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Effects of One Neurofeedback Session on Relationship between Fear-Of-Pain and Visual Avoidance of Pain

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By

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Avoidance of Pain

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Abstract

Chronic pain is increasingly prevalent and costly and will continue to be with the increasing mean age of America's population. It is important to identify interventions addressing pain-related biopsychosocial aspects. The purpose of the current study was to examine if a single session of specific neurofeedback (NF) protocols had an effect on subjective fear and physiological fear-avoidance behaviors in relation to pain-related stimuli. Correlational analyses revealed that FPQ-III minor pain scores were negatively associated with total fixation duration while looking at pain-related pictures. One-way ANOVAs revealed differences approaching significance for those trained on Left-Hemisphere NF protocols compared to those in Sham training for total fixation duration, moderate effect sizes were found. Statistically significant group differences were found for those trained on Right-Hemisphere protocols compared to those trained on Left-Hemisphere protocols for first fixation durations. Findings support research that implicates NF training as a neuromodulation technique for the subjective pain experience.

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Table of Contents

List of Figures

List of Tables

Introduction

Effects of One Neurofeedback Session on Relationship between Fear-Of-Pain and Visual Avoidance of Pain

Chronic pain is an extremely prevalent condition. It is estimated that 70 million Americans experience some form of acute, recurrent, or chronic pain each year and that 10 percent of the population report the presence of pain at least 100 days a year (Cassidy, Cote, Carroll, & Kristman, 2005; Covington, 2007). Chronic pain has been revealed to affect healthy, college aged students as well. Hastie, Riley, and Fillingim (2005) found a prevalence rate for painful experiences at 50% for their college-aged sample. Hastie et al. (2005) also found that the proportion of participants reporting painful experiences was comparable across three ethnic groups including African American, Hispanic, and Caucasian. Kennedy, Kassab, Gilkey, Linnel, and Morris (2008) found similar results with an annual prevalence rate of 42.8% for lower back pain in college students from a major university in Colorado.

Chronic pain complaints result in millions of physician office visits per year (Hing, Cherry & Woodwell, 2006), and as many as 150 million lost work days (Guo, Tanaka, Halperin, & Cameron, 1999). Chronic pain treatments often involve increasing doses of a variety of medications to gain a measure of relief. Unfortunately, these current pharmacological treatments are moderately effective

at best (Turk, Wilson, & Cahana, 2011), and often carry important side effects (Noble et al., 2007; Van Tulder, Scholten, Koes, & Deyo, 2000; Verdu, Decosterd, Buclin, Stiefel, & Berney, 2008). As a result, multiple treatment alternatives have been proposed. The Purpose of the current study was to explore one of these alternative treatment options, specifically Neurofeedback.

CHAPTER I

Pain

Johannes, Le, Zhou, Johnston, and Dworkin (2010) found a prevalence rate of 30% for chronic pain in a nationally representative sample. Half of those who reported having chronic pain also reported daily pain, and average pain intensity for the past three months as greater than or equal to 7 on a scale from 1 to 10. According to Gaskin and Richard (2012), the total annual cost of pain in 2010 was $560 to $635 billion with additional health care costs due to pain ranging from $261 to $300 billion. Pain also represents a significant loss in productivity in the workforce ranging from $299 to $335 billion annually (Gaskin & Richard, 2012). Chronic pain is an increasingly prevalent and costly condition and this trend will continue upward with the ever-increasing average age of America's population (Fredburger et al., 2009)

Pain has been defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, described in terms of such damage" (Merskey & Bogduk, 1994, *p*. 212). The function of pain, as an aversive experience, can be conceptualized as adaptive and/or protective in nature (Asmundson, Parkerson, & D'Ambrosio, 2015). In other words, the pain

sensation often draws the attention of the individual to potentially damaging stimuli within their environment. From a learning perspective, pain experiences facilitate discrimination and avoidance of any stimulus that may be dangerous to the individual or hinder their ability to heal from previous injury. Therefore, pain perception involves not only physiological components but also cognitive and emotional components.

Pain is often differentiated based on the duration of the experience: as acute or chronic. Acute pain is the sensation that comes from activation of specialized pain receptors (nociceptors) for a limited time and may or may not be associated with significant tissue damage (Dafny, 1997). Loeser and Melzack (1999) described acute pain in relation to the activation of nociceptors at the site of local tissue damage as well. In specific, Authors explain that an injury modifies characteristics of the pain receptors, their connections to the central nervous system, and the autonomic nervous system within that body region to produce an acute pain sensation. Typically, pain report subsides before the organic pathology is eventually resolved. The direct relation to actual tissue damage, and the limited nature of the pain perception and healing process make acute pain inherently different from other types of pain, specifically chronic pain (Dafny, 1997; Loeser & Melzack, 1999).

Pain is characterized as chronic when it is present on the individual for at least three months (Gatchel et al., 2007). Chronic pain is also often linked to an

inability of the organism to adapt or protect from actual or potential damage (Asmundson et al., 2015). Chronic spine pain, especially, has a high impact on the sufferer's everyday functioning, as a range of their activities are often severely limited, leading to difficulties with daily chores, social life, and work (Aronoff, 1991; Faucett & McCarthy, 2003; Nurmikko, Nash, & Wiles, 1998). Persistent or long-term pain can lead to increased irritability and impatience, which could heighten reactivity to daily life stressors (Asmundson et al., 2015). Considering this increased reactivity to stressors, those suffering from chronic pain tend to develop depression-like symptoms: difficulties with concentration, lack of interpersonal interaction, increased fatigue, and isolation (Basbaum, Bautista, Scherrer, & Julius, 2009; Basbaum & Woolf, 1999; Burkey, 2014; Dafny, 1997). Subsequently, the peripheral and central nervous systems attempt adapt to constant state of stress resulting in an anticipation of future pain-related events (Dafny, 1997). Together, these symptoms make it difficult for those suffering from chronic pain to process future pain in a healthy way (Amtmann et al., 2015). In fact, some researchers have found that chronic pain sufferers are at a higher risk for developing psychopathologies (Dersh, Gatchel, Polatin, & Mayer, 2002).

Chapter II

Biological Mechanisms of Pain

Pain perception starts with a physical process that affect specialized nerve fibers, which in turn signal the central nervous system that a painful event has occurred (i.e. nociception; Julius & Basbaum, 2001). Nociception is a group of biological processes in response to noxious stimuli and this can be measured objectively for every individual (Burkey, 2014). For nociception to become painful, there must be awareness of the elicited stimulus (Julius & Basbaum, 2001). Therefore, pain by definition, is affected by not only biological but also psychological processes (Gatchel, 2005). In fact, the pain experience depends on variables within everyone, which leads to pain being subjective in nature (i.e., each person experiences pain in a unique way) (Gatchel et al., 2007). The next sections describe the process of nociception:

Nociception

Peripheral. The process of nociception starts at the receptor site (or the specialized fibers that receive information) usually localized within the peripheral nervous system. The peripheral pathway of pain perception relies heavily on information transmitted from nociceptors, which fall within three distinct groups (Burkey, 2014; Dafny, 1997). The largest group of nociceptors, called C-fibers, conduct slowly due to unmyelinated axons, and typically respond to thermal,

mechanical, or chemical noxious stimuli (Burkey, 2014; Dafny, 1997). The second group of nociceptors, called A-delta fibers, conduct more rapidly because of their myelinated axons (Burkey, 2014; Dafny, 1997). These nociceptors are responsible for fast or sharp pain sensations. The final category of nociceptors, referred to as sleeping or silent nociceptors, typically respond only to noxious stimuli that falls within extreme ranges of intensity (Burkey, 2014; Dafny, 1997). Activation of any of these categories of nociceptors is unpleasant and can produce pain (Burkey, 2014; Dafny, 1997). Continuous or persistent activation of nociceptors could cause sensitization (lowering the threshold for activation), which could allow for normally inoffensive stimuli to provoke noxious sensations (Basbaum et al., 2009; Basbaum & Woolf, 1999).

Central. After activation of peripheral nerve tissue, the afferent nociceptive information enters the spinal cord and then the brain. Afferent spinal pathways include the spinothalamic, spinoparabrachio–amygdaloid and spinoreticulo–thalamic pathways (Dafny, 1997). (See Figure 1. Afferent Spinal Pathways). At the brain level, nociceptive information from the thalamus is projected to the insula, anterior cingulate cortex (ACC), primary somatosensory cortex (S1) and secondary somatosensory cortex (S2), and the Prefrontal Cortex (PFC), whereas information from the amygdala (AMY) is projected to the basal ganglia (BG) (Burkey, 2014). In addition, studies have found a correlation with observed lesions on the parietal lobe, and pain perception. A study by

Greenspan et al. (1999) demonstrated that the posterior parietal area (i.e., parietal operculum) is important for nociceptive input (as measured by evoked potentials, MEG, PET and fMRI) associated with painful stimuli. Each one of these different neural pathways plays an important part in the experience of pain (Burkey, 2014).

