervous system hyperarousal to mild stressors, and state fear (Sharp et al., 2015). Anxious arousal has historically been conceptualized within the psychopathology literature as a distinct dimension or subtype of anxiety per the framework of the tripartite model of anxiety and depression (Clark & Watson, 1991). The tripartite model, which aimed to explain the co-occurrence between anxiety and depression syndromes, suggests that although anxiety and depression share a substantial component of general affective distress (i.e., negative affect), they can be differentiated on the basis of factors specific to each syndrome (Clark & Watson, 1991). Anxious arousal has been differentiated as a trait-based construct referring to the propensity to experience state fear more

often and more easily with a temporally stable pattern of hypervigilance between acute fear responses (Sharp et al., 2015). In women with dysmenorrhea, somatic symptoms are strongly associated with non-cyclic pelvic pain (i.e., pelvic pain not occurring in relation to menstrual cycle that lasts over six months), even after the variance associated with depression and anxiety is accounted for (Latthe et al., 2006). Somatic symptoms are often defined as a physical condition without an organic cause (Gureje et al., 1998). While some have argued that somatic symptoms are purely a cognitive or perceptual issue (Eriksen & Ursin, 2004; Ursin, 1997), other researchers have started to investigate the biological factors that may contribute to the development and maintenance of somatic symptoms (Rief & Barsky, 2005). Abnormalities in central nervous system structure and function have been implicated in somatic symptoms, including the spinal cord, brainstem, somatosensory cortices, parietal cortex, frontal cortex, and cingulate cortex (Hakala et al., 2002).

In addition to the frequent co-occurrence with somatic symptoms, dysmenorrhea has been shown co-occur with depression and anxiety symptoms, though the findings have been mixed (Latthe et al., 2006). Increased depression symptoms are strongly associated with menstrual pain, and women with dysmenorrhea may have a more negative mood state during menstruation (Alonso & Coe, 2001; Dorn et al., 2009). Adolescent girls experiencing dysmenorrhea may be at an increased risk for depression, showing an early predisposition to mood disorders with the potential for long-term adverse effects (Balık et al., 2014). An extensive body of research indicates that depression is associated with abnormal patterns of cortical and subcortical brain function in regional brain networks that overlap with those implicated in pain perception (Davidson, 1998; Davidson & Irwin, 1999; Heller, 1990; Heller et al., 1997, 1998; Silton et al.,

2011). Thus, it remains plausible that the neurobiological correlates of depression and anxiety symptoms predispose women to a heightened sensitivity to visceral pain perception and/or dysmenorrhea. Given the study's primary focus on understanding the role of somatic symptoms and the substantial overlap in components of anxiety and depression as categorical constructs per the tripartite model, depression and anxiety will be included as covariates in order to account for the shared constructs. This will allow our study to focus on the role of somatic arousal symptoms on pain perception.

Present Study

Psychological symptoms and functional abnormalities in prefrontal and medial frontal brain activity likely contribute to heightened visceral pain in women with dysmenorrhea. Developing a better understanding regarding how psychological and physiological mechanisms confer risk for increased pain perception in women with dysmenorrhea will contribute to informing the development of effective intervention strategies. In the present study, the following hypotheses were evaluated in a sample of women with and without a diagnosis of dysmenorrhea: 1) replicating previous research, women with dysmenorrhea were expected to have increased somatic symptoms) and report higher levels of pain during experimental bladder distension, 2) women with greater visceral pain were predicted to have increased resting state frontal activity, particularly beta activity, and 3) increased frontal activity, particularly beta, during resting state was expected to be related to increased levels of experimentally-induced visceral pain in women with dysmenorrhea when variance associated with co-occurring psychological symptoms was accounted for.

Method

Female participants were recruited by flyers posted on college campuses, in the community, Craigslist advertisements, and by referral from local gynecology clinics. Potential participants were instructed to call a study hotline number and complete a phone screen to determine eligibility. Participants were not included in this specific study focusing on dysmenorrhea if they met criteria for other pain conditions. If eligible, participants were scheduled for an initial screening visit, during which self-report questionnaires on gynecological history, medical history, and mental health evaluations were administered. The screening visit was conducted by a board-certified Ob/Gyn with pain fellowship training to verify the likelihood that participants had primary dysmenorrhea. When necessary, ultrasound evaluation was performed for further confirmation. Eligible participants completed daily pain diaries to confirm menstrual pain and they were asked to abstain from using birth control pills for the duration of the study. Participants were scheduled for a mid-luteal phase assessment session, approximately 17-25 days after commencement of menses. A urinary luteinizing hormone testing kit was used to confirm entrance into the luteal phase. Participants were asked to avoid taking short-acting, over-the-counter analgesics (ibuprofen) and caffeine for at least six hours prior to the assessment session. Data were collected by trained research assistants. At the mid-luteal phase assessment visit, participants filled out additional questionnaires. At the end of the visit, participants performed resting state EEG, followed by brief tasks during EEG data collection. Only the resting state EEG paradigm was analyzed in the present study. The study was approved by the NorthShore University HealthSystem Institutional Review Board, and informed consent was obtained from each participant prior to performing any tests or filling out any questionnaires.

Participants

The present study included *N* = 130 women (age range:18 - 43, *M* = 25 years) with dysmenorrhea *(n* = 106) and healthy controls (*n* = 24). The following criteria were used to identify women with moderate dysmenorrhea: a) average menstrual pain ≥ 50/100 (0 = no pain and 100 = the worst imaginable pain) during menses or withdrawal uterine bleeding from cyclic oral contraceptives (OCs) without painkillers, b) menstrual pain in the region between the umbilicus and the perineum, above the level of the inguinal ligament and c) an indication the participant has attempted to resolve pain by medical means (including NSAIDs and/or OCs). In order to be considered a control, women could not be diagnosed with endometriosis or chronic pelvic pain.

Measures

Demographics

Participants responded to questions about demographic information, including their age and race/ethnicity (see Table 1).

Table 1. Demographic Information by Group (N = 130).

	Diagnostic Group					
	Control (n = 24)		Dysmenorrhea (n = 106)			
Variable	*M*	*SD*	*M*	*SD*	*F*	*p*
Age, y	23.04	6.33	24.38	6.18	.91	.34
Variable	*n*	%	*n*	%		
Race						
Caucasian	14	58.3	64	60.4		
Asian	8	33.3	21	19.8		
African American	0	0	13	12.3		
American Indian/Alaska Native	1	4.2	0	0		
Multiple Races Reported	0	0	8	7.5		
Declined to Report	1	4.2	0	0		

Note: * p <.05

Somatic Symptoms

Participants completed the 6-item somatization subtest from the 18-item Brief Symptoms Inventory (BSI-18) to evaluate somatic symptoms (Derogatis & Melisaratos, 1983). The BSI is a widely used self-report questionnaire that consists of eighteen descriptions of physical and emotional complaints. Respondents are asked to indicate on a 5-point Likert scale from 0 (not at all) through 4 (very much) to what extent they are troubled by the complaints (Kellett et al., 2003).

Depression

Participants completed the Patient Reported Outcomes Measurement Information System short form (PROMIS) to evaluate depression symptoms (α = .98; Bartlett et al., 2015). The PROMIS depression short-form scale consists of 8 items within a seven-day time frame and a five-point Likert scale (Cella et al., 2010; Pilkonis et al., 2011). Item content focuses on

emotional and cognitive manifestations of depression rather than somatic symptoms (e.g., fatigue, sleep, appetite). It was developed for use in both clinical and research settings.

Anxiety

Participants also completed the Patient Reported Outcomes Measurement Information System short form (PROMIS) to evaluate anxiety symptoms (α = .97; Bartlett et al., 2015). The PROMIS depression short-form scale consists of 8 items within a seven-day time frame and a five-point Likert scale (Cella et al., 2010; Pilkonis et al., 2011). Item content focuses on fear (e.g., worry, panic), dread, hyperarousal (e.g., tension, nervousness, restlessness), and somatic (i.e., arousal) symptoms (e.g., cardiovascular symptoms, dizziness). It was developed for use in both clinical and research settings.

Procedures

Experimentally Induced Visceral Pain

Participant underwent non-invasive experimental bladder distension (Tu et al., 2012). The bladder test mimics clinical retrograde cystometry, a diagnostic test used to evaluate bladder function, starting with an emptied bladder. After oral ingestion of 20 oz of water, participants were instructed to report when they reached three standard levels of bladder urgency: first sensation, first urge to void, and maximal capacity (Abrams et al., 2002). At baseline and each of these time points, three-dimensional sonographic measurements of the bladder were obtained (GE Voluson 750, Wauwatosa, WI), and participants rated their bladder pain and urgency on a 10 cm Visual Analog Scale (VAS) on a tablet computer. Experimental bladder pain assessment was capped at two hours, even if participants did not reach maximal capacity. Experimental bladder pain scores were determined by participants' ratings at first urge to void.

Resting State Measurement

Participants were instructed to relax for eight one-minute resting baseline periods during which EEG data were recorded. Prior to the start of each period, participants saw directions (counterbalanced “eyes open” or “eyes closed”) on a computer screen. During eyes-open recording, participants were instructed to focus their eyes on a small cross in the center of the screen. After each eyes-closed recording, via intercom the experimenter instructed participants to open their eyes. Per the recommendations of Tomarken et al., (1992) four eyes-open (O) and four eyes-closed (C) baseline periods, ordered COOCOCCO and OCCOCOOC, were randomly assigned to the participants.

EEG Data Recording and Collection

Participants were seated in a comfortable chair while EEG data were recorded with active electrodes using a standard 32-channel cap. Electrode placement was based on the International 10-20 System (Jasper, 1958), with the left mastoid used as the online reference. Eye and facial movements were also recorded with electrodes placed about the right eye, below the left eye, and in the middle of the brow in order to account for ocular artifact. Placement of the cap and electrodes was implemented by a trained research assistant, and impedances were maintained below 25 kOhms. Data were recorded at a sampling rate of 500 Hz. EEG recordings were amplified by the actiChamp amplifier (Brainvision, NC) using a 24-bit A/D converter. Physiological data were collected by Pycorder software (Brainvision, NC). Stimulus presentation and onset of physiological data collection were controlled by E-prime (Psychology Software Tools, PA) run on a PC.

EEG Data Reduction

EEG data were processed in Brain Electrical Source Analysis software (BESA Research 7.0). Visual inspection of the data was implemented to remove ocular and muscular artifacts. In order to reduce volume conduction contributions, EEG data were transformed to a current source density (CSD) Laplacian 27-channel virtual montage. CSD is a spatial high-pass filter that is relatively insensitive to deeper and more distributed sources (Allen & Reznik, 2015; Stewart et al., 2014). Next, a fast Fourier transform (FFT) was applied to 1.024 second epochs in order to obtain the power spectrum, producing spectral output in 0.977 Hz bins and then averaging all the power spectra across epochs within each sixty-second baseline. Spectra were averaged across eyes-open and eyes-closed baselines in order to allow for stable estimates of oscillatory activity (Henriques & Davidson, 1991). Alpha (8 – 13 Hz), Theta (4 – 7 Hz), and Beta (14 – 30 Hz) power was extracted from the power spectrum in 0.977-Hz bins. Lower-1 alpha (6 – 7.5 Hz), lower-2 alpha (8 – 9.5 Hz), and upper alpha (10 – 13 Hz) were also extracted. Electrode Fz was selected for analyses as a proxy for frontal activity.

Results

Sample Means and Zero-Order Pearson Correlations

One-way Analysis of Variance Analyses (ANOVA) were conducted to characterize differences between healthy controls and women with dysmenorrhea on study variables (Table 2). The groups were similar in ages, F (1, 128) = .91, p = .34. Compared to healthy controls, women with dysmenorrhea reported more sensitivity to visceral pain, and somatic symptoms. The two groups had comparable levels of depression and anxiety symptoms and were comparable across frontal resting state activity at different oscillatory bands (e.g., beta, theta, alpha, and alpha bands).

Table 2. Group Characteristics of Experimental Bladder Pain, Menstrual Pain, Psychological Symptoms, and Frontal Resting State Activity.

	Diagnostic Group					
	Control (n = 24)		Dysmenorrhea (n = 106)			
	M	*SD*	*M*	*SD*	*F* (1,128)	*p*
Experimental Bladder Pain	7.31	8.74	16.92	12.22	7.49	<.001*
Menstrual Pain	18.21	15.56	72.87	14.36	275.07	<.001*
Depression	14.25	5.19	14.49	6.43	.03	.86
Anxiety	14.54	5.16	15.68	5.96	.75	.39
Somatic Symptoms	1.50	1.50	3.20	3.10	6.82	.01*
Fz Beta Power	-6.48	.41	-6.51	.56	.06	.81
Fz Theta Power	-4.64	.49	-4.69	.57	.18	.68
Fz Alpha Power	-5.28	.58	-5.35	.70	.25	.62
Fz Lower-1 Alpha	-4.83	.62	-4.86	.69	.03	.87
Fz Lower-2 Alpha	-4.98	.65	-5.07	.79	.25	.62
Fz Upper Alpha	-5.53	.61	-5.59	.69	.16	.70

Note: * p <.05

Zero-order Pearson correlation coefficients among variables used in regression analyses are included in Table 3. Many of the variables were significantly related. As predicted, group was significantly related to experimental bladder pain (r = .24, p = .01), menstrual pain (r = .83, p = .00), and somatic symptoms (r = .23, p = .01). Bladder pain was significantly related to experimental menstrual pain *(*r = .30, p = .00), somatic symptoms (r = .23, p = .01) and frontal alpha power *(*r = .19, p = .03). Menstrual pain was significantly related to somatic symptoms *(*r = .22, p = .01). Depression was significantly correlated with anxiety symptoms *(*r = .80, p = .00)

and somatic symptoms *(r* = .43, *p* = .00). Anxiety was significantly correlated with somatic symptoms *(r* = 0.47, *p* < 0.01) and frontal beta power *(r* = .20, *p* = .02).

Table 3. Correlations Among Group, Pain, Psychological Symptoms, and Brain Activity (*N* = 130).

	1.	2.	3.	4.	5.	6.
1. Group						
2. Bladder Pain	.24*					
3. Menstrual Pain	.83*	.30*				
4. Depression	.02	.05	.08			
5. Anxiety	.08	.02	.11	.80*		
6. Somatic Symptoms	.23*	.23*	.22*	.43*	.47*	
7. Fz Beta Power	-.02	-.01	.00	.17	.20*	.06
8. Fz Theta Power	-.04	.01	-.01	.06	.11	.05
9. Fz Alpha Power	-.04	.19*	.04	.14	.14	.08
10. Fz Lower1 Alpha	-.02	.12	.05	.06	.13	.06
11. Fz Lower2 Alpha	-.04	.20*	.05	.11	.12	.09
12. Fz Upper Alpha	-.04	.16	-.02	.14	.14	.06

*Note: *p* <.05

Linear Regression

Three hierarchical linear regressions were run to test the hypothesis that somatic symptoms and frontal resting state activity would be primary predictors of experimental bladder pain. Group was dummy coded (0 = healthy controls, 1 = dysmenorrhea). The regressions were each run in five steps. The first step included group, and the second step included other covariates (depression and anxiety). Somatic symptoms were entered in the third step, and

frontal resting activity (i.e., beta, theta, alpha) was entered in the fourth step. The fifth step included the two-way interaction between somatic symptoms x frontal resting activity (Table 4).

Table 4. Hierarchical Linear Regressions of Psychological Symptoms and Brain Activity (Beta, Theta, Alpha) Predicting Experimental Bladder Pain.

