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LOMA LINDA UNIVERSITY
School of Behavioral Health
in conjunction with the
Faculty of Graduate Studies

Neurofeedback Training for Attentional Processing in Anxious Individuals

by

Caleb Benjamin Barcnas

A Dissertation submitted in partial satisfaction of
the requirements for the degree
Doctor of Philosophy in Clinical Psychology

June 2022

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Each person whose signature appears below certifies that this dissertation in his/her opinion is adequate, in scope and quality, as a dissertation for the degree Doctor of Philosophy.

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ABBREVIATIONS

CVD	Cardiovascular Disease
ANS	Autonomic Nervous System
HPA	Hypothalamic-Pituitary-Adrenal
SNS	Sympathetic Nervous System
PNS	Parasympathetic Nervous System
GAD	Generalized Anxiety Disorder
OCD	Obsessive Compulsive Disorder
PTSD	Post-Traumatic Stress Disorder
CBT	Cognitive Behavioral Therapy
ABM	Attention Bias Modification
AB	Attention Bias
WMT	Working Memory Training
EEG	Electroencephalogram
ADHD	Attention-Deficit/Hyperactivity Disorder
fMRI	Functional Magnetic Resonance Imaging
CPT	Continuous Performance Test
FRCQ	Full Scale Response Control Quotient
GWBS	General Well-Being Schedule
CSUSB	California State University San Bernardino
IVA-2	Integrated Visual and Auditory-Version 2
FAQ	Full Scale Attention Quotient
AAQ	Auditory Attention Quotient

VAQ	Visual Attention Quotient
ARCQ	Auditory Response Control Quotient
VRCQ	Visual Response Control Quotient
ANOVA	Analysis of Variance

ABSTRACT OF THE DISSERTATION

Neurofeedback Training for Attentional Processing in Anxious Individuals

by

Caleb Benjamin Barcenas

Doctor of Philosophy, Graduate Program in Psychology
Loma Linda University, October 2020
Dr. Grace J. Lee, Chairperson

The current study assessed the effectiveness of neurofeedback training for reducing anxiety symptoms and improving attention and response control in adults with self-reported anxiety. This paper presents a review of an archival database of a sample of individuals with reported attention concerns who received 20 to 40 neurofeedback training sessions at a university outpatient clinic. Participants were administered and completed the Integrated Visual and Auditory – Version 2 (IVA-2) Continuous Performance Test (CPT) and the General Well-Being Schedule (GWBS) before and after the intervention. Findings showed that participants significantly improved their scores on the Full Scale Attention Quotient (FAQ) and Full Scale Response Control Quotient (FRCQ) after completing 40 sessions of neurofeedback training. As a group, the 14 participants who completed 40 sessions of neurofeedback significantly increased their FAQ scores from a mean of 76.98 (Mildly to Moderately Impaired) at baseline to a mean of 93.66 (Average). As a group, the 14 participants who completed 40 sessions of neurofeedback significantly increased their FRCQ scores from a mean of 78.92 (Mildly to Moderately Impaired) at baseline to a mean of 93 (Average). There were no statistically significant changes in either overall GWBS scores nor anxiety subscale scores from baseline to post-intervention.

Keywords: neurofeedback; anxiety; attention; attentional processing, IVA-2; CPT;

GWBS

CHAPTER ONE

INTRODUCTION

Epidemiology of Anxiety and Related Symptoms

Despite being frequently underdiagnosed in primary care (Wittchen et al., 2002), anxiety disorders are the most prevalent psychiatric disorders (Chisholm et al., 2016; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012; Wittchen et al., 2011) with a current global prevalence rate of 7.3% (Stein, Scott, Jonge, & Kessler, 2017). Anxiety disorders are characterized by excessive fear and anxiety which lead to significant behavioral disturbance (American Psychiatric Association, 2013). An estimated 28.8% of adults in the U.S. experience an anxiety disorder at some time in their lives (Kessler et al., 2005). While anxiety disorders are often comorbid with a variety of other psychiatric conditions, research has demonstrated that anxiety disorders very often precede the onset of other psychiatric disorders (Kessler, Ruscio, Shear, & Wittchen, 2010) and that anxiety symptoms may be a predictor of worse outcomes (e.g., suicidality) in major affective disorders (Fawcett, 2013).