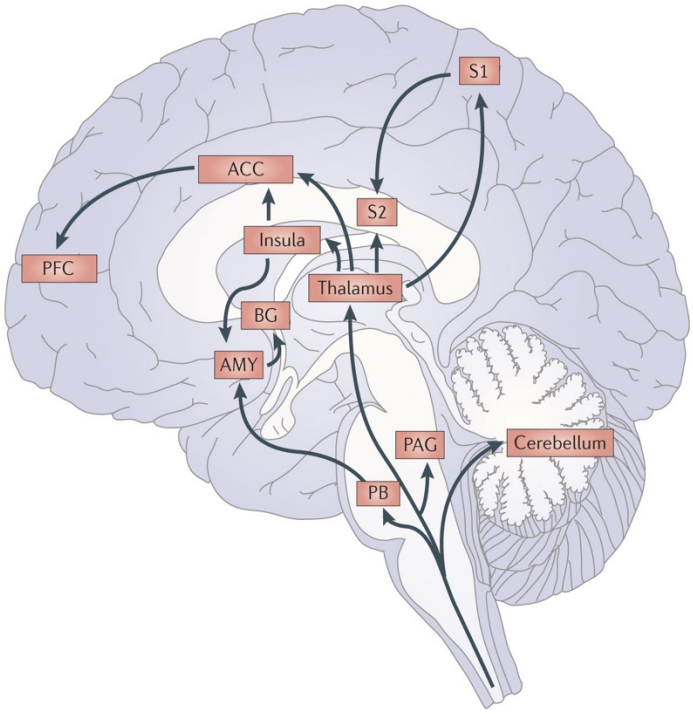

Figure 1. Afferent spinal pathways.

Interpretative studies of these pathways have demonstrated some links between these systems and its functionality. For instance, the somatosensory cortices (S1 and S2) encode sensory information from noxious stimuli, including

the location and duration of pain (Burkey, 2014). On the other hand, the insula and ACC, most often considered part of the limbic system, are crucial for encoding the emotional and motivational (i.e., fight, flight, and freeze) components of a painful experience (Lenz, Casey, Jones, & Willis, 2009).

Interestingly, researchers have revealed that associated pain-brain pathways can be activated without experiencing noxious stimuli (Lenz et al., 2009). For example, studies have shown that simply observing another person in pain may activate some of these pain-associated brain pathways and this is especially the case when the individual is observing a loved one in pain rather than a stranger (Burkey, 2014). This activation of pain-brain pathways without any actual nociception could act as a priming mechanism for the brain, which could lead to an enhanced pain experience (Basbaum et al., 2009; Basbaum & Woolf, 1999)

Overall, pain is a subjective experience that may serve a protective function under specific circumstances. In specific, acute pain sensations bring awareness to injury and possible tissue damage leading to the pursuit of medical attention. However, pain that is persistent or recurrent for three months or more ceases to be protective in nature. Chronic pain has been found to negatively affect activities of daily living and could limit activities such as social life, chores, and work. The process of pain perception begins with the physiological process of nociception via specialized pain receptors. These nociceptors transfer

sensory information up the spinal cord through the peripheral nervous system to the central nervous system and various neural pathways. Moreover, research has shown that these various neural pathways can be activated by non-noxious stimuli, suggesting that some psychological processes may influence pain perception.

Chapter III

Biopsychosocial Model of Pain

Biomedical models of pain assume that recovery from pain/injury occurrs after tissue damage has been resolved (Gatchel et al., 2007). However, many studies have demonstrated that some individuals may have no pain with organic findings (Melzack & Katz, 2001), while other individuals with no organic findings demonstrated disabling pain (Melzack, 1989). These individual differences in the pain perception, recovery, and general pain experience stem from complex interactions of biopsychosocial processes (Gatchel et al., 2007). These complex interactions include a multitude of psychosocial factors that increase or decrease individual recovery time. For instance, pain perception and disability can be influenced by cognitive, emotional, and social factors as well as the ability to cope with any complications related to the injury or recovery from the injury (Hawker et al., 2010; Lerman, Rudich, Brill, Shalev & Shahar 2015; Tran et al., 2015).

Psychosocial factors

Depression, anger, and anxiety have all been found to be interconnected with chronic pain (Amtmann et al., 2015; Kroenke et al., 2011; Sagheer, Khan, & Sharif, 2013). Studies have shown that these psychological emotions have an important role in the pain experience, including, making an individual inclined to

experience pain, be a cause of pain symptoms, modulate the severity of the pain experience (amplify or inhibit), be a consequence of chronic or persistent pain, or perpetuate the pain experience (Asmundson et al., 2015). Numerous studies have found that those experiencing chronic pain are at risk of experiencing higher levels of anxiety and depression as well (Lerman et al., 2015; Sagheer et al., 2013). In fact, chronic pain patients are at greater risk of developing severe psychopathologies (e.g. paranoia, major depressive disorder) (Dersh, Gatchel, Polatin, & Mayer, 2002).

The positive relationship between chronic pain and psychological distress could be related to chronic pain being considered a chronic stressor. The primary basis of stress response in any organism is correcting homeostatic imbalance (Weissman, 1990). In other words, pain could be considered an actual or perceived threat to an organism that disrupts normal functioning, which results in the activation of mechanisms that serve to motivate the restoration of basic functioning to normal levels (i.e. homeostatic imbalance; Weissman, 1990). Chronic pain produces a multitude of events that can facilitate homeostatic imbalance (stress) even after the actual damage has been resolved or in the absence of any organic pathology (Weissman, 1990). This prolonged condition of stress can have strong negative effects on the body and could cause a mutually reinforcing relationship between pain and stress response (Basbaumet al., 2009; Basbaum & Woolf, 1999; Burkey, 2014; Dafny, 1997).

In the brain, for instance, pain related stress can cause the hypothalamus to activate the pituitary gland that secretes adrenocorticotropic hormone, which causes the adrenal cortex to secrete cortisol (Weissman, 1990). Cortisol elevates blood sugar and increases metabolism, which allows for motivation of resources to counteract the perceived or real threat and restore balance to the system (Weissman, 1990). Prolonged activation of this system caused by chronic pain could have serious negative effects on an individual including muscle atrophy, suppression of immune system, alterations of brain structures, and impairment of tissue repair and growth (Weissman, 1990). Concurrently, body system changes could serve as priming mechanisms for the development and/or maintenance of chronic pain, and in turn, alter an individual's cognitions and behaviors creating a negative feedback loop between actual and perceived nociceptive threat (Basbaum et al., 2009; Basbaum & Woolf, 1999).

The relationship between chronic pain and psychological distress is exemplified by studies that have looked at the chronic pain/depression comorbidity. These studies have suggested that 40% - 50% of all chronic pain sufferers also suffer from depressive symptoms, but in most cases, the epidemiological nature of the relationship between chronic pain and depression is still misunderstood (Asmundson et al., 2015). The direction of this relationship is still unclear. Some studies have revealed that depression can cause chronic pain or that chronic pain causes depression, while other studies have shown that

they exist within a mutually reinforcing relationship (Asmundson et al., 2015). In a recent meta-analysis, Burke, Mathias, and Denson (2015) found that depression was the most commonly assessed psychological dimension within chronic pain literature. Researchers also found moderate to very large effect sizes for depression scores of those who had chronic pain. These scores were also consistently statistically significant and negative indicating that those whose suffer from chronic pain also had high levels of depression in comparison to those who did not have chronic pain.

In another study, Kroenke et al. (2011) examined the relationship between chronic pain and depression in a 12-month longitudinal study with 500 primary care patients who had persistent back, hip, or knee pain, and were also enrolled in the Stepped Care for Affective Disorders and Musculoskeletal Pain (SCAMP) study. Half of the participants were diagnosed with comorbid depression and the other half were non-depressed with similar pain reports. Participants with persistent pain and comorbid depression were randomized to a stepped care intervention (n =123) or a treatment as usual condition (n = 127) while non-depressed patients were followed in parallel cohort. Researchers assessed outcome measures at baseline, three, six, and twelve months and used mixed effects model repeated measures (MMRM) multivariable analysis to determine if change in pain severity predicted depression severity and vice versa. Results revealed that change in depression was a strong predictor of pain severity and

change in pain was an equally strong predictor of depression severity. Authors concluded that pain and depression have equally strong effects on each other when assessed longitudinally.