A. Frontal Beta Power

Predictor	***B***	***SE***	***t (p)***	***95% CI***	***R^2***	**Tolerance**	**VIF**
Step 1					R^2 = .04, F(1,128) = 4.84 ***p* = .03***		
Group	8.05	3.66	2.20 (***p* = .03***)	[.81, 15.28]		1.00	1.00
Step 2					ΔR^2 = .01, $\Delta F(2, 126)$ = .57 *p* = 0.57		
Anxiety	-.40	.41	-.96 (*p* = .34)	[-1.21, .42]		.35	2.82
Depression	.41	.39	1.05 (*p* = .30)	[-.36, 1.17]		.36	2.81
Step 3					ΔR^2 = 0.06, $\Delta F(1,125)$ = 8.03 ***p* = 0.01***		
Somatic Symptoms	1.56	.55	2.83 (***p* = .01***)	[.47, 2.66]		.73	1.36
Step 4					ΔR^2 = 0.00, $\Delta F(1,124)$ = 0.00 *p* = 0.81		
Frontal Beta Power	-.62	2.57	-.24 (*p* = .81)	[-5.71, 4.47]		.96	1.05

Step 5					$\Delta R^2 = 0.00$, $\Delta F(1,123) = 3.92$ $p = 0.10$		
Somatic Symptoms x Frontal Beta Power	-.01	.88	-.02 (p = .99)	[-1.76, 1.76]		.01	143.83

B. Frontal Theta Power

Predictor	***B***	***SE***	***t (p)***	***95% CI***	***R^2***	**Tolerance**	**VIF**
Step 1					$R^2 = .04$, $F(1,128) = 4.84$ ***p* = .03***		
Group	8.05	3.66	2.20 (***p* = .03***)	[.81,15.28]		1.00	1.00
Step 2					$\Delta R^2 = .01$, $\Delta F(2, 126) = .57$ $p = .57$		
Anxiety	-.40	.41	-.96 (p = .34)	[-1.21, .42]		.35	2.82
Depression	.41	.39	1.05 (p = .30)	[-.36, 1.17]		.36	2.81
Step 3					$\Delta R^2 = .06$, $\Delta F(1,125) = 8.03$ ***p* = .001***		
Somatic Symptoms	1.56	.55	2.83 (***p* = .01***)	[.47, 2.66]		.73	1.36
Step 4					$\Delta R^2 = .00$, $\Delta F(1,124) = .01$ $p = .91$		

Frontal Theta Power	.28	2.55	.11 (p = .91)	[-4.77, 5.33]		.98	1.02
Step 5					ΔR^2 = .01, $\Delta F(1,123)$ = .72 p = .40		
Somatic Symptoms x Frontal Theta Power	-.74	.87	-.85 (p = .40)	[-2.46, .99]		.01	74.75

C. Frontal Alpha Power

Predictor	***B***	***SE***	***t (p)***	***95% CI***	***R^2***	**Tolerance**	**VIF**
Step 1					R^2 = .04, $F(1,128)$ = 4.84 p = .03*		
Group	8.05	3.66	2.20 (***p* = .03***)	[0.81, 15.28]		1.00	1.00
Step 2					ΔR^2 = .01, $\Delta F(2, 126)$ = .57 p = .57		
Anxiety	-.40	.41	-.96 (p = .34)	[-1.21, .42]		.35	2.82
Depression	.41	.39	1.05 (p = .30)	[-.36, 1.17]		.36	2.81
Step 3					ΔR^2 = .06, $\Delta F(1,125)$ = 8.03 ***p* = 0.001***		
Somatic Symptoms	1.56	.55	2.83 (***p* = .01*)**	[.47, 2.66]		.73	1.36

Step 4					$\Delta R^2 = .02$, $\Delta F(1,124) = 2.95$ $p = .09$		
Frontal Alpha Power	3.54	2.06	1.72 (p = .09)	[-.54, 7.62]		.98	1.03
Step 5					$\Delta R^2 = .00$, $\Delta F(1,123) = .34$ $p = .56$		
Somatic Symptoms x Frontal Alpha Power	.39	.67	.58 (p = .56)	[-.94, 1.72]		.02	57.93

D. Frontal Alpha Power Bands

Predictor	***B***	***SE***	***t (p)***	***95% CI***	***R^2***	**Tolerance**	**VIF**
Step 1					$R^2 = .04$, $F(1,128) = 4.84$ ***p* = .03***		
Group	8.05	3.66	2.20 (***p* = .03***)	[.81,15.28]		1.00	1.00
Step 2					$\Delta R^2 = .01$, $\Delta F(2, 126) = .57$ $p = .57$		
Anxiety	-.40	.41	-.96 (p = .34)	[-1.21, .42]		.35	2.82
Depression	.41	.39	1.05 (p = .30)	[-.36, 1.17]		.36	2.81
Step 3					$\Delta R^2 = .06$, $\Delta F(1,125) = 8.03$ ***p* = .001***		

Somatic Symptoms	1.56	.55	2.83 (***p* = .01***)	[.47, 2.66]		.73	1.36
Step 4					$\Delta R^2 = .03$, $\Delta F(3,122) = 1.30$ $p = .28$		
Frontal Lower Alpha1	-.56	3.13	-.18 (*p* = .86)	[-6.77, 5.64]		.43	2.35
Frontal Lower Alpha2	4.71	3.5	1.35 (*p* = .18)	[-2.22, 11.64]		.27	3.69
Frontal Upper Alpha	-1.18	3.14	-.38 (*p* = .71)	[-7.40, 5.04]		.43	2.32

Note: *p <.05

As hypothesized, group was a significant predictor of experimental bladder pain, (F (1, 128) = 4.84, p = .03), such that individuals with dysmenorrhea were associated with increased experimental bladder pain. Further, increased somatic symptoms, (ΔF (1, 125) = 8.03, p = .01) were a significant predictor of increased experimental bladder pain. Anxiety and depression were not significant predictors of experimental bladder pain. Further, frontal resting state activity (e.g., beta, theta, and alpha power) and the interaction between resting state activity and somatic symptoms were not significant predictors of bladder pain. A fourth hierarchical linear regression was run to test the hypothesis that somatic symptoms and frontal alpha power separated into three bands would be primary predictors of bladder pain; however, the separate alpha power bands were not significant predictors of experimental bladder pain.

Discussion

The present study sought to replicate previous findings that women with greater menstrual pain would have increased somatic symptoms and report higher levels of experimental

bladder pain and also aimed to characterize the neural correlates associated with psychological symptoms and experimental bladder pain. As expected, women with dysmenorrhea in the study sample exhibited increased somatic symptoms and experimental menstrual pain compared to women without dysmenorrhea. However, women with dysmenorrhea were not found to have increased frontal activity compared to women without dysmenorrhea. Further, dysmenorrhea diagnosis and increased somatic symptoms predicted accounted for the variance associated with experimental bladder pain above and beyond other psychological symptoms (e.g., depression or anxiety) and frontal resting state activity (e.g., beta, theta, or alpha power bands). Of note, experimental bladder pain was correlated with frontal alpha power, specifically the second lower alpha band (8 – 9.5 Hz) and anxiety was associated with frontal beta power irrespective of group membership.

Somatic symptoms and functional abnormalities in prefrontal and medial frontal brain activity were predicted to contribute to heightened visceral pain in women with dysmenorrhea. While frontal resting state activity were not significant predictors of experimental bladder pain, experimental bladder pain was positively correlated with frontal alpha power, particularly in the second lower alpha band. Developing a better understanding regarding how these mechanisms confer risk for the development of chronic pelvic pain in these women is imperative in order to contribute to informing effective intervention strategies. Our results provide evidence that dysmenorrhea and psychological symptoms are related to increased levels of experimental bladder pain. Although depression and anxiety were both associated with somatic symptoms in the present study, neither emerged as a significant factor related to visceral sensitivity.

Results from hierarchical regressions showed that somatic symptoms were significant predictors of experimental bladder pain and may be a primary risk factor for the transition to chronic visceral pain. Somatic symptoms should be considered a key target for psychological prevention and intervention strategies. While the women in this study were not diagnosed with chronic pelvic pain, their reports of experimental pain sensitivity were comparable to participants with chronic pelvic pain in a recent study (Tu et al., 2017). This prior study also confirmed an important role for somatic symptoms. Thus, the women in the present study may be at risk for developing the complete profile of symptoms associated with chronic pelvic pain.

Limitations and Future Considerations

Although the present study advanced the characterization of the somatic and physiological factors associated with visceral pain in women with dysmenorrhea, some limitations exist. The cross-sectional study design limits our ability to fully understand the developmental trajectory of pain outcomes in women with dysmenorrhea. In particular, the role of psychological symptoms (e.g., depression, anxiety) with regard to pain outcomes may be better understood with longitudinal data, as noted above. Other research has illustrated that anxiety and depression scores were negatively correlated with beta power, such that enhanced resting state beta activity was associated with decreased cognitive control, but also less depressive symptoms in individuals with chronic pain (González-Roldán et al., 2016). While at first blush this may appear counterintuitive as depression symptoms typically accompany attentional control deficits (Silton et al., 2011), these findings may be due to a premature assumption of the emergence of depressive symptoms since clinically significant depressive symptoms may not have yet emerged in the sample. Further, the present study sample consists

primarily of young adult women, and developmental data spanning a broad age range would enhance the generalizability of the results from the present study.

Conclusion

The present study illustrated a relationship between somatic symptoms and visceral pain. Thus, treatments that reduce somatic symptoms may help ameliorate pain symptoms, such as mindfulness (Berkovich-Ohana et al., 2012; Travis et al., 2010), transcendental meditation (Jacobs et al., 1996) and the relaxation-response (Suskind et al., 2014). Notably, mindfulness exercises have been effective in not only reducing chronic pain, but also decreasing the likelihood of prolonged somatic symptoms (Kabat-Zinn, 1982). Mindfulness may aid in filtering inputs to the primary somatosensory cortex, which could broaden attentional resources that may be narrowly focused on pain and shift that concentration on awareness of the body in a nonjudgmental way (Kerr et al., 2013). Since chronic pain, depression, and somatic symptoms are associated with abnormalities in frontocingulate networks, and given the evidence supporting an association between affect and pain, understanding the multidimensionality of dysmenorrhea-related symptoms from an interdisciplinary perspective is imperative in developing effective treatments. Continuing to work toward identifying potentially modifiable psychological and physiological risk factors is critical to ameliorating the development of chronic pelvic pain in the context of dysmenorrhea.

CHAPTER THREE

CHRONIC PAIN AND INHIBITORY CONTROL IN EMERGING ADULTS: THE ROLE OF PSYCHOLOGICAL SYMPTOMS

Emerging adulthood (i.e., individuals between the ages of 18-29) is recognized as a unique period of life characterized by complex developmental transitions (e.g., independence from caregivers, increased responsibility, complex peer relationships; Arnett, 2014; Stinson et al., 2014) and distinct neurocognitive and emotional vulnerabilities (Taber-Thomas & Pérez-Edgar, 2014). Emerging adulthood (also referred to as "young adulthood"), is also a critical developmental window to optimize self-management strategies (e.g., self-monitoring, evaluation, reinforcement) to prevent long-term trajectories of severe pain and disability (Blyth et al., 2004; McCracken & Turk, 2002; Stinson et al., 2014).

Individuals with chronic pain often endorse difficulties with cognitive control that may impede their ability to effectively engage in pain self-management strategies and achieve salient educational and vocational milestones (Baker et al., 2016; Berryman et al., 2014). From the perspective of brain development, emerging adulthood is a vulnerable period of ongoing maturation of brain networks that implement executive functions (EFs; Taber-Thomas & Pérez-Edgar, 2014). EFs are interrelated high-level cognitive domains that enable individuals to navigate day-to-day experiences (Solberg et al., 2009) successfully, and definitions of EFs generally include aspects of cognitive function such as inhibition, shifting, and updating (Miyake et al., 2000). These general aspects of functioning are essential for the development of self-

regulatory processes such as decision making, goal-directed problem solving, selective attention, and voluntary response inhibition (Blakemore & Choudhury, 2006; Cartwright, 2012; Liew, \2012; Zelazo, 2015). In terms of self-reported EF abilities, adolescents and middle-aged adults with chronic pain have endorsed significantly impaired overall EF in daily life contexts compared to those without chronic pain or standardized norms (Baker et al., 2016; Berryman et al., 2014; Weiss et al., 2018). However, EF and chronic pain has not been adequately studied in emerging adults. EF deficits are of particular importance in the context of emerging adults as this group navigates important transitions (e.g., independent living/self-sufficiency and educational/vocational choices), and may partially explain why young adults with chronic pain evidence lower educational and vocational functioning compared to those without chronic pain (Kashikar-Zuck et al., 2014; Pinquart, 2014).

Among EF domains, inhibitory control may be specifically associated with individuals experiencing chronic pain. Past studies have found small to moderate impairment in task-related response inhibition in middle-aged adults experiencing chronic pain (Berryman et al., 2014; Landrø et al., 2013). Self-reported inhibitory control has also shown small to moderate impairments (Landrø et al., 2013). Overall, however, findings on inhibition have been mixed, and task-based and self-reported inhibitory control has not been studied exclusively in emerging adults. Inhibitory control is defined as the capacity to inhibit or regulate prepotent responses, or the ability to stop a planned or ongoing thought or action (Williams et al., 1999). Inhibitory control is required in many activities of daily living in which an individual needs to tune out stimuli that are irrelevant to a task or process at hand or to the mind’s current state.

Brain regions that are commonly implicated in pain (e.g., prefrontal cortex (PFC), anterior cingulate cortex (ACC), and insula; Banich et al., 2020; Shackman et al., 2011) also overlap with brain regions that support EFs and emotional regulation (Bushnell et al., 2013; Shackman et al., 2011). Moreover, depression and anxiety peak during the emerging adulthood period and are commonly co-morbid with chronic pain (Nicholson & Verma, 2004; Richardson et al., 2012). Thus, it is possible that common psychological symptoms may be associated with cognitive deficits including inhibitory control impairment in emerging adults with chronic pain. More broadly, the cognitive demands of chronic pain often involve managing the response to pain through redirecting attention and inhibiting or suppressing ruminative thoughts about pain (Solberg et al., 2009). Thus, emerging adults with chronic pain may be particularly vulnerable to perceiving deficits in inhibitory control function due to the underlying influence (i.e., mediating effects) of heightened psychological symptomatology. By characterizing psychological factors that maintain inhibitory deficits, future studies can aim to develop more effective treatments for emerging adults with chronic pain.

The present cross-sectional study had two aims. First, we report on several pain characteristics in an emerging adult population (current sample's age ranges from 18-27), for which there is little pain research. Specifically, we report on pain location, frequency, intensity, duration, bother, and activity limitations due to pain. Next, we evaluated the mediating influence of psychological symptoms on the relation between chronic pain status and perceived inhibitory control function (see Figure 1). Participants were coded as having chronic pain if endorsing pain lasting 3 months or longer on this item, which is in line with the International Association for the Study of Pain (IASP) definition of chronic pain (Merskey & Bogduk, 1994). For this aim, we

utilized a parallel mediation model with cross-sectional data. Given the overlap of pathways previously discussed, we hypothesized that psychological distress would influence the relation between chronic pain status and self-reported inhibitory control. Of note, age, gender, ethnicity, and race all have been linked to differences in pain, psychological symptoms, and inhibitory control (Affleck et al., 1999; Campbell et al., 2008; Riley et al., 2002; Tsang et al., 2008; Williams et al., 1999; Yuan et al., 2008). In order to account for differences associated with these constructs, which are not associated with the primary aims of this study, they will be included as covariates in the model.

Method

Participants

Emerging adults (N = 2481) enrolled in an urban Midwestern university were recruited from introductory psychology courses between the years of 2014 - 2019. Participants completed online questionnaires in a single session measuring psychological symptoms (e.g., depression, anxiety, and pain symptoms). These surveys were part of a larger battery of self-report questionnaires. Casewise deletion was used to omit participants (n = 31) who did not fit within the emerging adult age range (ages 18-29) as well as participants (n = 105) who had missing responses to any questionnaires used in the present study. The final sample used for analyses included 2345 participants between the ages of 18 – 27 years. The study was approved by the University's Institutional Review Board, and informed consent was provided to all participants before beginning the survey.

Measures

Demographics

Participants responded to questions about demographic information, including questions about age, gender, race, and ethnicity.

Chronic Pain Status

Participants reported on overall *pain duration* (e.g., how long they had aches or pains; 0 = "Not at all," 1 = "less than one month," 2 = "1 to 2 months" or 3 = "3 months or longer"). Participants were coded as having chronic pain if endorsing pain lasting 3 months or longer on this item, which is in line with the IASP definition of chronic pain (Merskey & Bogduk, 1994). Those without chronic pain endorsed a 0, 1 or 2 on a 4-point frequency scale.

Pain Characteristics

Participants reported on additional pain characteristics including *location, frequency, intensity, daily duration, bother/upset* due to pain, and *activity limitations* due to pain. Individuals also indicated the location(s) they experienced pain (i.e., "Where on your body do you have aches or pain?"). Emerging adults further reported on *pain frequency* (i.e., "In the past month, how often have you had aches or pains?") using a 7-point Likert rating scale (0 = "not at all" to 6 = "daily"; Toliver-Sokol et al., 2011). Participants also rated their usual *pain intensity* (i.e., "When you have aches or pains, how much hurt do you usually have?") using an 11-point Numerical Rating Scale (0 = "no pain" to 10 = "worst pain possible"), which has demonstrated adequate reliability and validity (Dworkin et al., 2005; von Baeyer, 2009), *daily pain duration* (i.e., "How long do your aches usually last?") was rated on a 4-point scale (1 = "less than an hour" to 4 = "all day"; Toliver-Sokol et al., 2011). Participants also rated how *bothersome* they

found their pain (i.e., “How much do aches or pains bother or upset you?) was rated on a 5-point scale (1 = “not at all” to 5 = “very much”; Holley et al., 2017; McGrath, 1993; von Baeyer et al., 2009). Finally, participants provided information on *activity limitations* (i.e., “How much do aches or pains limit or stop you from doing usual activities?”) using an 11-point Numerical Rating Scale (0 = “does not limit activity” to 10 = “limits all activity”; Dworkin et al., 2005; von Baeyer, 2009).

Affectivity and Anxious Arousal Symptoms

The Mood and Anxiety Symptoms Questionnaire (MASQ) was used to assess depression and anxiety components of the tripartite model (Watson, Clark, et al., 1995; Watson, Weber, et al., 1995). The following 3 subscales were used in this study: 8-item Negative Affectivity (NA) scale, 14-item Positive Affectivity (PA) scale, and 17-item Anxious Arousal (AA) scale. Higher scores reflect greater levels of symptomatology. McDonald’s Omega was obtained for each subscale in a study utilizing a sample of patients with chronic pain that supported the utility of the MASQ with this population, which are consistent with the original validation sample showing moderate to high internal consistency across all three subscales (Geisser et al., 2006). The present study replicated these findings (NA, ω = .81; PA, ω = .93; and AA, ω = .88).

Worry

The Penn-State Worry Questionnaire was used (Meyer et al., 1990). The PSWQ has 16 items and each item is rated on a scale from 1 (“not at all typical of me”) to 5 (“very typical of me”). Eleven items are worded in the direction of pathological anxious apprehension (i.e., worry), with higher numbers indicating more anxious apprehension (e.g., “Once I start worrying, I cannot stop”), while the remaining five items are worded to indicate that anxious apprehension

is not a problem, with higher numbers indicating less anxious apprehension (e.g., “I never worry about anything”). The total score was calculated by summing the first 11 items and the reverse-scores of the latter 5 items, with higher PSWQ scores reflecting greater levels of pathological anxious apprehension. Consistent with previous research (Henning et al., 2007), the present study found good internal consistency (ω = .94).

Inhibitory Control

The eight-item inhibition clinical scale subtest from the Behavior Rating Inventory of Executive Function – Self Report (BRIEF-A) was used to assess inhibitory control function and has been used to assess executive function in both children (through caregiver report) and adults with chronic pain (Baker et al., 2016; Gioia et al., 2000; Hocking et al., 2011). Participants indicate the extent to which a variety of behaviors (e.g., “I have trouble concentrating on tasks”) have been a problem (N = Never, S = Sometimes, O = Often). Internal consistency has been shown to be in the moderate range (Gioia et al., 2000) and the present study replicates these findings (ω = .77).

Data Analyses

All analyses were conducted with SPSS version 26.0. Descriptive statistics were calculated for continuous (e.g., pain intensity) and categorical (e.g., pain location) pain characteristics. Associations among the four pain characteristics (i.e., chronicity, one-month frequency, intensity, and duration), psychological symptoms (i.e., affectivity and anxiety), and inhibitory control were examined using Pearson two-tailed correlations. Finally, a parallel mediation analysis was conducted using the PROCESS v3.4 macro in SPSS (Hayes, 2017) to evaluate which psychological symptoms (positive affectivity, negative affectivity, anxious

arousal, and anxious apprehension (i.e., worry)) distinctly mediate the relationship between chronic pain status (chronic pain and no pain) and inhibitory control. Because age, gender, ethnicity, and race have been linked to differences in pain, psychological symptoms, and inhibitory control (Affleck et al., 1999; Campbell et al., 2008; Riley et al., 2002; Tsang et al., 2008; Williams et al., 1999; Yuan et al., 2008), all four were entered as covariates in the model.

Power Analysis

A sensitivity power analysis with a two-tailed significance level of $p < .05$ (Aiken et al., 1991; Cohen & Servan-Schreiber, 1992) was used to determine an appropriate effect size given our study's sample size using parallel mediation analysis (Cohen, 1988). The sensitivity power analysis was conducted in G*Power 3.1.9.2 (Faul et al., 2007) to test parallel mediation. With a sample size of 2345 participants and an alpha of .05, results showed that a minimum effect size of $R^2 = .008$ is needed to achieve power of 80% for a mediation analysis with four mediators (i.e., NA, PA, AA, anxious apprehension) and four covariates (i.e., age, gender, ethnicity, race).

Results

Characteristics of the Study Sample

Participants included 2345 emerging adults ages 18 to 27 years (mean age = 18.95 *SD* = 1.07). The majority of the full sample was female (74.4%; N = 1744). As shown in Table 5, approximately 18% (*n* = 419) of the total sample endorsed chronic pain. In addition, in the past month pain occurred one to three times per week for approximately 40% of the total sample (*n* = 932), and 5.6% reported experiencing daily pain. Pain in the past month usually lasted a few hours to half the day for about one third (29.0%; *n* = 681) of young adults. Common pain

locations included the back (*n* = 533; 22.7%), extremities (i.e., shoulders, ankles, knees; *n* = 472; 20.1%); and head/neck (*n* = 470; 20.0%).

Table 5. Demographic Information by Group (*N* = 2345).

	Group				
	Non-Chronic Pain (*n* = 1926)		Chronic Pain (*n* = 419)		
Variable	*M*	*SD*	*M*	*SD*	*p*
Age (years; range = 18-27)	18.95	1.07	18.94	1.08	.85[a]
Variable	N	%	N	%	
Female	1395	72.4	349	83.3	<.001[b]
Ethnicity					.71[b]
Hispanic/Latino	287	14.9	65	15.5	
Race					<.001[b]
American Indian/Alaska Native	12	.6	1	.2	
Asian	416	21.6	42	10.0	
Native Hawaiian/Pacific Islander	17	.9	3	.7	
African American	86	4.5	13	3.1	
Caucasian	1226	63.7	323	77.1	
Multiple Races Reported	98	5.1	24	5.7	
Did Not Report	71	3.6	13	3.2	

Note: Significance (*p*-value) after Bonferroni corrections in demographics across groups were evaluated with a *t*-test as designated by [a] or X^2 as designated by [b].

For individuals specifically reporting chronic pain (*n* = 419; Table 6), pain occurred one to three times per week for approximately 72% of the sample (*n* = 303), and 25% reported experiencing daily pain. In addition, pain usually lasted a few hours to half the day for over half (57.1%; *n* = 239) of young adults experiencing chronic pain, while 20.5% of the sample reported pain lasting the entire day. To evaluate differences in pain intensity, bother, and activity limitations among the two groups (non-chronic pain and chronic pain), Bonferroni-corrected

two-tailed independent sample *t*-tests were conducted to compare the group means. Individuals in the chronic pain group reported significantly more usual pain intensity, bother due to pain, and activity limitations due to pain (*p*s <.05) relative to the non-chronic pain group (Table 6).

Table 6. Group Characteristics of Pain, Psychological, and Inhibitory Control Variables.

	Group			
	Non-Chronic Pain (n = 1926)		Chronic Pain (n = 419)	
Variable				
Pain Location	*n*	%	*n*	%
None Listed	1133	58.8	10	2.4
Back	320	16.6	213	50.8
Head/Neck	310	16.1	160	38.2
Arms/Shoulders, Legs	292	15.2	82	19.6
Stomach/Abdomen	110	5.7	40	9.5
Chest/Lung	66	3.4	27	6.4
Generalized	27	1.4	13	3.1
Pain Prevalence	*n*	%	*n*	%
Chronicity				
Less than one month	1791	93.0	--	--
1-2 months	135	7.0	--	--
3 months or longer	--	--	419	100.0
One-Month Frequency				
Not at all	1171	60.8	11	2.6
1 time per week	464	24.1	75	17.9
2-3 times per week	228	11.8	165	39.4
4-6 times per week	31	1.6	63	15.0
Daily	26	1.4	105	25.1
No Response	6	0.3	0	0
Duration				
<1 hour	815	42.3	93	22.2
A few hours	328	17.1	149	35.6

Half the day	114	5.9	90	21.5	
All day	66	3.4	86	20.5	
No Response	603	31.3	1	.2	
	n (%)	M (SD)	*n* (%)	M (SD)	*p*
Pain Intensity	1629 (69.4)	3.46 (2.36)	418 (17.8)	5.77 (1.86)	<.001*
Pain Bother	1659 (70.7)	1.81 (0.90)	418 (17.8)	2.71 (0.86)	<.001*
Activity Limitations	1638 (69.9)	2.38 (2.07)	419 (17.8)	3.77 (2.30)	<.001*
	n (%)	M (SD)	*n* (%)	M (SD)	*p*
Positive Affectivity (PA)	1926 (82.1)	43.89 (11.32)	419 (17.9)	41.72 (12.06)	<.001*
Negative Affectivity (NA)	1926 (82.1)	18.35 (6.03)	419 (17.9)	19.76 (6.11)	<.001*
Anxious Arousal (AA)	1926 (82.1)	29.58 (10.35)	419 (17.9)	33.34 (11.31)	<.001*
Anxious Apprehension (APP)	1926 (82.1)	55.57 (13.93)	419 (17.9)	59.09 (14.09)	<.001*
	n (%)	M (SD)	*n* (%)	M (SD)	*p*
Inhibitory Control (IC)	1926 (82.1)	13.92 (3.23)	419 (17.9)	14.65 (3.08)	<.001*

Note: *p <.05

Correlations Among Pain, Psychological Health, and Executive Function

Pearson correlations were computed among the primary study variables (Table 7). Positive affectivity, negative affectivity, anxious arousal, anxious apprehension, and inhibitory control were significantly associated across the study sample (ps < .05).

Table 7. Correlations Among Affectivity and Inhibitory Control (*N* = 2345).

	1.	2.	3.	4.
1. Positive Affectivity (PA)				
2. Negative Affectivity (NA)	-.39			
3.Anxious Arousal (AA)	-.08	.60		
4. Anxious Apprehension (APP)	-.25	.40	.28	
5. Inhibitory Control (IC)	-.08	.38	.40	.19

Note. All shown correlations were significant (*p*s <.05)
PA = Positive Affectivity, NA = Negative Affectivity, AA = Anxious Arousal, APP = Anxious Apprehension, IC = Inhibitory Control

Psychological Symptoms and Inhibitory Control

To evaluate differences in psychological and inhibitory control symptoms among the two groups (non-chronic pain and chronic pain), two-tailed independent sample *t*-tests were conducted to compare the group means. Analyses comparing psychological symptoms (affectivity and anxiety) between the chronic pain and non- chronic pain groups revealed that individuals in the chronic pain group reported significantly more anxious arousal, negative affectivity, and anxious apprehension, as well as significantly less positive affectivity (*p*s < .05; Table 6), relative to those without chronic pain. The chronic pain group also reported significantly more inhibitory control difficulties compared to the no pain group (Table 6).

Parallel Mediation Analysis

A parallel mediation analysis (Figure 1) was conducted to evaluate whether self-reported psychological symptoms influenced the relation between chronic pain and inhibitory control deficits in individuals experiencing chronic pain. The model examined the effects of pain group

(non-chronic pain vs. chronic pain) mediated by psychological symptoms (positive affectivity, negative affectivity, anxious arousal, and anxious apprehension) on inhibitory control.

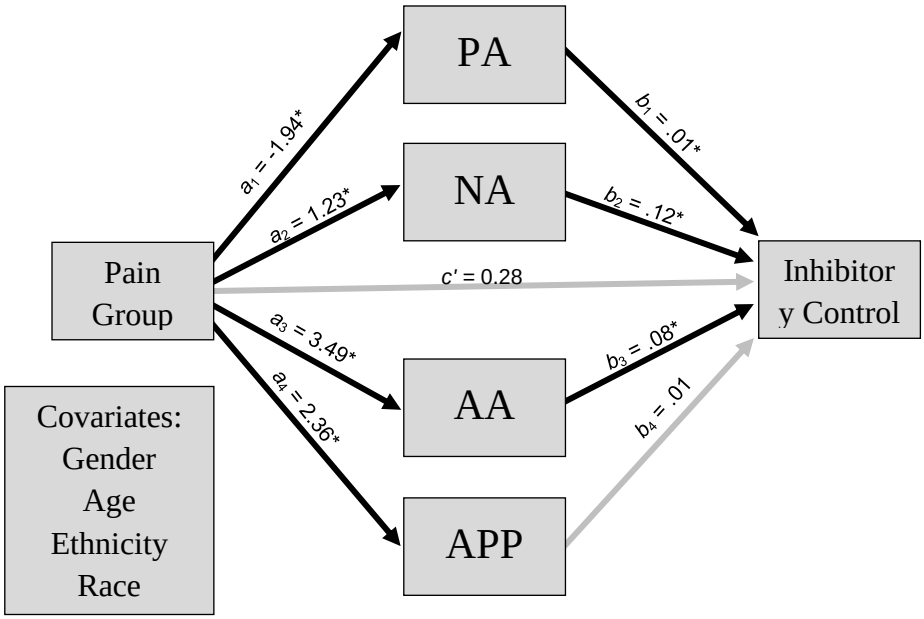
Age, gender, ethnicity, and race were included as covariates in the model (Table 8). In terms of these demographic covariates, gender was associated with positive affectivity, negative affectivity, and anxious apprehension, such that women were found to have higher levels of negative affectivity and anxious apprehension and lower levels of positive affectivity (*p*s < .05). Gender was not associated with anxious arousal. Older age was associated with higher levels of anxious apprehension and lower levels of positive affectivity (*p*s < .05). Race and ethnicity were not associated with psychological symptoms.

Regressions showed that pain group was associated with positive affectivity (path a_1 = -1.94, p <.05), negative affectivity (path a_2 = 1.23, p < .05), anxious arousal (path a_3 = 3.47, p < .05), and anxious apprehension (path a_4 = 2.36, p < .05) symptoms (Table 8). Chronic pain status (c' = .28, p = .09) was not *directly* significantly associated with inhibitory control when accounting for variance in demographic and psychological variables, indicating that individuals with chronic pain did not report higher levels of inhibitory control difficulties independent of variance associated with psychological symptoms. However, pain group was *indirectly* associated with inhibitory control via multiple pathways: positive affectivity, negative affectivity, and anxious arousal (*p*s < .05). 95% bootstrap confidence intervals based on 5,000 bootstrap samples were calculated for indirect effects. The 95% confidence intervals for the indirect effects of positive affectivity (95% CI [-.059, -.001], a_1b_1 = -.03), negative affectivity (95% CI [.066, .237], a_2b_2 = .15), and anxious arousal (95% CI [.167, .382], a_3b_3 = .268) did not include zero. Anxious apprehension was not associated with inhibitory control (b_4=.01; p =.10),

and the 95% confidence interval for the indirect effect for anxious apprehension (95% [-.005, .052], a_4b_4 = .019) included zero.