A longitudinal study by Hawker et al. (2010) examined whether osteoarthritis (OA) pain determined depressed mood considering fatigue and disability and found that chronic pain predicted future fatigue and disability, which in turn predicted depressed mood. However, researchers also found that depressed mood and fatigue were so interrelated that each exacerbated the other, and fatigue and disability led to worsening of chronic pain. Depressed mood seemed to indirectly affect severity of chronic pain, which could be said to increase the likelihood of future disability and fatigue. It would seem then, that the comorbidity between chronic pain and depression is a dynamic process that leads to the worsening of functional outcomes for the individual (Hawker et al., 2010)

Chronic Pain and Anxiety

Conceptually, anxiety is a future-oriented emotional state in response to an elusive threat with an unclear source (Leeuw et al., 2006). Anxiety is often associated with preventative and/or hyper-vigilant behavior, which may be adaptive in short-term contexts, but is often counterproductive in the long run (Asmundson et al., 2015; Leeuw et al., 2006). In chronic pain specifically, the threat (pain) is constantly present, which in turn causes a never-ending cycle of

anxiety response and preventative behaviors to avoid pain altogether (Asmundson et al., 2015). The long-term consequences of this cycle could be disability and disuse, which could lower the threshold at which later pain would be experienced. Numerous studies have found high levels of anxiety in chronic pain sufferers, and evidence that it does have an effect of functional outcomes of the individual (Asmundson et al., 2015; Leeuw et al., 2006).

Sagheer et al. (2013) conducted a study to examine the prevalence of anxiety depression in chronic low back pain patients at a tertiary care center. A total of 140 chronic low back pain patients completed demographic questionnaires and The Hospital Anxiety and Depression Scale (HADS). Researchers found abnormal levels of anxiety in 77 (55%) patients and borderline abnormal levels for anxiety in 54 (38.5%) of patients. Authors also found significant gender differences for levels of anxiety within their sample with 20 (14.28%) males and 57 (40.71%) females reporting abnormal levels. Sagheer et al. (2013) concluded that individuals with chronic low back pain were at high risk for anxiety and the risk was significantly higher for females.

Amtmann et al. (2015) examined the mediational effects of anxiety, fatigue, and sleep on the relationship between chronic pain and depression in 1,245 participants with multiple sclerosis (MS). Researchers used cross-sectional self-report symptoms, quality of life data and structural equation modeling to examine the variance in depression stemming from various pain-

related factors (anxiety, sleep, fatigue). Authors discovered an adequately fitting model of the indirect effects of pain on depression, which accounted for approximately 80% of variance in depression. Researchers concluded that higher pain was also associated with greater fatigue, anxiety, and sleep disturbance, which in turn was associated higher levels of depression. Essentially, high levels of chronic pain, indirect effects of that pain (anxiety), and depression were all linked within this study's sample. Considering these findings, anxiety could have acted as a possible mediator between chronic pain and depression.

In another study, Lerman et al. (2015) examined the longitudinal relationship between pain, pain-related disability, and symptoms of anxiety in 428 individuals with chronic pain receiving treatment at a specialty pain clinic. Participants completed questionnaires relating to their pain, state anxiety, and pain-related disability at four time points with a mean of 5 months between each point. Researchers used cross-lagged, structural equation modeling to examine longitudinal associations between the variables. More than half of the sample reported significant symptoms of anxiety (T1-69%, T2-68%, T3-75%, and T4-73%) at each of the four measurement points and half of the sample reported significant symptoms of both anxiety and pain-related disability at each measurement (T1-45%, T2-45%, T3-47%, and T4-48%). Researchers also found that a latent anxiety variable predicted pain and pain related disability, but

neither pain nor pain-related disability predicted anxiety. Authors concluded that in adult chronic pain patients, high levels of anxiety may exacerbate pain and pain-related disability.

Tran et al. (2015) examined whether anxiety and pain catastrophizing were distinct from each other in relation to functional outcomes in pediatric pain, and if they distinctly predict functional outcomes based on age. In a sample of 725 children and adolescents, researchers measured pain characteristics, anxiety, pain catastrophizing, functional disability, and health-related quality of life (HRQOL). Using structural equation modeling, authors found that anxiety and pain catastrophizing were distinct in their sample. Additionally, anxiety predicted HRQOL in children and adolescents, and functional disability in adolescents alone. Based on these findings, fearful personalities could influence the way individuals perceive threat as exemplified by a painful stimulus.

Fear of Pain

Fear is the emotional reaction to an immediate, identifiable threat, such as a dangerous situation or an injury (Leeuw et al., 2006). Like anxiety, fear is an adaptive response that serves to protect from harm by activating the fight or flight response (Leeuw et al., 2006). The three crucial components of fear (perception of a stimulus as threatening, increased arousal, and defensive behavior) are connected and can fluctuate at different rates (Leeuw et al., 2006). The fear response, specifically defensive escape behaviors, may alleviate fear in the short

term, but may also negatively reinforce fear-related behaviors in the future (Leeuw et al., 2006). Abnormally high levels of fear occurring more often may lead to a lowered threshold for defensive escape behaviors, which in turn may lead to a greater chance of pain related anxiety (Leeuw et al., 2006).

In relation to fear, the way perceived threat (i.e. pain stimulus) is interpreted may lead to two distinct outcomes (Vlaeyen & Linton, 2000). When the pain is perceived as non-threatening (low levels of fear), individuals are more likely to maintain regular levels of daily activity, which promotes functional recovery (Vlaeyen & Linton, 2000). This concept was supported by the findings of Vowles and Gross (2003), who found that decreased levels of fear-avoidance beliefs for work was an important factor for improving physical capability and enhanced return to work potential. However, when pain is interpreted as threatening (high levels of fear), typically the case with chronic pain, it could give rise to pain-related defensive escape behaviors such as avoidance (Vlaeyen & Linton, 2000). Asmundson and Norton (1995) supported this relationship by revealing that chronic back pain patients with high anxiety sensitivity reported greater levels of fear of pain and had greater avoidance of activities in comparison to those with lower anxiety sensitivity, despite both groups having equal pain.

Keogh, Ellery, Hunt, and Hannet (2001) examined whether fear of pain would be related to greater selective attentional bias in favor of pain-related

stimuli by exposing participants to varied stimuli in terms of being pain-related, socially threatening, or positive. Researchers found that those with high levels of fear of pain displayed a selective attentional bias towards pain-related stimuli compared to those who had lower levels of fear of pain. Researchers concluded that these results provide evidence for high levels of fear of pain biasing attentional processes, which in turn may make individuals more susceptible to negative experiences with pain. In other words, Keogh et al. (2001) suggested that higher levels of fear of pain can bias one's attention towards pain related information, priming them to react negatively to any further pain-related stimulation.

Peters, Vlaeyen, and Weber (2005) investigated the contribution of physical pathology, pain-related fear, and catastrophizing cognitions to pain intensity and disability in 100 participants with low back pain. Participants completed self-report measures and quantified physical pathology via medical charts using the MEDICS procedure. It was found that pain-related fear accounted for 10% of variance in regression models for pain intensity and disability. This means that fear of pain was an important predictor of pain intensity. Researchers concluded that fear of pain could lead to a preoccupation with pain and a heightened awareness of pain signals, which in turn could lead to increased pain perception.

Woby, Watson, Roach, and Urmston (2004) found similar results. In their study, 83 chronic pain participants completed a series of self-report measures before they participated in a physical therapist led intervention. Regression analysis indicated that fear-avoidance beliefs about work and physical activity were independently associated with levels of disability. In further analyses, researchers found that fear-avoidance beliefs about physical activity were the only statistically significant predictor of participants' levels of disability. These results indicated that those participants who endorsed greater levels of fear-avoidance beliefs specifically about physical activity tended to report greater levels of disability. Therefore, fear of pain is positively associated greater levels of disability (Peters et al., 2005; Woby et al., 2004).

De Gier, Peters, and Vlaeyen (2003) examined the role of pain related fear and attentional processes on tolerance for physical activity in 81 fibromyalgia participants. Researchers had high and low fearful fibromyalgia participants perform a physical task, a cognitive (reaction) task, and a dual task that combined both physical and cognitive components. Results revealed that low fearful participants demonstrated higher activity tolerance for both single and dual conditions. It was also found that high fearful participants responded slower on cognitive reaction time than low fearful participants. Researchers indicated that level of pain-related fear did not significantly affect toleration for physical performance task, but it was trending in that direction. In another more recent

study, Niederstrasser et al. (2015) found that pain-related fear affected physical perception of pain in 82 healthy university students. Researchers exposed participants to an experimental muscle injury protocol designed to induce pain in targeted body regions (left or right arm) and their respective muscle group and asked them to complete self-report fear of pain measures. A day after being exposed to this procedure, participants were asked to rate their pain while they lifted weighted canisters with both the targeted and non-targeted body regions.