Thus, the indirect relation among pain group and inhibitory control was mediated via positive affectivity, negative affectivity, and anxious arousal, but not anxious apprehension. Because the direct pathway between chronic pain status and inhibitory control was non-significant, these findings primarily implicate *indirect* mediation pathways (Lennon et al., 2015; Zhao et al., 2010).

Figure 1. Structural Diagram of Parallel Multiple Mediator Model. Four mediators (PA = positive affect, NA = negative affect, AA = anxious arousal, and anxious apprehension) with pain group (non-chronic pain vs. chronic pain) as the predictor. Black arrows specify significant pathways while gray arrows represent insignificant pathways. The diagram shows 1) the total effect of group on inhibitory control and 2) the direct effect and causal pathways associating group with inhibitory control.



Note. PA = Positive Affectivity, NA = Negative Affectivity, AA = Anxious Arousal, APP = Anxious Apprehension, IC = Inhibitory Control; *ps < .05

Table 8. Unstandardized Model Coefficients for the Parallel Mediation Analysis (as described in Figure 1).

		M_1 (PA)				M_2 (NA)				M_3 (AA)				M_4 (APP)				Y (IC)			***Indirect Effects***			
																							95% CI	
Antecedent		Coef.	*SE*	*p*		Coef.	*SE*	*p*		Coef.	*SE*	*p*		Coef.	*SE*	*p*		Coef.	*SE*	*p*		*Coef.*	*Upper*	*Lower*
X (Group)	a_1	-1.94	.64	.00	a_2	1.23	.34	.00	a_3	3.47	.58	.00	a_4	2.36	.75	.00	c'	.28	.16	.09				
M_1 (PA)		---	---	---		---	---	---		---	---	---		---	---	---	b_1	.01	.01	.03	a_1b_1	-.030	-.059	-.001
M_2 (NA)		---	---	---		---	---	---		---	---	---		---	---	---	b_2	.12	.01	.00	a_2b_2	.150	.066	.237
M_3 (AA)		---	---	---		---	---	---		---	---	---		---	---	---	b_3	.08	.01	.00	a_3b_3	.268	.167	.382
M_4 (APP)		---	---	---		---	---	---		---	---	---		---	---	---	b_4	.01	.00	.10	a_4b_4	.019	-.005	.052
Covariates:																								
C_1 (GEN)		-1.66	.57	.00		.79	.30	.01		.55	.52	.29		8.06	.67	.00		-.20	.15	.18				
C_2 (Age)		-.51	.23	.03		.10	.12	.44		-.11	.21	.59		.93	.27	.00		-.03	.06	.58				
C_3 (ETH)		.61	.75	.42		-.06	.40	.89		-1.21	.69	.08		-.77	.89	.39		-.37	.19	.05				
C_4 (Race)		.15	.19	.43		.01	.10	.99		-.15	.18	.39		.26	.23	.25		-.00	.05	.98				
Constant	i_{M1}	54.57	4.94	.00	i_{M2}	15.27	2.60	.00	i_{M3}	33.75	4.53	.00	i_{M4}	24.43	5.83	.00	i_Y	10.09	1.31	.00				
R^2			.01				.01				.02				.07				.19					
F			4.66				4.58				8.31				33.87				59.29					
p			<.01				<.01				<.01				<.01				<.01					

Note. PA = Positive Affectivity, NA = Negative Affectivity, AA = Anxious Arousal, APP = Anxious Apprehension, IC = Inhibitory Control, GEN=Gender, ETH = Ethnicity

Discussion

Our study provides initial evidence of impaired inhibitory control and its association with psychological symptoms in an emerging adult sample that has a substantial amount of chronic pain. Of note, 18% of the emerging adults in the present study reported chronic pain symptoms for three or more months, and this chronic pain prevalence is within the range of prevalence rates reported in epidemiological studies (7.6-21%; Eriksen et al., 2003; Iversen et al., 2017; Kennedy et al., 2014; Mallen et al., 2006; Rosenbloom et al., 2017; Stinson et al., 2013). In the present study, emerging adults with chronic pain reported increased psychological symptomatology that was associated with self-reported inhibitory control. Psychosocial interventions that target affectivity and anxious arousal symptoms may improve function in emerging adults with chronic pain.

Regarding psychological symptoms reported in our sample, results replicated previous research (Bayram & Bilgel, 2008; Eisenberg et al., 2007; McGorry et al., 2011) and supported our hypothesis that increased pain levels were associated with increased psychological symptoms. In general, chronic pain was associated with higher levels of negative affectivity, anxious apprehension, and anxious arousal, as well as lower levels of positive affectivity. However, results from our exploratory parallel mediation analysis showed that only negative affectivity, positive affectivity, and anxious arousal mediated the indirect relation between pain group and inhibitory control. Notably, anxious apprehension was not a significant mediator indicating that with regard to anxiety symptoms, anxious arousal may be distinctly related to cognitive outcomes for individuals with chronic pain. Anxious arousal has been identified as a trait-based propensity to experience state fear more often and more easily with a temporally

stable pattern of hypervigilance between acute fear responses while anxious apprehension is theorized to better represent state anxiety in response to an acutely threatening situation (Sharp et al., 2015). The experience of co-morbid chronic pain and psychological distress in emerging adults may increase the risk for impaired cognitive function, with negative consequences for daily life function. Similar brain regions (e.g., PFC, ACC, insula) have been implicated in pain, inhibitory control, and emotional regulation (Bushnell et al., 2013; Shackman et al., 2011). Thus, difficulties regulating inhibitory control may be exacerbated by the added challenges of psychological symptoms in emerging adults experiencing chronic pain. Further, the onset of internalizing symptoms (i.e., withdrawal, somatic symptoms, and anxiety/depression) has been shown to peak during the adolescent and emerging adulthood, and thus emerging adults as a group may be particularly vulnerable to experiencing elevated levels of depression and anxiety (Bayram & Bilgel, 2008; Eisenberg et al., 2007; Hysenbegasi et al., 2005; McGorry et al., 2011; Paul & Brier, 2001).

Interventions that aim to reduce the cognitive burden associated with chronic pain by specifically focusing on reducing anxious arousal and negative affectivity and increasing positive affectivity may improve overall inhibitory control function. Such interventions may dramatically improve the quality of life of emerging adults experiencing chronic pain. Further, promoting interventions that focus on reducing the cognitive and emotional burden of chronic pain in emerging adults may improve long-term functional and health outcomes for which this age group may be particularly vulnerable (e.g., opioid misuse and abuse; Atluri et al., 2014; Birnbaum et al., 2011; O'Brien et al., 2017; Vowles et al., 2015). Mindfulness has recently been a target of intervention study for chronic pain due to its specific attention to reducing somatic symptoms

and stress (Kerr et al., 2013; Morone et al., 2008; Rosenzweig et al., 2010; Zeidan & Vago, 2016). More recently, techniques incorporating standardized mindfulness practices have been hypothesized to make available attentional resources that were once pain-focused in individuals with chronic pain and provide a framework for potential interventions (Kerr et al., 2013). Moreover, cognitive-behavioral therapy (CBT) is another highly effective treatment of chronic pain. CBT for chronic pain incorporates several techniques that reduce pain and distress in adults, including deep breathing, progressive muscle relaxation, and thought restructuring (Ehde et al., 2014). Overall, further research is needed to understand how psychological interventions may be tailored to the unique developmental needs of emerging adults.

Limitations and Future Directions

While this study has several notable strengths, several questions remain. The results from the present study are based solely on self-report measures of inhibitory control. Future research should focus on experimental measures of inhibitory control in order to assess the implications of chronic pain and psychological symptoms on this domain of cognitive control. Additionally, the present study is limited by its cross-sectional design, which precludes unequivocal conclusions about cause and effect. While some researchers have advocated against this practice since correlational data do not afford causal interpretation (Maxwell et al., 2011), Hayes advocated for this position: “We should not let the limitations of our data collection efforts constrain the tools we bring to the task of trying to understand what our data might be telling us about the processes we are studying” (p. 18; Hayes, 2017). Many researchers share this sentiment as indicated by their use of mediation analyses with cross-sectional data (Blashill & Vander Wal, 2010; Gaunt & Scott, 2014; Goodin et al., 2009; Kung et al., 2016; Lee et al., 2014; Li et al., 2011; Osborne et

al., 2015; Rees & Freeman, 2009; Schaie, 2005; Smith et al., 2016; Thai et al., 2016; Thomas & Bowker, 2015; Torres & Taknint, 2015; Webb et al., 2016). Thus, this study represents a critical first step in delineating associations that in future work could be examined as a potential mechanism influencing the link between chronic pain and executive dysfunction. These associations should be replicated in micro-longitudinal (experience sampling) designs or experimental pain paradigms. Although a unique aspect of this study is that the emerging adult sample are all college-attending young adults with chronic pain, many adolescents with chronic pain may not transition to college or drop out early due to the burden of chronic pain (Murray et al., 2020). Thus, it is unknown whether these results are more pronounced in non-college attending emerging adults with perhaps more severe and disabling chronic pain.

Conclusions

Overall, the results of this study indicate that chronic pain was associated with increased perceived inhibition impairments and results from a parallel mediation analysis showed a significant indirect effect between chronic pain status and perceived inhibition impairments via negative affectivity, positive affectivity, and anxious arousal. Future research can extend these results by examining longitudinal trajectories to expand our understanding of the impact of chronic pain on executive function deficits in emerging adults, including the key role of psychological symptoms in driving this association. Moving forward, it appears critical to develop effective evidenced-based interventions that target specific dimensions of psychological symptoms to improve the quality of life in individuals with chronic pain disorders.

CHAPTER FOUR

ANXIOUS AROUSAL AND ANXIOUS APPREHENSION ARE DISTINCTLY RELATED TO THE TEMPORAL COURSE OF RESPONSE INHIBITION

Successfully navigating day-to-day experiences through goal-directed behavior is dependent upon intertwined healthy emotional and cognitive processes. Anxiety is associated with numerous cognitive impairments and biases that often impede quality of life and the ability to successfully carry out activities of daily living. Both state and trait anxiety symptoms negatively impact a range of cognitive processes, including impaired inhibitory control functions (Banich et al., 2009; Engels et al., 2007; Notebaert et al., 2018; Toh & Yang, 2020; Warren et al., 2013). Since emotional information is often prioritized during information processing (Stockdale et al., 2020), the ability to exert control over automatic reactivity to emotional information is paramount to salubrious affective function. Notably, abnormalities in emotion reactivity and regulation processes are present across various types of psychopathology, including anxiety and depression (Pessoa & Ungerleider, 2004; Sharp et al., 2015).

More specifically, anxiety symptoms are associated with increased selective attentional bias for threat, decreased attentional control capacity in the context of emotional stimuli, and the impaired ability to suppress behaviors that are inappropriate in a given context that interfere with goal-driven behavior (Bar-Haim et al., 2007;
Derryberry & Reed, 2002; Pacheco-Unguetti et al., 2012; Sass et al., 2010). Response inhibition, or the ability to withhold automatic responses that are *not* task-relevant (Mostofsky &

Simmonds, 2008), is important to carrying out daily life activities in an effective manner. Response inhibition is implemented by a network of brain regions including dorsomedial and lateral PFC, right inferior frontal cortex, and dorsal anterior cingulate cortex (dACC; Aron et al., 2014; Banich et al., 2009; Miller & Cohen, 2001; Silton et al., 2010). Previous research has shown that distinct dimensions of anxiety symptoms (e.g., anxious arousal and anxious apprehension) are disruptive to inhibitory control processes, including response inhibition (Engels et al., 2007, 2010; Guha et al., 2020; Sharp et al., 2015; Silton et al., 2011; Warren et al., 2013; Williams et al., 1999). Understanding how anxious arousal and anxious apprehension are uniquely associated with the temporal course of the neural correlates of response inhibition is paramount to developing effective treatment methods that can target and modify specific anxiety symptom subtypes.

Anxious arousal is marked by sympathetic hyperarousal and hypervigilance in the presence of mild stressors (Heller et al., 1997; Nitschke et al., 2000; Sharp et al., 2015; Watson, 2005). Anxious arousal refers to a trait-based propensity to experience state fear more often and more readily with a temporally stable pattern of hypervigilance between acute fear responses (Sharp et al., 2015). Anxious arousal is associated with increased sensitivity to distracting and mildly threatening stimuli, which in turn appears to impact attentional networks via an overactive engagement of bottom-up attention (e.g., attention guided by externally driven factors; Spielberg et al., 2013). Thus, anxious arousal may be associated with abnormalities in response inhibition due to tonic levels of heightened arousal in response to frequently perceiving exteroceptive and interoceptive stimuli as noxious.

While some studies have used descriptors such as “worry” or “fear” to characterize anxious arousal, these terms are theorized to better represent state anxiety in response to an acutely threatening situation and are distinguished as anxious apprehension (Sharp et al., 2015). Anxious apprehension is conceptualized as a persistent pattern of engaging in negative, repetitive thinking about (both proximal and distal) perceived threats, which also can be thought of as an enduring pattern of state worry (Engels et al., 2007; Ruscio et al., 2001). In threating situations, individuals high in anxious apprehension appear to immediately engage neural networks that implement worry, leading to a reduced initial threat response (Sharp et al., 2015). Anxious apprehension may hinder the ability to inhibit negative, maladaptive thinking patterns which in turn appears to impact attentional networks via an interference with top-down attentional control (i.e., attention guided by internal factors) and adaptive conflict resolution in the context of both negative affective distractors as well as irrelevant stimuli in Stroop paradigms without affective distractors (Engels et al., 2007, 2010; Guha et al., 2020; Herrington et al., 2010; Silton et al., 2011), suggesting a more general impairment in inhibitory control processes (Sharp et al., 2015).

Anxious Arousal and Anxious Apprehension are Characterized by Distinct Patterns of Neural Activity

Anxious arousal has been characterized by greater right hemisphere activity, primarily in lateral frontal areas in functional Magnetic Resonance Imaging (fMRI) studies (Nitschke et al., 1999), although also involving temporal and parietal regions that are involved in hypervigilance, attentional biases, and dispositional tendencies (Burdwood et al., 2016; Compton et al., 2003; Engels et al., 2007; Nitschke et al., 1999). Specifically, anxious arousal has been associated with activity in right inferior temporal gyrus (ITG) and middle temporal gyrus (MTG), both of which

are thought to implement threat detection (Engels et al., 2007; Nitschke et al., 2000; Spielberg et al., 2013). High anxious arousal was related to hyperactivity in these temporal regions while attempting to ignore negatively valanced words, indicating a lower threshold to engage in a state-based fear response when exposed to mildly threatening stimuli. Results from the Engels et al., (2007) study showed that negative emotional words evoked greater right-hemisphere temporoparietal activity specific to individuals with high trait anxious arousal. These results are consistent with the theory that the right hemisphere houses an integrated system for responding to threat, promoting sympathetic nervous system activity, spatial attention, visual scanning of the environment, and sensitivity to meaningful nonverbal cues (Compton et al., 2003; Engels et al., 2007; Keller et al., 2000). In the Engels et al. (2007) study, the anxious arousal group showed less frontal activity than the control group from medial PFC extending into rostral anterior cingulate cortex (rACC), which is consistent with brain regions that are associated with specific event-related potentials (ERPs; N200 and N450) that are observed during a range of cognitive control tasks (Engels et al., 2007; Hanslmayr et al., 2008; Liotti, Woldor, et al., 2000; Szűcs et al., 2009), and are discussed in more detail below. Further, individuals with high anxious arousal have shown early engagement of a threat response system, evidenced by activity in right superior frontal gyrus (SFG), ITG, and MTG that habituated over time (Sharp et al., 2015).

Anxious apprehension is primarily implemented in neural circuits that specialize in language production and verbal working memory (Engels et al., 2007, 2010; Nitschke et al., 1999, 2000). Engels and colleagues (2007) found left inferior frontal gyrus (IFG; i.e., Broca's area) activity was related to biased processing of negative information in individuals with anxious apprehension, such that activation of left IFG was only observed when contrasting

negatively valanced stimuli to neutral stimuli. It has also been surmised that left IFG is activated in individuals high in anxious apprehension due to the rehearsal of task rules via articulation, which has been shown via increased activity in left IFG in verbal working memory tasks (Paulesu et al., 1993). Further, a recent study by Guha and colleagues (2020) revealed that greater connectivity from Broca's area to amygdala during tasks requiring greater cognitive control. Findings revealed that greater connectivity may serve as a compensatory mechanism by which individuals high in anxious apprehension may engage in successful compensation for interference effects while employing inhibitory control (Guha, 2020). Related, anxious apprehension was associated with modulated activity in a frontocingulate network, with earlier left DLPFC (300 – 440) activity preceding later dACC activity (520 – 680 ms) during a cognitive Color-Word Stroop Task (CWST), with increased later dACC activity thought to reflect a compensatory mechanism for an initial failure in earlier top-down control of attention implemented by DLPFC for individuals high in anxious apprehension (Sharp et al., 2015; Silton et al., 2011).

A critical caveat is that with the exception of Silton et al. (2011), nearly all of the reviewed extant literature regarding the neural correlates of anxious arousal and anxious apprehension was conducted using attentional control paradigms with affective stimuli and/or fMRI methods. Although these studies are paramount to advancing our understanding of anxiety subtypes, they provide minimal insight regarding how these anxiety subtypes might influence the precise temporal course of response inhibition implemented in an experimental context that does not include affective stimuli (e.g., emotion words or images). Thus, while anxious arousal may distinctly impact brain regions related to earlier bottom-up processing and anxious apprehension

may interfere with brain regions associated with later top-down control functions, these findings may be specific to attentional control tasks with affective stimuli. In order to evaluate how anxiety subtypes influence the temporal course of response inhibition that is not mitigated by affective stimuli, the present study was conducted with a traditional cognitive Color-Word Stroop Task (CWST) using incongruent/congruent color words and neutral word stimuli.

The Temporal Course of Neural Activity during Inhibitory Control Processes

A number of electrophysiological and functional imaging studies have implicated several brain regions during response inhibition, including ACC, DLPFC, posterior parietal cortex, and inferior parietal cortex (Egner et al., 2007; MacLeod & MacDonald, 2000; Peterson et al., 2002; Silton et al., 2010; van Veen & Carter, 2005). Notably, many of these brain regions overlap with patterns of abnormal brain activity observed in individuals with anxious arousal (Engels et al., 2007; Nitschke et al., 2000; Spielberg et al., 2013). EEG methods, including event-related potentials (ERPs), offer precise temporal resolution to characterize the rapid succession of neural events that occur during response inhibition, particularly with regard to resolving stimulus interference. While several ERP components have been associated with interference (i.e., slower response time to incongruent than congruent color word stimuli) on the CWST, findings suggest that additional research is needed to resolve the precise time course of neural processes observed in the CWST (Coderre et al., 2011; Galer et al., 2014). Previous research investigating the time course of inhibitory control processes evoked by the CWST has generally included the N200, N450, and conflict SP components. While prior research has addressed the influence of anxious arousal specifically on the recruitment and allocation of neural resources during emotion interference processing using state-based threatening information (Engels et al., 2007, 2010;

Guha et al., 2020), the present study investigated the influence of anxious arousal on the three most established ERP components (N200, N450, and conflict SP) observed during response inhibition on the CWST.

N200

The N200 (a negative deflection with a peak latency approximately 200 to 250 ms following the stimulus presentation) has been observed in CWST paradigms (Holmes & Pizzagalli, 2008; Silton et al., 2010) as well as in other visual interference and response inhibition tasks that involve monitoring for stimulus conflict (Folstein & Van Petten, 2008; Kopp et al., 1996; Yeung et al., 2004). Relevant to the CWST, some studies have shown that the N200 differentiates between incongruent and congruent trials, with greater negative amplitude for incongruent than congruent conditions (Folstein & Van Petten, 2008; Holmes & Pizzagalli, 2008; Kopp et al., 1996; Silton et al., 2010; Yeung et al., 2004). This negative, frontally distributed component is thought to be generated by inferior/lateral PFC or dACC (Jackson et al., 2001; Kiefer et al., 1998). The N200 has been related to the detection of perceptual novelty, response conflict, error monitoring, and inhibiting a prepotent response system (Folstein & Van Petten, 2008). While studies have found decreased N200 amplitude for conflict adaptation (i.e., the adjustment of performance based on previous-trial conflict) in individuals diagnosed with generalized anxiety disorder (GAD) and increased N200 amplitude in individuals with anxiety sensitivity (i.e., a self-monitoring trait of anxiety), these studies were not characterized by the subtypes of anxious arousal or anxious apprehension (Larson et al., 2013; Sehlmeyer et al., 2010). Previous studies have not characterized the association among anxious arousal or anxious apprehension and the N200 component during an inhibitory control task.

N450

The N450 is one of the most commonly identified ERP components associated with the CWST. Distinguishable as a negative-going deflection 300 – 500 ms following stimulus onset with frontocentral topography, the N450 ERP waveform typically peaks around 450ms following the presentation of an incongruent stimulus (Liotti et al., 2000; van Veen & Carter, 2005; West & Alain, 1999). N450 results are relatively robust, with multiple studies reporting that incongruent conditions elicit increased amplitude relative to congruent or neutral conditions (Hanslmayr et al., 2008; West et al., 2005). EEG source localization efforts have consistently reported that ACC dipoles account for the most variance in the topography of the N450 (Hanslmayr et al., 2008; Liotti, Woldorff, et al., 2000; Szűcs et al., 2009). Further, evidence accrued from functional magnetic resonance imaging studies (fMRI) have implicated increased ACC activity in response to incongruent compared to congruent stimuli, consistent with a role in the detection of stimulus conflict (MacLeod & MacDonald, 2000; Szűcs & Soltész, 2012). Thus, N450 is considered to reflect a more automatic conflict monitoring mechanism rather than indicating intentional control functions (Suárez-Pellicioni et al., 2014). Previous studies have not characterized the associations among anxiety subtypes and the N450 component.

Conflict SP

On response inhibition tasks, the conflict slow potential (SP) follows the N450, emerging at central sites approximately 500 ms after stimulus onset. It is thought to be a relatively tonic, positive sustained brain potential that is generated by the middle or inferior frontal gyrus (LPFC) and the left extrastriatal cortices (Suárez-Pellicioni et al., 2014), and topographically appears as sustained parietal positivity/lateral frontal negativity (Larson et al., 2009). It has been observed

to be enhanced following correct incongruent trials compared to congruent trials or errors and may play a role by resolving response monitoring detected by the N200 and resolving conflict monitoring detected by the N450 (Larson et al., 2009; Suárez-Pellicioni et al., 2014). However, the specific cognitive processes associated with the conflict SP remain under investigation. Past studies have shown that the conflict SP is associated with response time and accuracy (West et al., 2005). This component has not yet been characterized in individuals with anxious apprehension or anxious arousal.

The Present Study

The present study was conducted in order to evaluate whether anxious arousal symptoms compared to anxious apprehension symptoms differentially influence the implementation of the temporal course of neural activity during response inhibition. The analyses were conducted in an exploratory manner since limited research has studied the relation among anxiety symptoms and ERPs during a "cognitive" response inhibition task. As noted above, previous research investigating these anxiety subtypes was mostly conducted with fMRI and/or within the context of experimental tasks involving affective stimuli. Behavioral (response time and accuracy) and ERP components (N200, N450, and conflict SP) were analyzed in the present study.

Both categorical and dimensional approaches were used to probe the relations among distinct dimensions of anxiety symptoms and the behavioral and neural correlates of response inhibition. Similar to methods used in previous research to categorize individuals with predominantly anxious arousal or anxious apprehension symptoms (Engels et al., 2007; Nitschke et al., 1999, 2000; Sass et al., 2010; Silton et al., 2010, 2011), participants were identified as those who scored high on anxious arousal symptoms, but low on anxious apprehension

symptoms (Anxious Arousal Group; ARO), and vice versa (Anxious Apprehension Group; APP). Participants in both groups scored low on affectivity measures indicative of depression symptoms. A group of participants was also identified as those who scored low on anxious arousal, anxious apprehensions, and depression symptoms (Control Group; CG). However, a dimensional approach was also implemented since The National Institute of Mental Health (NIMH) Research Domain Criteria Initiative (RDoc; Insel & Cuthbert, 2009) encourages applying dimensional approaches to transcend traditional categorical classification (Blanchard-Fields, 2005; Blanchette & Richards, 2010; Mitchell & Phillips, 2007; Yiend, 2004) in order to accurately characterize dimensions of function that are associated with the range of human behavior. Further, we also included positive and negative affectivity dimensions to account for variance associated with co-occurring depression symptoms.

Method

Study Overview

The present research study used archival data from a large research study that presented color-word and emotion-word Stroop tasks during EEG and fMRI data acquisition in two separate counter-balanced sessions. Data were collected between 2002 – 2007. Only CWST data collected during the EEG session were investigated in the proposed study. Participants enrolled in this study were recruited from a pool of undergraduate students who completed a series of questionnaires as partial fulfillment of enrollment in an introductory psychology course. The University's Institutional Review Board approved all study methods.

Participants

Participants (N = 97) were paid volunteers recruited from introductory psychology classes via group questionnaire screening sessions. Three participants were considered outliers

on ERP measures (over three SDs from the mean for at least one component) and were omitted from subsequent analyses. Casewise deletion was used to omit participants (n = 2) that had missing responses to any questionnaires used in the present study. The final sample used for analyses included n = 92 participants (55% female, 85% Caucasian) between the ages of 18 - 21 (M = 18.86, SD = .91). All participants were right-handed, native speakers of English with self-reported normal color vision.

Demographics

Participants reported information regarding age, gender, handedness, and ethnicity via Scantron multiple-choice answer sheets.

Questionnaire Measures and Clinical Interview Informed Participant Selection

Participants for the present study were selected from a larger sample and categorized into three groups (ARO, APP, CG) based on threshold cutoffs on measures of depression and anxiety (Engels et al., 2007; Nitschke et al., 1999) for the Anhedonic Depression and Anxious Arousal scales of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995) and the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990).

Cutoff scores for group membership (ARO, n = 29; APP, n = 25; CG, n = 38) were assigned using 50th and 80th percentiles based on sample distributions for each measure. All ARO and CG participants scored below the 50th percentile on the PSWQ. ARO participants scored above the 80th percentile on the 17-item subscale of the MASQ-AA and below the 50th percentile on the PSWQ and MASQ 8-item anhedonic depression (MASQ-AD-8; Nitschke et al., 2000). APP participants scored above the 80th percentile on the PSWQ and below the 50th percentile on the MASQ-AA and MASQ-AD-8. CG participants scored below the 50th

percentile on the PSWQ and the MASQ-AA and anhedonic subscales. The Structured Clinical Interview for Axis I Disorders, Non-patient edition (SCID; First et al., 1997) was also administered to assess psychopathology. In the present study, CG participants were also required to be free of clinical diagnoses per the SCID. All participants completed these questionnaire measures again during their first lab visit and those are the scores that are reported in this present study. McDonald's Omega was obtained for each subscale of the MASQ with the original validation sample showing moderate to high internal consistency across all three subscales (Watson et al., 1995). The present study replicated these findings (NA, ω = .81; PA, ω = .93; and AA, ω = .89). The present study found good internal consistency for the PSWQ (ω = .94), which consistent with previous research (Henning et al., 2007).

Task Design

Participants completed a color-word and emotion-word Stroop tasks during an fMRI session and an EEG session. Behavioral and EEG data from the CWST were included in the present study. The order of presentation of the two Stroop tasks within a session was counterbalanced across participants, as was the order of the EEG and fMRI sessions. The CWST consisted of blocks of color-congruent or color-incongruent words alternating with blocks of neutral words, with 256 trials in 16 blocks (4 color-congruent, 4 color-incongruent, 8 neutral). Half of the trials in congruent and incongruent blocks were neutral to prevent the development of word-reading strategies. There were eight orders of stimulus presentation for each Stroop task, designed specifically to control stimulus order effects. Each participant received one of the eight orders.

Each trial consisted of one word presented in one of four ink colors (red, yellow, green, blue). Trials began with the presentation of a word for 1500 ms, followed by a fixation cross for 275 ms to 725 ms (onset to onset ITI 2000 +/− 225 ms). Word presentation and response recording were controlled by STIM software (James Long Company, Caroga Lake, NY). Words were presented on a CRT monitor 1.35 m from the participants' eyes, for a vertical span of 1.5 degrees and a horizontal span of 3.2 – 8.7 degrees. Participants responded with their middle and index fingers, with each task using the same mapping of color to button. There was a color-to-key-mapping acquisition phase of 32 practice trials. In addition to the 16 word-blocks, there were four fixation blocks – one at the beginning, one at the end, and two in the middle of the session. In the fixation condition, a brightened fixation cross was presented for 1500 ms.

Stroop Interference

Stroop interference occurs when conflicting stimuli are simultaneously presented. In the case of the CWST, conflicting stimuli are presented such that the name of one color (e.g., the word "RED") is printed in the ink of another color (e.g., blue). These task trials are referred to as incongruent trials. Interference does not occur when stimuli characteristics agree (e.g., the word "RED" is printed in red ink). Trials in which the word name and ink color match are referred to as congruent trials. To obtain an index of reaction time (RT) interference, RT on correct congruent trials was subtracted from RT on correct incongruent trials. To obtain an index of accuracy interference, accuracy for incongruent trials was subtracted from accuracy for congruent trials.

Apparatus and Physiological Recording

EEG was recorded with a custom-designed Falk Minow 64-channel cap with Ag/AgCl electrodes spaced equidistantly, extending inferiorly to the F9/F10 ring of the 10-10 System. The left mastoid served as the online reference for all EEG and EOG sites. Electrodes were placed above and below each eye and near the outer canthus of each eye recorded vertical and horizontal EOG for off-line eye-movement artifact correction of EEG. Electrode impedances were maintained below 20 kOhms. Half-power amplifier bandpass was 0.1 to 100 Hz, and data were digitized at 250 Hz. Electrode positions were recorded using a Zebris ELPOS digitizer (Zebris Medizintechnik, Tübingen, Germany).