Niederstrasser et al. (2015) indicated that the experimental pain protocol was effective at producing pain, which was indicated by increased pain report for the targeted arm during session two. Although the non-targeted arm was unaffected by the pain inducing protocol, pain report for the non-targeted arm increased during the canister lift in session two across all lifts at a significantly greater rate compared to session one. That is to say, that an inherently non-noxious stimulus produced higher levels of verbally reported pain in the non-targeted arm. Researchers postulated that interactions between pain report and levels of pain-related fear, which were only present during session 2, predicted increased pain report. Interestingly, participants with high and low levels of fear reported similar levels of pain during the first session, but only those with higher levels of fear reported increased pain in the non-targeted arm.

Vowles and Gross (2003) also found interesting results in relation to pain-related fear, fear-avoidance behaviors, and changes in physical capability in their

study. In this study, 65 participants with chronic pain complete an interdisciplinary functional restoration program and collected pre- and post-treatment measures of fear-avoidance beliefs (FABQ – Waddell, Newton, Henderson, Somerville, & Main, 1993), short form McGill pain questionnaire (MPQ-SF; Melzack, 1987), and measures of physical ability for work. Results indicated significant decreases in fear-avoidance beliefs, decreases in pain severity, and increased ability for work at post-treatment. A secondary aspect of the study was to examine the validity of measures of fear-avoidance in the prediction of actual physical capability for work. Results revealed that changes in fear-avoidance beliefs for work had a meaningful relation to changes in capability for work. These findings suggest that changing fear-avoidance beliefs for work was an important factor for improving physical capability and enhanced return to work potential.

Based on the above findings, fear of pain could have an important effect on the cognition and behavior of those who are suffering from pain. These results could be due to that fear of pain can bias attentional processes in favor of pain-related stimuli, which could result in greater number of negative experiences with pain (Keogh et al., 2001). Fear of pain could also influence avoidance related behaviors, which could lead to greater disability (Peters et al., 2005, Woby et al., 2004). Furthermore, changes in pain-related fear and fear-avoidance beliefs seemed necessary for increased physical capability and work

potential (Vowles & Gross, 2003). Therefore, it is crucial to identify interventions that can contribute to decreasing this detrimental emotional state in individuals with pain.

Chapter IV

Neurofeedback

QEEG

EEG is the measure of electrical changes within one or more of the cortical regions of the brain via electrodes placed on the scalp (Cannon, 2015; Hammond, 2011; Kaiser, 2007). The Electrodes are placed on specific sites according to an International 10-20 system, which divides the skull into proportional sections in relation to distinguishable landmarks: dent of the nose, protrusion in the back of the head, and preauricular points directly in front of each ear (Cannon, 2015; Hammond, 2011; Kaiser, 2007). For a visual representation of the International 10-20 system, see Figure 2.

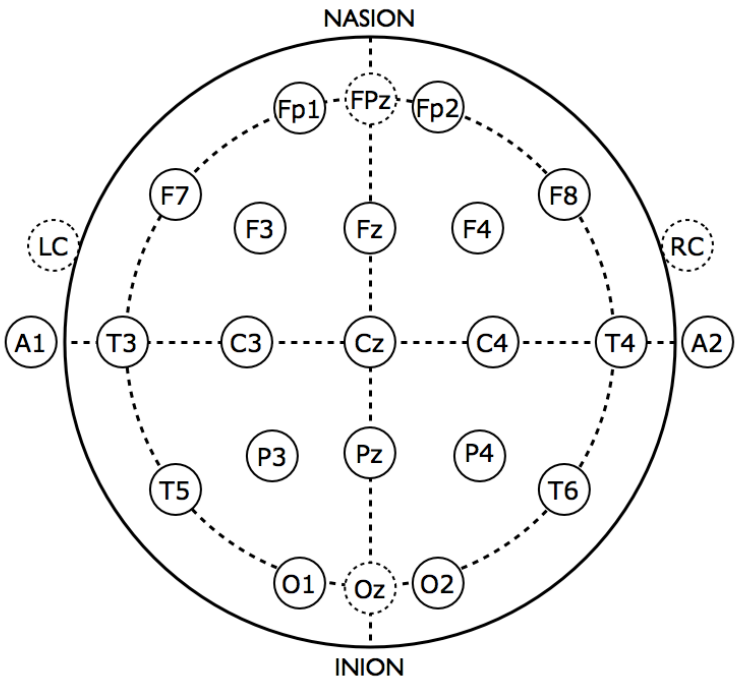

Figure 2. The international 10-20 system.

Each electrode bears a particular label that corresponds to underlying brain areas: F for frontal, FP for frontal pole, P for parietal, C for central, T for temporal, and O for occipital (Cannon, 2015; Hammond, 2011; Kaiser, 2007). Sites are sequenced numerically from the midline (Z), with odd number on the left hemisphere and even numbers on the right hemisphere (Cannon, 2015; Hammond, 2011; Kaiser, 2007). Electrical activity is identified as a difference in potential between two electrodes in a grounded system (Cannon, 2015; Hammond, 2011; Kaiser, 2007). This difference in potential can be measured in reference to another electrode, usually along the midline (Cz) or a linked ear (A1

or A2), this is often referred to as referential recording (Cannon, 2015; Hammond, 2011; Kaiser, 2007). Recordings can also be completed in a bipolar manner where electrodes are paired together with no common reference (i.e., site F4 is linked to C4; C4 to P4, P4 to O2) (Cannon, 2015; Hammond, 2011; Kaiser, 2007).

The electrical activity or rhythm produced by the brain falls within conventional frequency bands measured in cycles per second or hertz (Hz) (Cannon, 2015; Hammond, 2011; Kaiser, 2007). Generally, each frequency band has been associated with a particular mental state, such as, Delta activity (0.5-3.5Hz), associated with deep sleep (Hammond, 2011); Theta activity (4-8 Hz), associated with a very relaxed state, sometimes referred to as the twilight zone between waking and sleep; Alpha activity (8-12 Hz), associated with a relaxed, disengaged state; Sensorimotor rhythm activity (13-15 Hz), associated with a relaxed attentive state; Beta activity (13-30 Hz), associated with intellectual activity and outward focus; and Gamma activity (30+ Hz), associated with intense focus, attention, and with processes that involve multiple brain networks communicating with each other (Cannon, 2015; Hammond, 2011; Kaiser, 2007). It is important to note, that varying degrees of each of these brainwave frequencies have been found to occur simultaneously in different parts of the brain for most individuals (Cannon, 2015; Hammond, 2011; Kaiser, 2007).

In general, dominant brainwave patterns are an indication of awareness level (what is NF) (Cannon, 2015; Hammond, 2011; Kaiser, 2007).

Quantitative EEG (QEEG) is the sophisticated processing of EEG recordings that quantifies the electrical activity gathered from electrodes, which can be used to provide numerical values that represent patterns of activity occurring in the brain (Kaiser, 2007). QEEG takes raw brain activity and uses mathematical processing to quantify and compare that activity to a large normative database of EEG activity (Kaiser, 2007). QEEG produces standard scores for an individual's brain activity based on the activity of others with the same age and gender (Kaiser, 2007). This process allows for comparison of brain activity based on a normative sample (i.e., others that are similar in age and gender) (Kaiser, 2007). It also allows for comparisons of brain activity within the same individual (Kaiser, 2007). A clinician/therapist could complete a QEEG assessment at one point and then again at a later point in order to compare the scores and ascertain the magnitude and direction of any changes in brain activity (Kaiser, 2007).

Neurofeedback Therapy

Neurofeedback Therapy (NFT) is just audio and/or visual feedback based on QEEG or brainwave activity (Cannon, 2015; Hammond, 2011). The process for NFT closely resembles the process for gathering EEG or QEEG data, but with the addition of providing feedback to the individual whose brainwave activity is

being measured (Cannon, 2015; Hammond, 2011). In a typical training session, one or more electrodes are placed on the scalp and the earlobes (Cannon, 2015; Hammond, 2011). The electrodes measure electrical activity occurring at the scalp and send this information to a computer program that records that data and provides instantaneous audio or visual feedback about EEG activity to the individual being trained (Cannon, 2015; Hammond, 2011).

Typically, participants would not be able to willfully influence their brainwave activity because they are not consciously aware of their activity level (Cannon, 2015; Hammond, 2011). However, when participants see or hear a representation of their brainwave activity occurring in real-time, it allows them to gradually change that activity (Cannon, 2015; Hammond, 2011). Conceptually, this closely relates to operant conditioning, where participants recondition and retrain their brainwave activity to match a predetermined criterion (Cannon, 2015; Hammond, 2011). Once this criterion has been met, the participant receives rewards in the form of audio and visual feedback (Cannon, 2015; Hammond, 2011). For instance, in one session, a typical participant may receive anywhere from 300 to 1500 rewards depending on time-frame, participant variables (attention, fatigue level etc.), and criterion difficulty level (Cannon, 2015; Hammond, 2011).