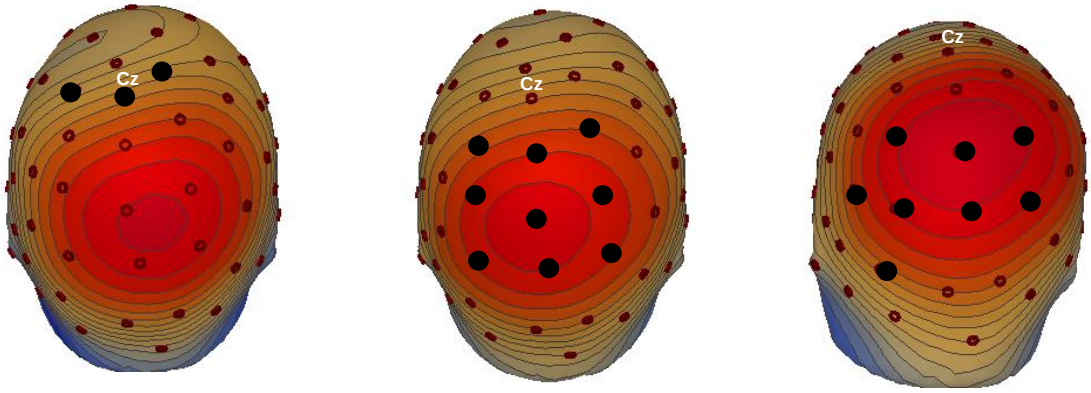
EEG Data Reduction

EEG data processing was performed in Brain Electrical Source Analysis software (BESA, Version 7.0). Following the adaptive artifact correction method (Ille, Berg, Scherg, 2002) ocular artifacts were corrected using a spatial PCA filter. Muscle and other artifacts were removed through automated artifact scan of raw data (fixed threshold criterion: amplitude > 120uV; gradient > 75 uV, low signal <.01hz). Baseline adjustment was computed using the averaged amplitude of 200 ms pre-stimulus onset. Data were re-referenced using an average reference for analyses and digitally filtered with a 0.1 Hz high-pass filter and 30 Hz low-pass filter. Stimulus-locked averages were calculated to ascertain mean amplitudes for N200, N450, and the conflict SP. Centroid latency (Clayson et al., 2013; Dien, 2010) was computed for N200 and N450 components across conditions. Electrode selection and temporal windows were informed by visual inspection of data as well as *a priori* judgments based on findings from previous studies investigating these ERP components in similar contexts (i.e., Silton et al., 2010).

Amplitudes were extracted within the following latency windows: N200: 220-320ms, N450: 36-472ms, conflict SP: 600-900ms. EEG electrode sites for each component were based on visual inspection of grand average waveforms and scalp topography (see Figure 2).

Figure 2. Scalp Topographies. Scalp topographies illustrating areas of maximal voltage for N200, N450, and conflict SP after stimulus onset. For N200, values range from red = +6.38 uV/cm^2 to blue = -6.38 uV/cm^2. For N450, values range from red = +9.02 uV/cm^2 to blue = -9.02 uV/cm^2. For conflict SP, values range from red = +3.19 uV/cm^2 to blue = -3.19 uV/cm^2. BESA time-domain filter used for all graphs: 0.1 - 30 Hz.

A. N200, 276 ms **B.** N450, 428 ms **C.** conflict SP, 660 ms



Data Analysis Plan

All analyses were conducted with SPSS version 26.0. Descriptive statistics were calculated for all variables (self-report measures, behavioral measures, and ERP components). A series of repeated measures analysis of variance (ANOVAs) was conducted to examine whether ERP components vary by congruency (i.e., congruent and incongruent trials) and to evaluate whether group differences were observed for the ERP components. Associations among psychological symptoms (i.e., anxious arousal, anxious apprehension, and affectivity), behavioral measures (i.e., RT and accuracy), and ERP components (i.e., N200, N450, and

conflict SP amplitude, N200 and N450 centroid latency) were examined using Pearson two-tailed correlations. Finally, a series of linear regressions was conducted to evaluate the influence of dimensional psychological symptoms on behavioral measures and ERP components.

Power Analysis

For the categorical approach, a sensitivity power analysis with a two-tailed significance level of $p < .05$ (Aiken et al., 1991; Cohen & Servan-Schreiber, 1992) was used to determine an appropriate effect size given our study's sample size (Cohen, 1988). The sensitivity power analysis was conducted in G*Power 3.1.9.2.(Faul et al., 2007) to evaluate a between-subjects ANOVA with three levels of group. With a sample size of 92 participants and an alpha of .05, results showed that a minimum effect size of $R^2 = .16$ is needed to achieve power of 80%. For the dimensional approach, a sensitivity power analysis with a two-tailed significance level of $p < .05$ was conducted to evaluate a linear regression analysis with four predictors (i.e., Anxious Arousal, Anxious Apprehension, Negative Affectivity, and Positive Affectivity) and one covariate (gender). With a sample size of 92 participants and an alpha of .05, results showed that a minimum effect size of $R^2 = .15$ is needed to achieve power of 80%.

Results

Group Demographics and Characteristics

Demographic characteristics of the full study sample and each diagnostic group (CG, ARO, APP) are presented in Table 9. The groups were similar in age ($F(2, 89) = .71$, $p = .50$), although they differed in gender composition ($\chi^2(2, n = 92) = 9.00$, $p = .01$), with a higher percentage of women in the anxious apprehension group compared to the other two groups. Participants completed SCID interviews and diagnostic counts for lifetime history of anxiety,

depression, or co-occurring anxiety/depressive disorders are reported in Table 10. A series of one-way ANOVAs was calculated to confirm that groups (ARO, APP, CG) differed on dimensional psychopathology measures (affectivity, anxious arousal, and anxious apprehension; Table 10). Bonferroni adjusted post-hoc *t*-tests revealed a significant difference between groups on the anxious arousal scale ($F(2,89 = 22.87$, $p = <.001$) such that ARO participants reported significantly higher levels of anxious arousal compared to APP ($p = <.001$) and CG ($p = <.001$). APP and CG participants did not differ on anxious arousal scores. There was also a significant difference between groups on the anxious apprehension scale ($F(2, 89) = 72.10$, $p = <.001$) such that APP participants reported more anxious apprehension than CG participants ($p =<.001$) and ARO participants ($p = <.001$). There was also a significant difference between groups on negative affectivity (F (2, 89) = 9.21, $p = <.001$) such that APP participants reported significantly more negative affectivity than CG participants ($p = <.001$), although no group differences were found between APP and ARO. There were no group differences in scores for positive affectivity.

Table 9. Demographic Information by Full Sample and by Group (*N* = 92).

			Diagnostic Group							
	Full Sample		Control (n = 38)		Anxious Arousal (*n* = 29)		Anxious Apprehension (*n* = 25)			
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F* (2, 89)	*p*
Age (years)	18.86	.91	18.89	1.01	18.97	.91	18.68	.75	.71	.50
Variable	*N*	%	*N*	%	*N*	%	*N*	%	X^2	*p*
Female	51	55.4	16	42.1	15	51.7	20	80.0	9.00	**.01***
Race										
Caucasian	78	84.8	35	92.1	24	82.8	19	76.0		
Asian	7	7.6	2	5.3	2	6.9	3	12.0		
African American	3	3.3	1	2.6	2	6.9	1	4.0		
Multiracial	3	3.3	0	0	1	3.4	1	4.0		
No Response	1	1.1	0	0	0	0	1	4.0		

Note: $*p < .05$

Table 10. Group Characteristics of Psychological Symptoms, Behavioral Measures of Response Inhibition, and ERP Components.

	Diagnostic Group							
	Control Group (*n* = 38)		Anxious Arousal (*n* = 29)		Anxious Apprehension (*n* = 25)			
SCID Diagnostic Group	*N*	*%*	*N*	*%*	*N*	*%*		
Depression	0	0	0	0	1	4.0		
Anxiety	0	0	2	6.9	5	20.0		
Comorbid Anx/Dep	0	0	0	0	1	4.0		
Dimensional Measures of Psychopathology	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F* (2,89)	*p*
MASQ-AA	21.24	3.48	31.07	8.03	24.52	6.05	22.86	**<.001*^**
MASQ-NA	12.39	2.28	14.17	2.87	15.88	4.46	9.21	**<.001**$^{+}$
MASQ-PA	32.79	8.77	31.34	10.75	35.56	8.65	1.38	.26
PSWQ - AP	35.42	10.70	38.24	11.73	65.32	7.16	72.10	**<.001*$^{+}$**
Congruent Trials								
Accuracy (errors)	.71	1.35	1.03	1.32	.60	.82	.31	.73
Response Time (ms)	622.14	76.42	630.49	109.32	610.59	94.77	.96	.39
N200 Amplitude (uV)	.52	2.55	.87	2.37	1.76	1.62	2.28	.11
N200 Latency (ms)	407.95	7.78	408.75	7.06	409.89	9.78	.42	.66
N450 Amplitude (uV)	4.88	2.30	4.76	2.65	5.09	2.01	.13	.88
N450 Latency (ms)	413.20	4.48	413.48	5.08	414.64	5.22	.69	.50
SP Amplitude (uV)	1.29	2.06	1.08	1.82	1.44	1.66	.25	.78
Incongruent Trials								
Accuracy (errors)	2.18	2.26	3.10	3.31	2.16	2.06	.08	.93
Response Time (ms)	807.09	142.35	799.45	126.39	793.74	132.98	1.28	.28
N200 Amplitude (uV)	-.28	2.70	.74	2.31	1.23	1.54	3.53	**.03**$^{+}$
N200 Latency (ms)	411.02	8.45	411.91	10.01	416.16	11.36	2.21	.12
N450 Amplitude (uV)	4.11	1.82	3.89	1.92	4.44	2.25	.53	.59
N450 Latency (ms)	413.56	5.06	414.25	4.87	416.23	5.53	2.09	.13
SP Amplitude (uV)	2.04	1.60	1.78	1.94	2.45	2.14	.85	.43
Stroop Interference								
Accuracy (errors) (Con Acc-Inc Acc)	2.32	2.78	2.31	3.34	1.96	2.44	.14	.87
Reaction Time (ms) (Inc RT – Con RT)	184.95	113.26	168.96	100.21	183.15	105.54	.20	.82

Note. AA = anxious arousal, NA = negative affect, PA = positive affect, AP = anxious apprehension/worry, RT – response time

*ARO is significantly different from APP; ^ARO is significantly different than CG; $^{+}$APP is significant different from CG

Behavioral Results: Confirmation of Stroop Interference Effect and Evaluation of Group Effects

Initial repeated measures ANOVAs were conducted to ensure that a Stroop interference effect was observed for the present sample (Table 11). Indeed, incongruent RT was greater than congruent RT ($p < .001$) and participants were less accurate on incongruent than congruent trials ($p < .001$). Pearson correlation analyses revealed RT and accuracy for congruent and incongruent trials were associated with each other (*r*s range from .23 – .61; Table 12), and slower RT was generally related to increased accuracy indicating a RT-accuracy tradeoff. One-way ANOVAs were conducted to evaluate the effects of categorical group on RT and accuracy for incongruent and congruent trials. Group differences were not observed for RT, accuracy, or Stroop interference effects (Table 11).

Table 11. Repeated Measures ANOVAs for Task Condition (Congruency) for Behavioral Data and ERPs.

A. Accuracy (errors)			
	F	*p*	partial η2
Congruency	39.13	< **.001***	.31
Congruency x Group	.48	.62	.01
B. Response Time (ms)			
	F	*p*	partial η2
Congruency	248.90*	**< .001***	.74
Congruency x Group	0.20	.82	.01
C. N200 Amplitude			

	F	*p*	partial η2
Congruency	6.71*	**.01***	.07
Congruency x Group	1.18	.31	.00

D. N200 Latency

	F	*p*	partial η2
Congruency	19.67*	< **.001***	.18
Congruency x Group	1.16	.32	.03

E. N450 Amplitude

	F	*p*	partial η2
Congruency	18.33	**< .001***	.17
Congruency x Group	.12	.89	.00

Note: *p <.05

F. N450 Latency

	F	*p*	partial η2
Congruency	2.78	.10	.03
Congruency x Group	.43	.65	.01

G. Conflict SP Amplitude

	F	*p*	partial η2
Congruency	29.32	**< .001***	.25
Congruency x Group	.35	.71	.01

Table 12. Pearson Correlations (Bonferroni adjusted) for Full Sample.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. MASQ-AA										
2. MASQ-NA	**.30***									
3. MASQ-PA	.03	**.32***								
4. PSQW	.06	**.35***	**.27***							
5. Congruent - RT	-.03	-.04	-.03	-.03						
6. Congruent - ACC	.09	.08	-.02	-.06	**.27***					
7. Incongruent - RT	-.07	-.13	-.06	-.07	**.61***	.05				
8. Incongruent - ACC	.04	.02	-.05	-.06	**.23***	**.27***	**.40***			
9. Interference – RT	-.07	-.14	-.05	-.06	-.10	-.17	**.73***	**.30***		
10. Interference – Acc	-.02	-.05	-.11	-.01	.13	-.09	**.31***	**.81***	**.28***	
11. N200 Congruent Amplitude	.11	.16	.06	**.21***	**-.25***	-.20	-.19	-.15	-.02	-.11
12. N200 Congruent Latency	.03	-.09	.06	**.22***	.01	.04	-.07	-.14	-.10	-.16
13. N200 Incongruent Amplitude	.19	.14	.06	**.22***	.03	-.02	-.07	-.06	.06	-.01
14. N200 Incongruent Latency	.02	.06	.12	**-.31***	-.03	-.00	-.04	-.06	-.03	-.07
15. N450 Congruent Amplitude	-.14	-.03	.06	.05	**-.31***	-.08	**-.28***	-.03	-.08	.07
16. N450 Congruent Latency	-.05	-.14	.10	.15	-.09	.04	-.13	-.19	-.09	**-.25***
17. N450 Incongruent Amplitude	-.17	.07	.05	.10	-.05	-.06	-.13	.03	-.12	.05
18. N450 Incongruent Latency	-.13	-.07	.13	.20	-.02	.02	-.03	.14	.05	.08
19. SP Congruent Amplitude	**-.22***	-.12	-.02	.07	.10	-.01	.00	.03	-.09	.11
20. SP Incongruent Amplitude	**-.33***	-.04	.04	.06	.03	-.09	-.00	.09	-.03	.05