Psychopathologies. Neurofeedback has been utilized in therapy for various disorders with increasing empirical support of its effectiveness compared

to other therapies and placebo. Researchers interested in comparing NFT to medication in the treatment of ADD/ADHD have consistently found NFT to produce comparable, sometimes superior, improvements in attention and concentration compared to taking typical ADD/ADHD medication (Leins et al., 2007; Rossiter & LaVaque, 1995). In one study, Rossiter and LaVaque (1995) sought to examine the efficacy of 20 sessions of EEG biofeedback in reducing ADHD symptoms and to compare the results with psychostimulant medication. Researchers compared an EEG group with a matched (age, IQ, gender, and diagnosis) stimulant group on the Test of Variables of Attention (TOVA) measure, which was administered pre- and post-treatment. Results indicated that both groups improved on TOVA measures of attention, impulsivity, information processing, and variability but did not differ from each other on change scores. Authors indicated that these findings support EEG biofeedback as an effective alternative to stimulant medication for ADHD symptoms.

In another randomized controlled study, Leins et al. (2007) investigated whether neurofeedback treatment lead to an improvement in cognition and behavior in 38 participants with ADHD aged 8-13 years-old. The treatment procedure involved three phases of 10 sessions each of Neurofeedback training using one of two different protocols: positive and negative slow cortical potential shifts (SCPs); suppress Theta (4-8 Hz) while increasing Beta (12-20 Hz). Results revealed that both neurofeedback protocols produced improvements in

cognition and behavior with effect sizes ranging from small (ES =.34) to large (ES= 1.02) and six-month follow-up results that did not differ significantly from end-of-treatment results. Both parents and teachers reported seeing significant improvements as well. These findings support the idea that neurofeedback can be used to make meaningful changes in cognition and behavior in a developmental disability (ADHD) context. In fact, after a meta-analysis conducted in 2009, researchers concluded that NFT had met criteria for being classified as an efficacious and specific treatment for ADD/ADHD (Arns, Heinrich, & Strehl, 2014).

Neurofeedback has been shown to be an effective treatment for psychological distress. For instance, Choobforoushzadeh, Neshat-Doost, Molavi, and Abedi (2015) evaluated the effectiveness of NFT in treating depression and fatigue in 24 participants with multiple sclerosis (MS) in a randomized control study. Participants were randomized into two groups, neurofeedback (16 sessions of NFT) training or treatment as usual and evaluated three times (baseline, end of treatment, and two-month follow-up) throughout the study using the Fatigue Severity Scale and Depression subscale of the Hospital Anxiety and Depression Scale. Using a repeated measures analysis of variance, researchers found that NFT significantly decreased symptoms of depression and fatigue compared to the treatment as usual condition, and effects were maintained at two-month follow-up.

In another study, Rice, Blanchard, and Purcell (1993) investigated the effectiveness of NFT in 45 participants who had generalized anxiety disorder in comparison to a waiting list control group. Participants were randomly assigned to one of four different NFT protocols or a pseudo meditation control condition and evaluated on STAI-Trait Anxiety and psychophysiological symptoms using the Psychosomatic Symptom Checklist. Results revealed that all participants who received NFT treatment showed significant reduction in anxiety and psychophysiological symptoms in comparison to the wait-list, pseudo meditation control group and that decreased self-report of anxiety was maintained at six weeks follow-up.

Pain. Neurofeedback has also been used to treat pain conditions. In a pilot study, Caro and Winter (2011) used NFT in their investigation of how it would affect attention and somatic symptoms within a sample of fibromyalgia syndrome (FMS) patients with attention problems, as indicated via continuous performance test (CPT). Researchers measured pain, fatigue, psychological distress, morning stiffness, and tenderness while having some participants complete 40 or more NFT sessions and having another control group who received standard medical only. Those trained with NFT improved their visual, but not auditory, attention on CPT measures and also showed improvement in tenderness, pain, and fatigue. Although it was not significant, results also revealed a trend toward improved psychological distress and morning stiffness,

but only after forty or more session of NFT. Based on these findings, it seems that NFT could be an effective therapy option for those suffering from chronic pain as seen in patients with FMS.

In another study, Kayıran, Dursun, Dursun, Ermutlu, and Karamürsel (2010) used a randomized, rater blind study to assess the efficacy of NFT in 36 participants with FMS. Researchers randomly divided participants into two groups: one group received twenty sessions of NFT (four weeks) and another received 10 mg per day of escitalopram (control) for eight weeks. All participants received visual analogue scales for pain and fatigue, Hamilton and Beck Depression and Anxiety Inventory Scales, Fibromyalgia Impact Questionnaire and Short Form 36 as outcome measures. Results showed improvements on all measures for both groups, but the NFT group showed greater benefits than controls ($p < .05$). These findings indicate that NFT training had comparable and even superior benefits compared to Selective Serotonin Reuptake Inhibitor (SSRI) treatment, which is typically used in FMS patients to help with mood and fatigue.

Jensen, Grierson, Tracy-Smith, Bacigalupi, and Othmer (2007) conducted a study evaluating the effects of NFT on pain in participants with chronic pain. Researchers sought to determine the average decrease in pain, identify the percentage of pain decreases that were clinically meaningful, and document other benefits of NFT training in a sample of 18 individuals with Complex

Regional Pain Syndrome Type 1 (CRPS-I). Participants were given 0-10 numerical rating scale measures for pain intensity and other symptoms before and after a 30 minute NFT training session. Researchers also performed a series of t-tests to determine the significance of any changes, as well as, computed effect sizes and percent change in order to quantify observed improvements in symptoms. Results revealed substantial and significant decrease in pain intensity with half of the participants reporting changes that were clinically meaningful. Researchers concluded that many patients who receive NFT training report short-term reductions in pain-related symptoms, but long-term effects of NFT training for chronic pain requires further research to evaluate its effectiveness as a treatment option.

Chapter V

Rationale

Chronic pain is an increasingly prevalent and costly condition and this trend will continue upward with the ever-increasing average age of America's population. Individual differences in pain perception, recovery, and general pain experience stem from complex interactions of biopsychosocial processes. The biological aspect of pain perception concludes with the brain analyzing and interpreting pain related information that comes from nociceptors in the peripheral nervous system. This pain related information is interpreted in the brain in the context of the individual's current cognitive and affective state and his or her environmental surroundings.

Pain perception becomes a subjective experience, which is dictated entirely by individual differences within the biopsychosocial processes. Research has shown that chronic pain is strongly associated with psychological components such as: depression, anxiety, stress, and pain-related fear. These psychological aspects of chronic pain interact within the individual to produce greater negative functional outcomes and greater chances of disability. Therefore, it is important to identify interventions that can help reduce the psychological distress that is associated with pain.

Scientific advances in QEEG technology have made it possible for individuals to be aware of their brain activity occurring in real-time. Using Neurofeedback therapy, researchers have found that individuals can change their brainwave activity in a therapeutic way. Neurofeedback has been shown to work in various psychological disorders, as well as with chronic pain and pain related symptoms. Unfortunately, there are no studies that have looked at the effects of Neurofeedback therapy on fear of pain.

Purpose

The purpose of this study was to examine how Neurofeedback may affect an element of pain, specifically fear of pain. In specific, this study sought to examine if a single training session on specific neurofeedback protocols had an effect on avoidant behavior and interfered with the relationship between subjective fear (as measured by scores on the Fear of Pain Questionnaire – III; FPQ – III) and physiological fear-avoidance behaviors in relation to pain-related stimuli (measured via the Tobii X-260 eye-tracker).

Hypotheses

I. Participants in the control group that have high scores on the FPQ-III will exhibit more fear-avoidant behaviors as indicated by less and/or shorter fixations within pain-related area of interest.

II. Individuals trained on neurofeedback targeting Right Hemisphere pain pathways will have decreased fear-avoidant visual behaviors when compared to Left-Hemisphere protocols and Controls.

CHAPTER VI

Methods

Participants

The data for the current study was collected from 121 male and female undergraduate students studying at a Southwestern University in the United States with a final sample of 99 participants. Participants were screened on arrival using a multi-item personal and family history wellness questionnaire that covered injury, traumatic brain injury (TBI), multiple conditions known to cause persisting pain (carpel tunnel syndrome, recurrent migraines, recurrent back pain etc.) prescription drug use, and existing psychiatric and developmental disorders. Recruitment for the study was accomplished through an online recruitment program used by the university, Sona-Systems. The study was approved by the Institutional Review Board (IRB). Participants were excluded if they had a history of seizure or epilepsy (N=3), lack of NF training (N=6), or were missing eye-tracking data (N=13). Final sample was 99 participants.