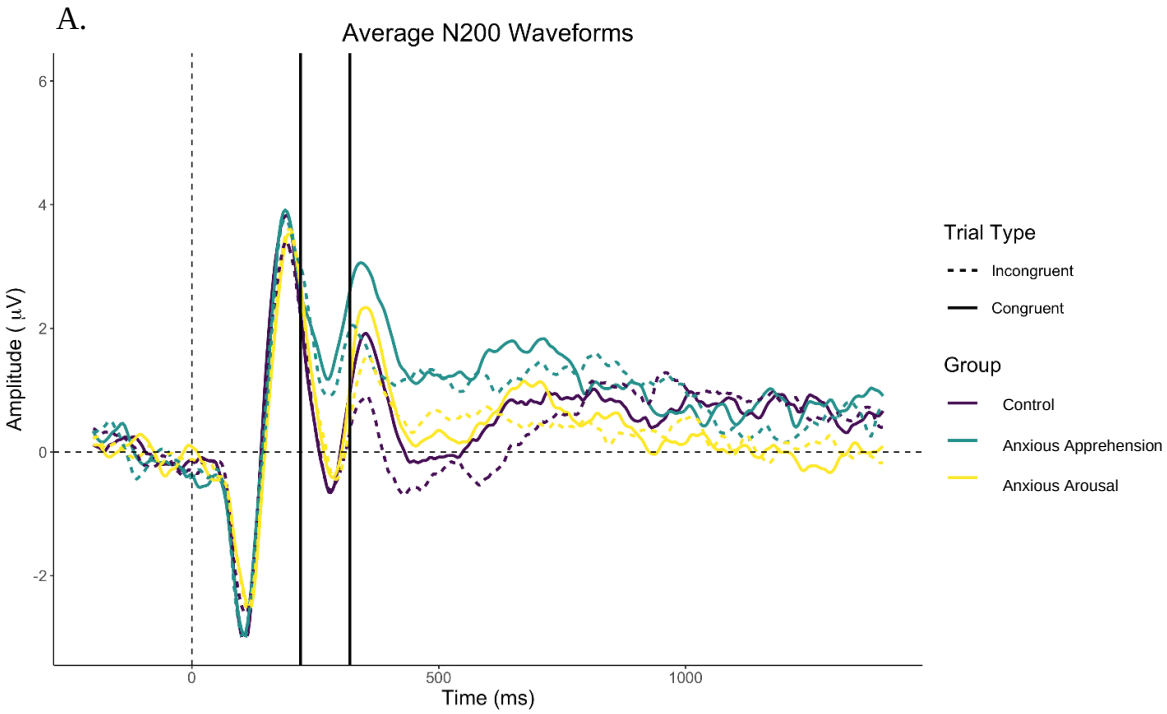
*Note: *p* <.05

ERP Components

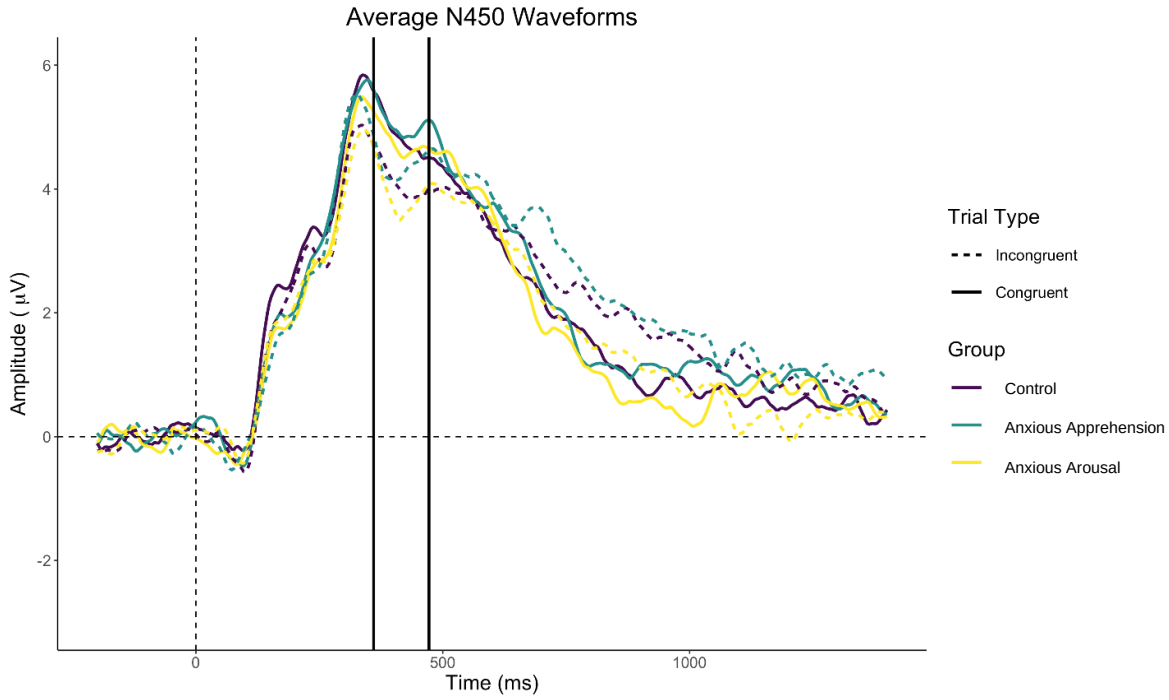
Congruency

In order to evaluate whether the ERP components distinguish between task condition (incongruent/congruent), and to explore potential interactions with group, a series of repeated measure ANOVAs were conducted. Figure 3 presents the grand-average waveforms. A main effect was observed for congruency with increased N200, N450, and conflict SP amplitude observed on incongruent trials compared to congruent trials. A main effect was also found for congruency with shorter N200 latency onset for congruent trials compared to incongruent trials. A main effect was not observed for N450 latency. No significant group by congruency interactions effects were observed.

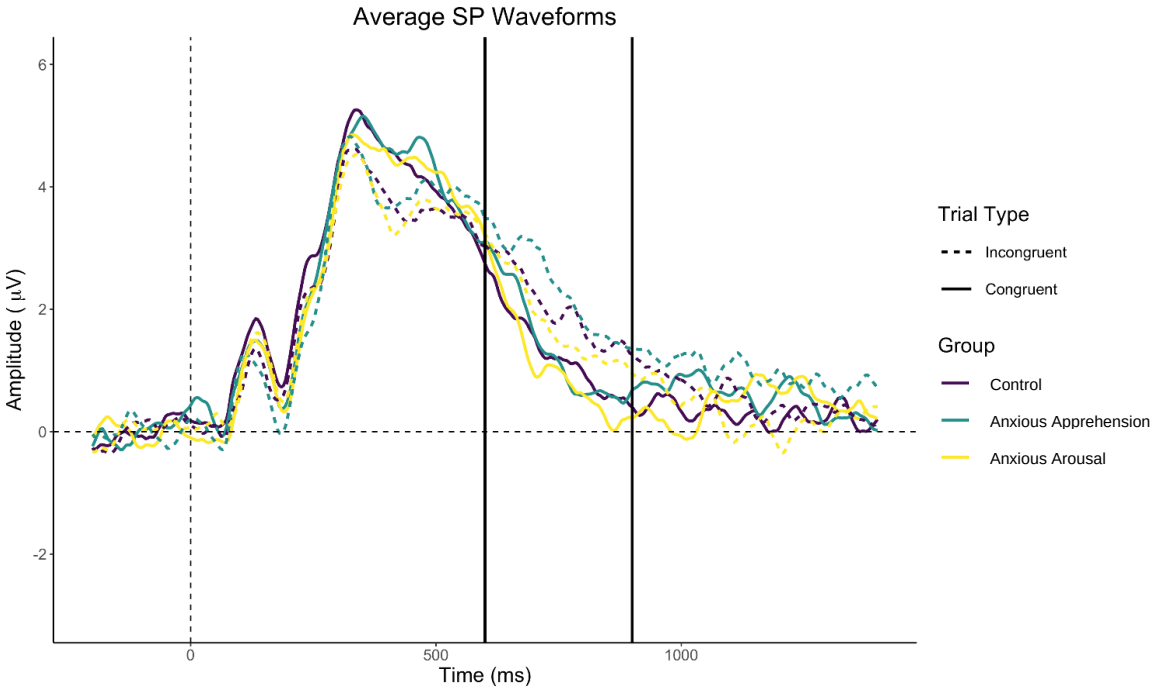
Figure 3. Grand-Average Waveforms for Each Group. Waveforms are highlighting A) N200, B) N450, and C) conflict SP components. Time course is depicted on the x-axis beginning 200ms before stimulus presentation. Stimulus onset = 0ms.



B.


Average N450 Waveforms
Amplitude (μV)
6
4
2
0
-2
0
500
1000
Time (ms)
Trial Type
Incongruent
Congruent
Group
Control
Anxious Apprehension
Anxious Arousal

C.


Average SP Waveforms
Amplitude (μV)
6
4
2
0
-2
0
500
1000
Time (ms)
Trial Type
Incongruent
Congruent
Group
Control
Anxious Apprehension
Anxious Arousal

Evaluation of Categorical Group Differences on the Temporal Course of Response Inhibition

In order to characterize group differences on ERP components, repeated measures ANOVAs were conducted for each ERP component (N200 incongruent/congruent amplitude and centroid latency, N450 incongruent/congruent amplitude and centroid latency, conflict SP incongruent/congruent amplitude). Bonferroni adjusted post-hoc *t*-tests revealed a greater N200 amplitude on incongruent trials for APP participants compared to CG participants ($p < .001$). There were no other significant group differences for ERP components. Table 11 provides statistical results of the group comparisons for ERP components.

Correlations among psychological symptoms, behavioral measures, and ERPs

Prior to conducting dimensional regression analyses, Pearson correlations were computed among the primary study variables to evaluate the relations between psychological symptoms, behavioral measures, and ERP components (Table 12). Key relations are highlighted in the following sections.

Psychological symptoms

Psychological symptoms were generally related to each other (*r*s range .27 – .35), although anxious arousal was not related to positive affectivity or anxious apprehension. Higher levels of anxious arousal were related to attenuated conflict SP amplitude for both congruent and incongruent trials (*r*s = -.22 and -.33, respectively). Higher levels of worry were associated with larger N200 amplitudes for congruent and incongruent trials (*r*s = .21 and .22, respectively). Further, increased anxious apprehension was associated with N200 latency onset for congruent trials ($r = .22$), with later latency associated with increased anxious apprehension. A relation in

the opposite direction was observed with regard to latency for incongruent trials such that increased anxious apprehension was related to earlier latency (r = -.31).

RT, accuracy, and ERP components

RT and accuracy for congruent and incongruent trials were associated with each other ($r$srange from .23 – .61), and slower RT was generally related to increased accuracy. Increased accuracy for congruent trials was associated with increased accuracy for incongruent trials (r = .27). Congruent trial RT was associated with N200 and N450 amplitude for congruent trials (rs = -.25, -.31), such that slower RT was associated with reduced N200 and N450 amplitudes for congruent trials. Lastly, Stroop interference accuracy was associated with slower N450 latency.

Evaluation of the influence of dimensional psychopathology symptoms on the temporal course of response inhibition

A series of linear regressions was conducted to evaluate the dimensional effect of psychopathological symptoms on response inhibition. Outcome variables were behavioral measures and ERP components. Psychopathology symptoms (anxious arousal, anxious apprehension, affectivity) were simultaneously entered as predictors. Significant findings are reviewed below and regression results for all models can be found in Tables 13 - 15.

N200

There were no significant findings observed for N200 amplitude for congruent or incongruent trials. With regard to congruent latency, higher levels of anxious apprehension were related to later N200 latency (B = .14, p = .01). The regression model was not significant (F(4,87) = 2.12, p = .09). For incongruent trials, higher levels of anxious apprehension were related to later N200 latency (B = .20, p < .001) and higher negative affectivity was related to

earlier N200 latency (*B* = -.72, *p* = .03). The regression model was significant (*F*(4, 87) = 3.62, *p* = .01).

N450

There were no significant findings for N450 amplitude for congruent or incongruent trials. With regard to N450 congruent latency, higher negative affectivity was related to earlier N450 latency (*B* = .07, *p* = .03). The regression model was not significant (*F*(4, 87) = 1.84, *p* = .13). With regard to N450 incongruent latency, higher levels of anxious apprehension were related to later N450 latency (*B* = .07, *p* = .04). The regression model was not significant (*F*(4, 87) = 2.01, *p* = .09).

Conflict SP Amplitude

Psychological symptoms did not predict congruent conflict SP amplitude. For incongruent trials, results showed that higher levels of anxious arousal symptoms were related to more negative conflict SP amplitude (*B* = -.09, *p* = .00). The regression model was significant (*F*(4, 87) = 2.91, *p* = .03).

Table 13. Dimensional Regression Summary Statistics for Stroop Interference.

A. Stroop Interference Reaction Time					
	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .02, *F* (4, 87) = .44 *p* = .78
MASQ-AA	-.47	1.65	-.29 (.78)	[-3.74, 2.81]	
MASQ-NA	-3.75	3.80	-.99 (.33)	[-11.30, 3.80]	
MASQ-PA	-.08	1.29	.06 (.95)	[-2.63, 2.49]	

PSWQ	-.72	0.75	-.10 (.92)	[-1.56, 1.41]	

B. Stroop Interference Accuracy

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .01, F (4, 87) = .28 p = .89
MASQ-AA	-.01	.04	-.12 (0.91)	[-.09, .08]	
MASQ-NA	-.01	.10	-.01 (0.92)	[-.21, .19]	
MASQ-PA	-.03	.04	-.10 (0.34)	[-.10, .04]	
PSWQ	.00	.02	.19 (0.85)	[-.04, .04]	

Table 14. Dimensional Regression Summary Statistics for Congruent Trials.

A. Reaction Time for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .02, F (4, 87) = .05 p = .996
MASQ-AA	-.24	1.44	-.17 (.87)	[-3.31, 2.48]	
MASQ-NA	-.43	3.32	-.13 (.90)	[-6.78, 6.51]	
MASQ-PA	-.16	1.12	-.15 (.89)	[-2.36, 2.12]	
PSW	-.12	.65	-.18 (.86)	[-1.75, 1.05]	

B. Accuracy for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .02, F (4, 87) = .41 p = .80
MASQ-AA	.01	.02	.59 (.56)	[-.03, .05]	
MASQ-NA	.03	.04	.79 (.43)	[-.05, .12]	
MASQ-PA	-.00	.02	-.22 (.83)	[-.03, .03]	
PSWQ	-.01	.01	-.76 (.45)	[-.02, .01]	

C. N200 Amplitude for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .06, F (4, 87) = 1.29 p = .28
MASQ-AA	.02	.04	.66 (.51)	[-.05, .09]	
MASQ-NA	.05	.08	.63 (.53)	[-.10, .22]	
MASQ-PA	-.01	.03	-.16 (.87)	[-.06, .05]	
PSWQ	0.03	0.02	1.60 (.11)	[-.01, .06]	

D. N200 Latency for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .09, F (4, 87) = 2.12 p = .09
MASQ-AA	.10	.12	.79 (.46)	[-.15, .34]	
MASQ-NA	-.55	.28	-1.97 (.06)	[-1.11, .01]	
MASQ-PA	.05	.10	.49 (.62)	[-.14, .23]	
PSWQ	.14	.06	**2.25 (.01)***	[.03, .25]	

E. N450 Amplitude for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R^2***
Variable					R^2 = .03, F (4, 87) =.58 p = .68
MASQ-AA	-.04	.04	-1.21 (.23)	[-.12, .03]	
MASQ-NA	-.02	.08	-.23 (.82)	[-.18, .15]	
MASQ-PA	.02	.03	.54 (.59)	[-.04, .07]	
PSWQ-AP	.01	.02	.50 (.65)	[-.03, .04]	

F. N450 Latency for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R^2***
Variable					R^2 = .08, F (4, 87) = 1.84 p = .13
MASQ-AA	.01	.07	.13 (.90)	[-.14, .16]	
MASQ-NA	-.36	.17	**-2.1 (.04)***	[-.70, -.02]	
MASQ-PA	.07	.06	1.16 (.25)	[-.05, .18]	
PSWQ	.06	.03	1.85 (.07)	[-.005, .13]	

G. SP Amplitude for Congruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R^2***
Variable					R^2 = .06, F (4, 87) = 1.40 p = .24
MASQ-AA	-.05	.03	-1.77 (.08)	[-.11, .01]	
MASQ-NA	-.05	.07	-.78 (.44)	[-.18, .08]	
MASQ-PA	-.002	.02	-.08 (.94)	[-.05, .04]	
PSWQ	.01	.01	.995 (.32)	[-.01, .04]	

Table 15. Dimensional Regression for Incongruent Trials.

A. Reaction Time for Incongruent Trials					
	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .02, F (4, 87) = .44 p = .78
MASQ-AA	-.71	2.07	-.34 (.73)	[-4.83, 3.41]	
MASQ-NA	-4.19	4.78	-.88 (.38)	[-13.68, 5.30]	
MASQ-PA	-.24	1.61	-.15 (.88)	[-3.45, 2.97]	
PSWQ	-.19	.94	-.20 (.84)	[-2.06, 1.68]	
B. Accuracy for Incongruent Trials					
	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .01, F (4, 87) = .17 p = .95
MASQ-AA	.01	.04	.23 (0.81)	[-.07, .09]	
MASQ-NA	.03	.09	.36 (0.64)	[-.15, .22]	
MASQ-PA	-.01	.03	-.39 (0.73)	[-.08, .05]	
PSWQ	-.01	.02	-.58 (0.37)	[-.05, .03]	
C. N200 Amplitude for Incongruent Trials					
	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .08, F (4, 87) = 1.88 p = .12

MASQ-AA	.06	.04	1.56 (.12)	[-.02, .13]	
MASQ-NA	.01	.08	.15 (.88)	[-.15, .18]	
MASQ-PA	-.003	.03	-.10 (.92)	[-.06, .05]	
PSWQ	.03	.02	1.86 (.07)	[-.002, .06]	

D. N200 Latency for Incongruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .14, F (4, 87) = 3.62 **p = .01***
MASQ-AA	.10	.14	.70 (.49)	[-.19, .39]	
MASQ-NA	-.72	.33	**-2.20 (.03)***	[-1.38, -.06]	
MASQ-PA	.11	.11	.99 (.33)	[-.11, .33]	
PSWQ	.20	.07	**3.37 (< .001)***	[.09, .35]	

E. N450 Amplitude for Incongruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .05, F (4, 87) = 1.12 p = .35
MASQ-AA	-.06	.03	-1.87 (.07)	[-.12, .004]	
MASQ-NA	.06	.07	.91 (.37)	[-.07, .20]	
MASQ-PA	.00	.02	-.01 (.99)	[-.05, .05]	
PSWQ	.01	.02	.60 (.55)	[-.02, .04]	

F. N450 Latency for Incongruent Trials

	B	***SE***	***t (p)***	**95% CI**	***R²***
Variable					R^2 = .09, F (4, 87) = 2.09 p = .09

MASQ-AA	-.07	.08	-.93 (.36)	[-.26, .08]
MASQ-NA	-.24	.18	-1.36 (.18)	[-.60, .11]
MASQ-PA	.07	.06	1.08 (.28)	[-.06, .19]
PSWQ	.07	.04	**2.11 (.04)***	[.04, .15]

G. SP Amplitude for Incongruent Trials

	B	***SE***	***t (p)***	**95% CI**	R^2
Variable					R^2 = .12, F (4, 87) = 2.91 **p = .03***
MASQ-AA	-.09	.03	**-3.28 (<.001)***	[-.15, -.04]	
MASQ-NA	.02	.06	.34 (.72)	[-.10, .15]	
MASQ-PA	.004	.02	.17 (.86)	[-.04, .05]	
PSWQ-AP	.01	.01	.53 (.68)	[-.02, .03]	

Note: *p <.05

Discussion

The present study was conducted in order to determine whether dimensions of anxiety symptoms (i.e., anxious arousal and anxious apprehension) distinctly modulated the temporal course of neural activity related to response inhibition during a cognitive Color-Word Stroop Task (CWST). ERP results indicated that dimensional psychopathology symptoms uniquely impact the cascade of cognitive resources implemented during response inhibition. Relative to categorical group analyses, results from dimensional analyses with psychopathology symptoms (anxious arousal, anxious apprehension, and affectivity) offered a more nuanced view regarding how different dimensions of anxiety symptoms impact the temporal course of response inhibition, such that anxious apprehension modulated N200 and N450, and anxious arousal influenced the conflict SP. Anxious apprehension was found to impact response inhibition earlier

in the processing stream, which may represent attentional interference and difficulties efficiently monitoring stimuli, while anxious arousal impacted response inhibition later in the processing stream, which may indicate a failure to efficiently sustain effective top-down control due to difficulty regulating sustained inhibitory control.

Categorical group analyses showed that the anxious apprehension group elicited a greater N200 amplitude on incongruent trials compared to the control group, suggesting difficulties with conflict monitoring for those with anxious apprehension. Related to this finding, dimensional analyses showed that higher levels of anxious apprehension were related to later N200 latency for congruent and incongruent trials. Higher negative affectivity (anhedonic depression symptoms) was also related to earlier N200 latency for incongruent trials, indicating opposing effects of anxious apprehension and negative affectivity on early conflict monitoring processes. In sum, during early conflict monitoring processes as indexed by N200, high levels of anxious apprehension were associated with increased amplitude and later latency for incongruent trials which indicates that increased neural resources (likely reflective of ACC generators) were recruited and corresponded with a later engagement with conflict monitoring processes. Of note, although we used a "cognitive" color-word Stroop task in the present study, resolving cognitive conflict may generate state negative affect (Inzlicht et al., 2015), which could instantiate worry and ruminative processes in individuals with anxious apprehension, resulting in attentional interference and difficulties efficiently monitoring stimuli.

Similar to the pattern of results that emerged for N200, only anxious apprehension impacted incongruent N450 latency (and negative affectivity was related to congruent N450 latency). N450 has been associated with conflict resolution and it is theorized to also reflect ACC

activity (Liotti, Woldorff, et al., 2000; MacLeod & MacDonald, 2000; Szűcs & Soltész, 2012). Thus, anxious apprehension was related to later engagement with conflict resolution processes, likely indicating difficulties with quickly selecting responses for incongruent stimuli, which may be related to ruminative tendencies that slow down decision-making processes.

Anxious arousal distinctly attenuated the later conflict SP amplitude for incongruent trials. Conflict SP has been theorized to reflect later response inhibition processes such as response selection and conflict adaptation/resolution processes, indicative of middle or inferior frontal gyrus and left extrastriatal cortical function and is suggestive of a more sustained, top-down control mechanism (Chen et al., 2011; Larson et al., 2011, 2014; Smith, 2017). Reduced conflict SP amplitude for incongruent trials in individuals reporting higher levels of anxious arousal may indicate a failure to sustain efficient top-down inhibitory control processing and poor signaling of conflict resolution (Smith, 2017).

As expected from previous findings (e.g., Harnishfeger, 1995; MacLeod, 1991; Silton et al., 2011; West et al., 2005), the present study showed that the behavioral costs of interference processing were observed through increased difficulty (i.e., increased RT and error rate) and increased recruitment of neural resources (e.g., increased ERP amplitude; delayed N200 centroid latency) for incongruent trials compared to congruent trials across the entire sample. We did not observe a main effect of psychological symptoms on behavioral outcomes in either the categorical or dimensional analyses. Further, it appears that effect size for behavioral outcomes is smaller than our observed effect sizes for our ERP data, indicating that a larger sample would be required to observe relations among psychopathology symptoms and behavioral outcomes. Alternatively, it remains plausible that compensation is occurring in other subcortical brain

regions that are not fully observable via ERP scalp analyses that measure cortical regions located near the surface of the brain.

These findings not only support dissociable brain processes during the temporal course of response inhibition, but also provide additional support for orthogonal anxiety symptoms. Consistent with previous research identifying arousal and anxious apprehension as unique subtypes of anxiety (Engels et al., 2007, 2010; Guha et al., 2020; Heller et al., 1997), anxious apprehension was shown to impact early inhibitory control processes, indicating increased recruitment of neural resources and later engagement with conflict monitoring and conflict resolution processes. Anxious arousal was related to later inhibitory control processes involved in response selection and monitoring of response. Further supporting the theory that anxious arousal and anxious apprehension represent orthogonal symptom dimensions, nearly all psychological symptoms were correlated with each other with the exception of anxious arousal, which was not correlated with either anxious apprehension or positive affectivity.

Limitations and Future Directions

While this study has several strengths, several limitations should be noted. Though the study sample size was confirmed to be appropriately powered for a minimum effect size of R^2 = .16, it may be underpowered to detect behavioral differences. Further, the sample was compromised of undergraduate students largely representing a subclinical sample. It would be useful to replicate this study in a community-based sample with a range of clinical anxiety disorders for generalizability. Chronic pain has been shown to be distinctly associated with anxious arousal and self-reported inhibitory control difficulties (Polnaszek et al., in prep). Understanding the temporal course of response inhibition through experimental measures may be

helpful in discerning the mechanisms by which anxious arousal may affect response inhibition in chronic pain. Treatments targeting this mechanism could be efficacious in ameliorating chronic pain symptoms associated with poor inhibitory control.

Conclusions

In this exploratory study, we evaluated whether anxious arousal symptoms compared to anxious apprehension symptoms differentially influence the implementation of the temporal course of neural activity during response inhibition. Anxious apprehension impacted response inhibition earlier in the processing stream, which may represent attentional interference and difficulties efficiently monitoring stimuli due to ruminative processes (i.e., worry). Relative to healthy controls, individuals with anxious arousal exhibited similar patterns of neural activity earlier in the processing stream; however, response inhibition was impacted later in the processing stream for individuals high in anxious arousal, which may indicate a failure to efficiently sustain effective top-down control due to difficulty regulating sustained inhibitory control. This may be due to difficulty tuning out irrelevant stimuli and maintaining attention to the task at hand. It is possible that those with high anxious arousal may work harder to exert effort early on during response inhibition, but they are unable to sustain this effort throughout each trial. Through studying both categorical and dimensional approaches to probe the relations among distinct dimensions of anxiety symptoms and their relations with behavioral and neural correlates of response inhibition, this study illustrated the importance of dimensional anxiety subtypes in order to advance our understanding of the distinct mechanisms by which they influence response inhibition. Understanding these distinct mechanisms may help inform

treatments that target the unique effects of anxious arousal and anxious apprehension on inhibitory control.

CHAPTER FIVE

DISCUSSION

The present set of studies assessed the relation between neural, psychological, and cognitive mechanisms that have been shown to influence the experience of chronic pain. Specifically, Studies One and Two advanced our understanding of chronic pain by illustrating its unique relationship with anxious arousal and frontal neural activity on inhibitory control difficulties. Study Three focuses on targeting how anxious arousal is distinctly associated with neural correlates of inhibitory control in order to provide a framework that can be applied to individuals experiencing chronic pain. Together, these studies aim to inform the development of evidenced-based interventions that target anxious arousal in order to enhance the effectiveness of chronic pain management.

Study One, "Somatic Symptoms Distinctly Influence Visceral Nociceptive Mechanisms in Primary Dysmenorrhea," advanced our understanding regarding how psychological (somatic symptoms, depression, anxiety) and physiological (EEG resting state activity) mechanisms relate to increased pain perception in women with and without dysmenorrhea. Findings from this study indicated that chronic pain (i.e., dysmenorrhea) and increased somatic symptoms were significant predictors of increased pain perception during an experimentally induced bladder pain task. Depression and anxiety were associated with experimental bladder pain, although neither significantly predicted bladder pain. While PFC resting-state activity was not a significant

predictor of experimental bladder pain, bladder pain was correlated with increased frontal alpha power, specifically the lower-2 alpha band, and anxiety was associated with increased frontal

beta power irrespective of group membership. Frontal alpha and beta activity have been associated with anticipatory processes and attentional demands (particularly expectancy) (Klimesch et al., 1998; Klimesch, 1999; Michels et al., 2008) which have a key role in pain perception and modulation (Moont et al., 2010; Quiton & Greenspan, 2007). Abnormalities in PFC function have also been implicated in somatic symptoms (Hakala et al., 2002) as well as depression and anxiety disorders (Latthe et al., 2006).

Individuals who live with chronic pain report deficits in cognitive control (Shackman et al., 2011), which could be related to frequent and perseverative rumination about pain, or repetitively thinking about the causes and consequences of negative pain-related experiences (Sullivan et al., 2001). The findings from this study indicated that individuals experiencing pain and reporting high levels of somatic symptoms, or somatic arousal, were predictive of perceiving more pain when exposed to additional pain through an experimental pain task. Further, brain activity in the alpha and beta bands were associated with pain and anxiety, though not predictive of increased experimental pain. Thus, understanding the mechanisms by which anxious (i.e., somatic) arousal and PFC activity, particularly in the involvement of cognitive (i.e., inhibitory) control, affect pain perception sensitivity in individuals experiencing chronic pain is essential in reducing the risk for developing increased chronic pain symptoms in the future.

Some of the factors that influence the relation between psychological symptoms, EF, and neural correlates are illustrated by the Study Two titled, “Chronic Pain and Inhibitory Control in

Emerging Adults: The Role of Psychological Symptoms." Study Two utilized a cross-sectional approach to understanding the role psychological symptoms play in the relation between chronic pain and inhibitory control. Individuals experiencing chronic pain may be especially vulnerable to inhibitory control deficits. The study used a sample of emerging adults, as this age range (18 - 27) has been identified as a critical developmental window for executive function optimization, particularly inhibitory control. Co-occurring psychological symptoms may also exacerbate inhibitory control dysfunction. Motivated by findings from Study One which showed that high levels of anxious (i.e., somatic) arousal were related to higher discomfort during an experimental bladder task in individuals with dysmenorrhea, Study Two characterized the role of anxious arousal in an emerging adult population with chronic pain and disentangled the contributions of depression and anxiety symptoms with regard to impairments in inhibitory control. Results showed that chronic pain was associated with higher levels of negative affectivity, worry, and anxious arousal, as well as lower levels of positive affectivity. However, results from our exploratory parallel mediation analysis showed that only negative affectivity, positive affectivity, and anxious arousal mediated the indirect relation between pain group and inhibitory control. Notably, worry, or anxious apprehension, was not a significant mediator, indicating that with regard to anxiety symptoms, anxious arousal may be distinctly related to cognitive outcomes for individuals with chronic pain. Furthermore, Study Two characterized chronic pain prevalence in a large sample of emerging adults, a population that has been relatively understudied in chronic pain research despite risk factors associated with this age group (e.g. complex developmental and social transitions that necessitate the need for good self-management strategies). Thus,

psychosocial interventions that target affectivity and anxious arousal symptoms may improve inhibitory control function in emerging adults with chronic pain.

The third study in this series, "Anxious Arousal and Anxious Apprehension are Distinctly Related to the Temporal Course of Response Inhibition," built upon findings from Study Two, by identifying how distinct dimensions of anxiety symptoms are associated with neural correlates of inhibitory control. The study utilized an experimental cognitive neuroscience measure of response inhibition (The Color Word Stroop Task). The study used both categorical and dimensional approaches to probe the relations among these distinct dimensions of anxiety symptoms and the behavioral and neural correlates of response inhibition. ERP results indicated that dimensional psychopathology symptoms distinctly impact the cascade of cognitive resources implemented during response inhibition. Relative to categorical group analyses (e.g., anxiety subtype groups), results from dimensional analyses with psychopathology symptoms (anxious arousal, anxious apprehension, and affectivity) offered a more nuanced view regarding how different symptoms impact the temporal course of response inhibition. Anxious apprehension impacted response inhibition earlier in the processing stream, which may represent attentional interference and difficulties efficiently monitoring stimuli. Anxious arousal impacted response inhibition later in the processing stream, which may indicate a failure to efficiently sustain effective top-down control due to difficulty regulating sustained inhibitory control. This may be due to difficulty tuning out irrelevant stimuli and maintaining attention to focus on the task at hand. Understanding these distinct mechanisms may help inform treatments that target the unique effects of anxious arousal and anxious apprehension on inhibitory control.

Overall, the findings from this set of studies highlighted the cross-sectional relation between EF, psychological symptoms, and neural mechanisms and specifically distinguished the importance of anxious arousal, inhibitory control, and neural mechanisms measured with EEG (i.e., frontal alpha and beta) and ERP (N200, N450, Conflict SP) methodologies.

Future Directions

Considering the separate dimensions that are encompassed under the term "anxiety", studying the influence of anxious arousal on the neural correlates of inhibitory control helps to clarify how these constructs are related, and the extent to which inhibitory control deficits are a transdiagnostic cognitive symptom that may be present across anxiety types, particularly when considering the role of anxiety in chronic pain per the fear-anxiety-avoidance model. Studies One and Two provide evidence for the distinct relationship between anxious arousal and self-reported and observed (i.e., frontal neural mechanisms) inhibitory control in individuals experiencing chronic pain. Study Three identifies the distinct temporal course of inhibitory control via anxious arousal. Thus, focusing studies that further elucidate this relationship, including a follow-up study of Study Three with individuals experiencing chronic pain, is an imperative next step.

Another future step includes studying interventions that may specifically target anxious arousal in individuals experiencing chronic pain. A promising line of research is emerging with mindfulness-based interventions. Studies have shown that mindfulness may ameliorate pain symptoms by reducing somatic (i.e., anxious) arousal (Berkovich-Ohana et al., 2012; Travis et al., 2010). Mindfulness practices have been hypothesized to reallocate cognitive resources that are often focused on pain perception in individuals with chronic pain. This may allow cognitive

processes that are important for implementing activities of daily living to be utilized more readily (Kerr et al., 2013). However, studies are still elucidating how, and for whom, mindfulness interventions for pain management are most effective (Day et al., 2014). Understanding the nuanced roles of dimensional anxiety symptoms may be helpful in closing the theoretical gap in the literature that is needed to improve empirically-based treatments for chronic pain management (Day et al., 2014). Thus, studies that focus on tailoring mindfulness therapies for chronic pain management that specifically target anxious arousal may lead to developing more effective treatments for individuals with chronic pain.

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