Materials & Equipment

FPQ-III. Fear of pain was determined using a 30-item self-report measure developed by McNeil and Rainwater, (1998). The FPQ-III contains short sentences describing painful experiences that participants rate on a five-point (1-5) Likert-type scale. Participants were asked to rate the degree of fear they

anticipate experiencing related to the painful event. Higher scores indicate greater fear and lower scoreindicate less fear of pain. The measure consists of ten-item subscales including fears of severe pain, minor pain, and medical pain.

PCS. The Pain Catastrophization Scale (PCS) measures the level of catastrophic thinking in relation to pain experience with catastrophizing defined as "an exaggerated negative mental set brought to bear during actual or anticipated painful experience" (Sullivan et al., 1995; Sullivan et al., 2001). Pain Catastrophization as measured by the PCS consists of three main factors. These factors are threat magnification, rumination, and learned helplessness. The Cronbach alpha index for the total PCS was .93 when measured using a sample of 288 college students at a Midwestern university (Osman et al., 1997). Researchers measuring reliability across of the magnification subscale on the PCS across three studies showed an average of Cronbach's alpha .74 for females, and .71 for males (Osman et al., 1997).

ASRS v1.1. The Adult ADHD Self-Report Scale Symptom Checklist Version 1.1 (ASRS v1.1) was used to assess for ADHD symptomology. The ASRS v1.1. consists of eighteen items assessing the ADHD DSM-IV-TR criteria. Critical items are the core of the ASRS v1.1 Screener. The ASRS v1.1 Screener also twelve supplemental questions that align with ADHD symptomatology. The ASRS v1.1 is a 5 point Likert Scale ranging from Never to Very Often. The ASRS has been shown to be valid and reliable within the adolescent and adult

populations. Specifically, Adler et al. (2006) found a high internal consistency between the items with a Cronbach a of 0.93 at time 1 and 0.94 at time 2 of the study. Within the adult population, the ASRS was also found to have high internal consistency for patient and rater-administered versions of the scale (Cronbach's a 0.88 for patient self-report and 0.89 for rater-administered).

Neurofeedback. EEG NFT training was carried out using the using the BrainAvatar software and a 19-channel signal amplifier, Discovery 24E from Brain Master Technologies, Inc. Brain Avatar is a single platform that allows for patient assessment and training to be blended with EEG (Proler & Bass, 2012). EEG data was recorded from four electrodes placed (F3, F4, C3, P4) on the skull (10/20 system), with ground and reference electrodes placed on the earlobe. Neurofeedback training is achieved by displaying participant's real-time brain activity to teach self-regulation of brain function. Brain activity is presented to the participants in the form of a visual and auditory stimulus. For this study, this feedback was quantified to determine capacity to train (i.e., *Beeps Total*).

Tobii X-260 eye-tracker. Eye tracking was performed using the Tobii X-260 eye-tracker (see http://www.tobii.se/). The Tobii X-260 eye-tacker allowed for the mapping of eye movements to various features on the screen during task performance. It is a state of the art eye-tracking device that tracks the eye-movement of participants in real time and gives data on maintenance of gaze on a single location. Areas of interest (AOIs) were generated by a JavaScript

application. This application provided the screen coordinates for each element that was of interest for a given image (i.e., pain-related stimuli vs. non-pain-related stimuli; please see appendix A for examples of these stimuli with outlined area of interest). For the purpose of this study, fear-avoidant visual behavior was defined as the difference between pain-related and non-pain-related stimuli on eye-tracking measures. Relevant Eye tracking variables examined were *Time to First Fixation (TFF), First Fixation Duration (FFD), Total Fixation Duration (TFD), Fixation Count (FC),* and *Visit Count (VC)*.

***Eye-Tracking variables Operational Definitions*.**

- *TFF* is length of time in milliseconds until the participants eye fixates on a particular point within an AOI;
- *FFD* is defined as the length of time in milliseconds that a fixation lasts;
- *TFD* is defined as the sum of the duration for all fixations within an AOI;
- *FC* is the quantified as the number of fixations (i.e. maintaining of the visual gaze on a single location for 50-600 ms) within an AOI;
- *VC* is defined as the number of visits (i.e. time interval between first fixation on active AOI and end of last fixation within same AOI) within an AOI.

Procedure

Consent, screening and subject preparation. Upon arrival, participants were informed about the nature of the study and given the informed consent. If they accepted participation, they completed prescreening questionnaires. Subjects that reported an injury, drug abuse, any psychiatric disorder, current pain, history of chronic pain, or seizures/epilepsy were asked to stop the experiment without any penalties. Those participants that passed the prescreening then completed FPQ-III, PCS, and the ASRS v1.1. After completion of questionnaires, participants' skull was prepared for Neurofeedback training.

Training. Participants were randomly assigned to one of three Neurofeedback groups, 1) the Right Hemisphere training protocol group, 2) the Left Hemisphere training protocol, and 3) the Sham condition. The Right Hemisphere protocol consisted of neurofeedback training on locations F4 (GO 12-15Hz; STOP 20Hz and up) and P4 (GO 4-12Hz; STOP 15Hz and up). The Left Hemisphere protocol consisted of neurofeedback training on locations F3 (GO 15-20Hz; STOP 4-7Hz and 20Hz and up) and C3 (GO 4-12Hz; STOP 15Hz and up). The Sham protocol consisted of no direct training; instead participants watched a pre-recorded "sham" training session. Feedback was achieved by showing the participant their own brain activity. Such information was presented to the participants in the form of auditory and visual feedback (beeps).

Regardless of the condition, each participant trained for 30 minutes and received continuous auditory and visual reinforcement.

Pain-relevant visualization task. After training occurred, participants completed the non-pain/pain visualization tasks on the Tobii eye-tracker for a total of 5 minutes. The task was combination of looking at images, either non-pain-relevant or pain-relevant, and answering questions about those images shortly afterwards. Participants were shown an initial set of 32 non-pain-relevant images, each image shown for second. Participants were then shown another group of 32 pain-relevant images followed by a series of ten questions about details either seen or unseen in the images. This task was designed to measure fear-avoidant behavior (eye movement) in relation to pain-related stimuli.

CHAPTER VII

Results

Descriptive statistics for the full sample are presented. The total number of participants in the final sample was n = 99. The final sample M_{age} = 19.34 years (SD = 2.04); with a minimum age of 18 and a maximum age of 33. The sample was primarily Caucasian (58.6%, n = 58) followed by Hispanic (24.2%, n = 24), African-American (14.1%, n = 14), and Asian/Other (3%, n = 3). In terms of school classification, the sample was mostly Freshmen (58.6%, n = 58) and Sophomores (19.2%, n = 19) followed by Juniors (12.1%, n = 12), and Senior/Other (10.1%, n = 10). For gender, the sample had a majority of female participants (68.7%, n = 68) followed by male participants (31.3%, n = 31). Randomly assigned participants were distributed between the three experimental conditions in relatively equal fashion with a slight majority being in the sham condition (39.4%, n = 39) followed by left hemisphere condition (29.3%, n = 29), and right hemisphere condition (31.3%, n = 31). Table 1 presents a summary of demographic variables divided by participant condition.

Table 1

Descriptive statistics for final sample divided by participant condition (N = 99)

	Sham n (%)	Left Hemisphere n (%)	Right Hemisphere n (%)	*Total* *n (%)*
Gender				
Female	27 (27.3)	20 (20.2)	21 (21.2)	*68 (68.7)*
Male	12 (12.0)	9 (9.0)	10 (10.0)	*31 (31.3)*
Classification				
Freshmen	23 (23.2)	16 (16.2)	19 (19.2)	*58 (58.6)*
Sophomore	11 (11.1)	4 (4.0)	4 (4.0)	*19 (19.2)*
Junior	1 (1.0)	6 (6.1)	5 (5.1)	*12 (12.1)*
Senior	3 (3.0)	3 (3.0)	3 (3.0)	*9 (9.1)*
Other	1 (1.0)	-- --	-- --	*1 (1.0)*
Ethnicity				
Caucasian	22 (22.2)	18 (18.2)	18 (18.2)	*58 (58.6)*
Hispanic	13 (13.1)	3 (3.0)	8 (8.1)	*24 (24.2)*
African-American	3 (3.0)	7 (7.1)	4 (4.0)	*14 (14.1)*
Asian	1 (1.0)	-- --	-- --	*1 (1.0)*
Other	-- --	1 (1.0)	1 (1.0)	*2 (2.0)*

Note: % = percentage of final sample; n = frequency within sample; Sham = sham NF training condition; Left Hemisphere = left hemisphere NF training condition; Right Hemisphere = right hemisphere NF training Condition.

Table 2 presents the frequency distribution for other possible confounds that occurred in the sample. Frequencies are separated by variable as well as by condition. Across all conditions, 19 participants indicated experiencing a concussion at some point in their life with a slight minority (n = 3) occurring in the right hemisphere condition. In terms of chronic pain (pain lasting longer and three months), 7 participants indicated that they had experienced chronic pain in their lifetime with a majority (n = 5) occurring in the left hemisphere condition. Sixteen participants indicated that they had experienced recurrent back pain with relatively equal distribution across conditions. For psychiatric diagnoses, 12 participants reported having a diagnosis with a slight majority (n = 6) occurring in the sham condition. Eight participants reported having a developmental diagnosis equally distributed across conditions. In terms of taking psychiatric drugs during their lifetime, 13 participants reported doing so with a slight majority (n = 6) occurring in the sham condition.

Table 2

Frequency of Other Possible Confounds (N = 99)

	Sham Training n (%)	Left Hemisphere Training n (%)	Right Hemisphere Training n (%)	*All Groups n (%)*
Concussion				
No	31 (31.3)	21 (21.2)	28 (28.3)	*80 (80.8)*
Yes	8 (8.1)	8 (8.1)	3 (3.0)	*19 (19.2)*
Chronic Pain				
No	38 (38.4)	24 (24.2)	30 (30.3)	*92 (92.9)*
Yes	1 (1.0)	5 (5.1)	1 (1.0)	*7 (7.1)*
Recurrent Back Pain				
No	33 (33.3)	23 (23.2)	27 (27.3)	*83 (83.8)*
Yes	6 (6.1)	6 (6.1)	4 (4.0)	*16 (16.2)*
Psychiatric Diagnosis				
No	33 (33.3)	27 (27.3)	27 (27.3)	*87 (87.9)*
Yes	6 (6.1)	2 (2.0)	4 (4.0)	*12 (12.1)*
Developmental Diagnosis				
No	36 (36.4)	26 (26.3)	29 (29.3)	*91 (91.9)*
Yes	3 (3.0)	3 (3.0)	2 (2.0)	*8 (8.1)*
Taking Psychiatric Drugs				
No	33 (33.3)	26 (26.3)	27 (27.3)	*86 (86.9)*
Yes	6 (6.1)	3 (3.0)	4 (4.0)	*13 (13.1)*

Note: % = percentage of final sample; n = frequency within sample; Sham = sham NF training condition; Left Hemisphere = left hemisphere NF training condition; Right Hemisphere = right hemisphere NF training Condition.

A Chi-square analysis was conducted to determine if ethnicity, classification, or gender differed across conditions. Results indicated that conditions were very similar in all above demographics ($X^2(2) < 9.836$, $p > .28$). An ANOVA was conducted to determine if age, training, ADHD reported symptoms, Pain Catastrophization scores, fear of pain scores (total, minor, severe, and medical), or pain sensitivity were significantly different between the groups. Table 3 shows no significant differences between groups in any demographic, training, ADHD or fear of pain variables.

Table 3

Summary of ANOVA for Differences in Demographic Variables per Condition

Variable	Sham Training		Left Hemisphere Training		Right Hemisphere Training			
	M	(SD)	M	(SD)	M	(SD)	F	*p*
Age	19.28	(2.97)	19.17	(1.34)	19.29	(2.12)	.082	*.922*
Beeps Total	-	-	1346.97	(154.15)	1332.00	(137.56)	.260*	*.612*
ADHD Critical	8.46	(3.97)	8.90	(3.03)	8.87	(3.82)	.156	*.855*
ADHD Total	28.26	(12.13)	28.72	(9.54)	28.61	(11.18)	.017	*.983*
PCS Total	12.26	(8.48)	14.62	(10.68)	11.71	(9.35)	.812	*.447*
Fear-of-Pain: Total	84.51	(21.34)	82.90	(22.39)	82.68	(24.10)	.070	*.932*
Fear-of-Pain: Minor	21.87	(8.80)	21.66	(8.38)	20.90	(8.24)	.118	*.889*
Fear-of-Pain: Severe	34.49	(8.44)	34.59	(9.12)	35.16	(8.88)	.056	*.945*
Fear-of-Pain: Medical	28.13	(8.59)	26.69	(8.78)	26.71	(10.02)	.290	*.749*

Note: * T-test was conducted to determine differences in training rewards (Beeps Total) between the left hemisphere training condition and the right hemisphere training condition. Age = mean age of participants in each condition; Beeps Total = mean cumulative rewards (beeps) received for participants in each condition; ADHD = Attention Deficit Hyperactivity Disorder; PCS = Pain Catastrophization Scale; Fear-of-Pain = total and subscale scores on Fear-of-Pain Questionnaire 3; Sham = sham NF training condition; Left Hemisphere = left hemisphere NF training condition; Right Hemisphere = right hemisphere NF training Condition.

Table 4 displays correlations between potential covariates including ADHD variables, PCS, FPQ-III scores (i.e., total scores, minor pain scores, severe pain scores, and medical pain scores) and fear-avoidant eye behavior (e.g. TFF = Time to first fixation; FFD = First fixation duration; TFD = Total fixation duration; FC = Fixation count; VC = Visit count) during the pain and no-pain visual stimulus for participants in the sham (control) condition only. This analysis was conducted with the sham condition only because it was assumed that participants in this group had zero-level of neurofeedback training. Results of the Pearson correlation revealed the following: There were no significant correlations between FFD and any other eye-tracking variables or other covariates. Time to first fixation was significantly correlated with both FC and VC. Fixation count was significantly correlated with VC and TFD. Total fixation duration was the only eye-tracking variable that significantly correlated with other covariates, specifically the FPQ-III subscale minor pain. No other eye-tracking variables were significantly correlated with any other covariates (i.e., fear-of-pain, PCS, or ADHD).

FPQ-III total scores were significantly correlated with all minor subscales including minor pain, severe pain, and medical pain. Scores on the medical pain subscale were also significantly correlated with minor pain subscale and severe pain subscale score. In other covariates, PCS scores significantly correlated with FPQ-III total score, minor pain subscale, severe pain subscale, and medical pain

subscale as well as ADHD-related scores including critical scores, and total scores. ADHD total scores significantly correlated with minor pain subscale scores, and ADHD critical scores.

Table 4

Correlation Matrix for Eye-tracking Variables, Fear of Pain Variables, and Other Covariates in the Sham (Control) Minus Those Excluded from Group Analysis (N = 38)

		2	3	4	5	6	7	8	9	10	11	12
Eye-tracking												
1	TFF	.23	.24	-.28*	-.44**	.09	.09	.06	.08	.09	.05	.05
2	FFD	-	-.03	-.13	-.12	-.04	.01	-.05	-.05	.07	-.06	-.06
3	TFD		-	.45**	.06	-.05	-.24*	.03	.07	.06	.03	.11
4	FC			-	.55**	-.15	-.16	-.12	-.11	-.02	-.03	-.03
5	VC				-	-.06	-.17	.04	-.04	-.09	.00	.01
FPQ-III												
6	Total					-	.80**	.88**	.88**	.37**	.07	.17
7	Minor						-	.53**	.54**	.32**	.17	.23*
8	Severe							-	.70**	.27**	.04	.09
9	Medical								-	.35**	.01	.14
PCS												
10	Total									-	.24*	.33**
ADHD												
11	Critical										-	.86**
12	Total											-

Note: TFF = Time to first fixation; FFD = First fixation duration; TFD = Total fixation duration; FC = Fixation count; VC = Visit count. All eye-tracking variable scores are the estimated differences between pain and no-pain images (e.g., TFF pain - TFF no-pain = TFF). Fear of pain = scores on FPQ-III: Total = total score; Minor = score on minor pain subscale; Severe = score on severe pain subscale; Medical = score on medical pain subscale. PCS = Pain Catastrophization Scale: Total = total score. ADHD = scores on ASRS v1.1: Critical = sum of critical item scores; Total = total. ** = $p < 0.01$ (2-tailed); * = $p < 0.05$ (2-tailed).

A number of one-way ANOVAs were conducted to determine the effect of training conditions (sham, left hemisphere training, and right hemisphere training) on fear-avoidant eye behavior. Fear-avoidant eye behavior, again, was defined as the difference between pain-related and non-pain-related stimuli (e.g., fixation count on pain-related stimuli – fixation count on non-pain-related stimuli = fixation count difference). Results of the ANOVA indicated significant differences for training conditions on FFD difference and marginally significant differences on total fixation duration difference. Post hoc comparisons using the Tukey HSD test indicated that the mean first fixation duration difference for the right hemisphere training condition was significantly greater than the left hemisphere training condition. However, the sham condition did not significantly differ from right hemisphere or left hemisphere training conditions (see Table 5).

Table 5

Group Analysis of Variance for Effect Training Conditions on Fear-avoidant Eye Behavior Differences

	Sham	Left Hemisphere Training	Right Hemisphere Training			Cohen's d	
	N = 39	N = 29	N = 31				
Fear-Avoidant Eye Behavior	M (SD)	M (SD)	M (SD)	F	*P*	Left	Right
TFF	0.13 (1.04)	0.16 (3.71)	-0.12 (3.92)	0.05	*.944*	.01	-.09
FFD	0.04 (0.28)	-0.05 (0.18)	0.13 (0.30)	3.37	*.039*	-.37	.31
TFD	6.57 (9.55)	11.80 (4.36)	9.28 (7.96)	3.07	*.052*	.67	.30
FC	12.28 (27.07)	18.82 (26.85)	11.64 (29.06)	0.60	*.549*	.24	-.05
VC	-3.00 (8.39)	-2.78 (9.15)	-5.79 (7.36)	1.17	*.312*	.03	-.35

Note: Cohen's d = Left and right hemisphere conditions compared to sham condition; All eye-tracking variables represent differences in those measures on pain vs non-pain related stimuli (e.g., TFFpain – TFFnon-pain = TFF); Sham = sham NF training condition; Left Hemisphere = left hemisphere NF training condition; Right Hemisphere = right hemisphere NF training Condition.

CHAPTER IV

Discussion

The purpose of this study was to determine if one session of neurofeedback (NF) could affect a subjective pain experience and visual behavior. In specific, this study looked to determine if 30 minutes of hemisphere-specific NF training significantly decreased fear-of-pain related avoidant behavior measured via eye-tracking.

Relationship between Fear of Pain and Eye-Tracking Variables

This study found a significant negative correlation between an FPQ-III subscale (i.e., minor pain), and the eye-tracking variable total fixation duration when participants were looking at pain-related pictures. This fear-of-pain and eye-tracking correlation finding, suggests that selective attentional biases towards pain-related stimuli (Keogh et al. 2001) can be measured via eye-tracking (Bannerman, Milders, & Sahraie, 2010a, 2010b). This finding is supported by Asmundson and Norton (1995) who found that chronic pain patients reporting greater levels of fear-of-pain also displayed greater avoidance than those with lower levels of fear, even after controlling for perceived level of pain. This finding is also supported by De Gier et al. (2003) who found that participants with high pain-related fear differed significantly from their low fear counterparts by demonstrating slower reaction times on a cognitive task.

However, eye-tracking in this sample, did not correlate with report of fear of medical injuries. Considering that our sample is composed of healthy young adults, it is possible that participants in the current sample have not been exposed to medical conditions that lead to severe pain, and thus, they are less avoidant of such images. This conclusion is supported by early fear acquisition models which suggest that previous experiences are crucial for establishing and maintaining future fear-related behavior (Mineka & Cook, 1986; Muris, Steerneman, Merckelbach, & Meesters, 1996; Rachman, 1977; Rachman, 1991).

NF training effects on Fixation Duration and Time to First Fixation

Contrary to what we expected, group analyses revealed that those individuals who received Left-Hemisphere NF training had longer Total Fixation Duration within pain-related areas of interest than those who received Sham training, although results are at the level of approaching statistical significance (p<=.052). Effect size for the comparison Left Hemisphere NF training and Sham were considerate moderate (d = .67) suggesting possible effects of NF on fear-avoidance eye-behavior.

The current study found significant group differences on first fixation duration when looking at pain-related pictures. Specifically, those trained on Right Hemisphere NF training protocols held their first visual gaze on a single location longer than those trained on Left Hemisphere NF training protocols. Interpretation of this finding is difficult given that this eye-tracking measure did

not significantly correlate with any measure on the FPQ-III or the ADHD-related measure. It is possible that this finding may reflect an unaccounted-for variable within the study such as novelty (i.e., new pain-related features in the second set of images), curiosity (i.e., differences in the second set of images lead to increased fixations), or other visual perception factors. However, this is still not clear, and future studies are needed to test these possibilities.

Interpretation of Results

Our findings seem to support studies that have suggested NF training as a neuromodulation technique for reducing the fear of pain in clinical settings. In specific, Jensen et al. (2007) found clinically meaningful decreases in pain intensity at primary pain sites for 18 spinal cord injury participants. Researchers found statistically significant improvement on measures of psychological well-being. Despite the lack of connection between some fear-of-pain, ADHD-related symptoms, and eye-tracking behavior, it is possible that NF training protocols, specifically right hemisphere specific protocols influenced attention-related brain networks in such a way that resulted in longer first fixation durations within pain-related stimuli (Gevensleben et. al, 2009a; Gevensleben et. al, 2009b).

Studies using similar to our NF training protocols, have found evidence of significant improvements in attention-related behavior measures in individuals diagnosed with ADHD have (Gevensleben et. al, 2009a; Gevensleben et. al, 2009b). Specifically, Gevensleben et. al (2009a) found that the improvement for

combined NF training group were superior to the placebo group with moderate effect sizes (ES = .60). Effects sizes for NF training on attention-related processes, such as the approach explained above, have been reportedly comparable to effect sizes reported for pharmacological treatments (methylphenidate; ES NF = 0.81 vs. ES methylphenidate = 0.84). These findings seem to indicate that NF training protocols targeting specific attention-related neural networks result in significant improvements in attentional processes.

Limitations and Future Studies

Eye-tracking behavior as measured in the current study failed to significantly correlate with overall fear-of-pain scores, ADHD-related scores, and scores on the PCS. As such, there is potentially no true relationship between eye-tracking measures and behavioral outcome measures in the current study. With this initial assumption of relationship not being met, it is difficult to make definitive conclusions about the meaning of performance on eye-tracking measures in relation to the manipulated variable (i.e., NF training). Future studies should aim to establish connections between measures of eye-tracking (e.g., first fixation duration, total fixation duration, and fixation count) and other behavioral outcome measures before testing assumed effects on eye-tracking measures. These established relationships could serve as the foundations of future predictions about eye-tracking and behavior in response to manipulation of independent variables.

The current study made use of a convenient sample of healthy college students which could result in generalizability issues (i.e., any conclusions made are in reference to sample itself and not greater population), under representation of sociodemographic differences (misrepresentation of ethnic minorities), and introduce modest amounts of variability within groups resulting in unpredictable results and inconsistent findings (Bornstein, Jager, & Putnick, 2013). This modest variability could have translated to increased variability within conditions in the current study resulting in inconsistencies in statistical significance and effect sizes (Bornstein et al., 2013). Future studies could aim to decrease within group variability by focusing on a specific target population such as those individuals suffering from persistent chronic pain or any other unique population.

In collecting data for the current study, many participants (n = 22) were excluded for various reasons including issues with technology, poor effort, and/or history of mental or physical health concerns. Future studies should aim to refine data collection procedures using technology such as eye-tracking software to decrease the likelihood of lost participant data due to technological difficulties.

Finally, it is typical for participants to be exposed to multiple NF sessions. In the current study, participants were exposed to just one training session which may have translated into smaller effect sizes overall. Future studies should examine the cost and benefits of exposing participants to multiple sessions of NF

training as a way of ensuring that the appropriate level of effect is achieved in order to compare to other similar NF training studies.

Conclusion

In the current study, it was found that FPQ-III subscale (minor pain) scores were negatively correlated with total fixation duration when participants were looking at pain-related pictures suggesting that higher levels of fear of minor pain may be associated with attentional biases towards pain-related stimuli. However, other eye-tracking variables measured in the current study showed no significant correlations with FPQ-III total scores and medical or severe pain subscale scores. Previous research indicates that these suggested attentional biases may be influenced by prior experience which could explain findings in the current study.

Analyses revealed group differences approaching significance for the individuals who received Left-Hemisphere NF training compared to those who received Sham training for total fixation duration. Although not significant, effect sizes for this for comparison were considered moderate. There were significant group differences found between those trained on Right-Hemisphere NF protocols compared to those who received Left-Hemisphere NF training on first fixation duration. However, this finding is difficult to interpret given that first fixation duration did not significantly correlate with any FPQ-III measures, ADHD-related measure, or the PCS measure. Overall, findings from the current study

provide some support for previous research implicating NF training as a neuromodulation technique for affecting the subjective pain experience.

Neurofeedback could potentially be useful to decrease the psychological outcome often related to chronic pain. Validation of this beneficial therapeutic approach for chronic pain and related symptoms requires further scrutiny. Specific variables related to its effectiveness including behavioral correlates with pain-related fear should be examined more closely to determine their relationship to the experience of pain and how specific NF training protocols may affect them.

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Appendix A

Pain-Related Stimuli with Area-Of-Interest

Non-Pain-Related Stimuli with Area-of-Interest

Vita

After completing high school at Evadale High School in Evadale, Texas Timothy went on to study Psychology at Stephen F. Austin State University in Nacogdoches, Texas. He completed his Bachelors of Science in Psychology as well as a minor in Rehabilitation Services in May 2014. Timothy then returned to Stephen F. Austin State University in August of 2014 where he received his Master of Arts in School Psychology on May 2018.

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