In addition to having poorer overall quality of life (Olatunji, Cisler, & Tolin, 2007), high levels of anxiety throughout life has a negative impact on physical health. Anxiety disorders are associated with significant individual impairment (Leon, Portera, & Weissman, 1995) and can often times be more impairing than physical disorders (Suliman, Stein, Myer, Williams, & Seedat, 2010). In 2010, anxiety disorders were the sixth leading cause of disability in terms of years of life lived with a disability (Baxter, Vos, Scott, Ferrari, & Whiteford, 2014). Anxiety disorders increase the risk of

cardiovascular disease (CVD) three- to fourfold (Härter, Conway, & Merikangas, 2003; Vogelzangs et al., 2010) and risk of cardiac mortality twofold (Janszky, Ahnve, Lundberg, & Hemmingsson, 2010; Roest, Martens, de Jonge, & Denollet, 2010; Shibeshi, Young-Xu, & Blatt, 2007). Similarly, anxiety disorders are associated with significant reductions in heart rate variability (Chalmers, Quintana, Abbott, & Kemp, 2014) which is an important marker of psychological well-being, general cardiovascular health, and is a major predictor of mortality (Dekker et al., 2000; Kemp & Quintana, 2013; Thayer, Yamamoto, & Brosschot, 2010). Further, individuals who experience chronically high levels of anxiety are at increased risk for several diseases including autoimmune and neurodegenerative diseases (Li et al., 2008; Roy-Byrne et al., 2008; Spitzer et al., 2009). Research has also demonstrated that anxiety is an independent risk factor for major cardiac events and mortality in individuals with coronary heart disease (Martens et al., 2010; Roest et al., 2010; Smith & Blumenthal, 2011) and there is also emerging evidence of associations of anxiety and stroke and diabetes (Scott, 2014).

Anxiety disorders are also among the costliest medical conditions worldwide, having substantial direct and indirect economic costs (Greenberg et al., 1999; Hoffman, Dukes, & Wittchen, 2008; Wittchen et al., 2011). In 1990, the total cost of anxiety disorders in the United States was estimated to be approximately \$42.3 billion (Greenberg et al., 1999). A more recent study revealed that the estimated cost of anxiety disorders in Europe in 2010 was approximately €74 billion [\$98 billion] (Gustavsson et al., 2011).

Mechanism of Anxiety and Related Symptoms

Emotions represent the subjective experiences that arise in response to events that are appraised to be of importance to an individual (Frijda, 1988). The emotions elicited by environmental events have been characterized as organismic responses which serve as an efficient mechanism to rapidly mobilize and organize disparate response systems to deal with demands or threats from the environment (Levenson, 1988). From an evolutionary standpoint, the ability to perceive and respond to threats from the environment is fundamental to survival. The innate threat perception and response systems comprise a complex yet extremely well-coordinated network that extends across both central and peripheral bodily systems (Stein & Nesse, 2011; Woody & Szechtman, 2011).

According to the neurovisceral integration model (Thayer & Lane, 2000), the coordination of threat detection and response is carried out by a complex network of neural structures that enable humans to adaptively respond to environmental, physiological, cognitive, behavioral, and emotional influences. In a properly functioning system, both the level of vigilance (threat perception system) and the subsequent biological and behavioral responses are commensurate to the level of threat from the environment. At the highest levels of perceived environmental danger, both threat-related vigilance and response systems are biologically costly and can only safely be maintained for brief periods of time. Thus, both must be tightly regulated and appropriately calibrated to the environment (Blanchard, Griebel, Pobbe, & Blanchard, 2011).

The current conceptualization of the etiology of anxiety disorders includes an interaction between psychosocial factors and a genetic vulnerability, which manifests in

both neurobiological and neuropsychological dysfunctions (Bandelow, Michaelis, & Wedekind, 2017). However, despite the differences between anxiety disorders, it is understood that anxious individuals in general, overestimate the danger of perceived environmental threats, and this cognitive appraisal is associated with a state of arousal and autonomic activation to prepare the individual for a “flight or fight” reaction (O’Donovan, Slavich, Epel, & Neylan, 2013).

Research on threat-related information processing has suggested that anxious individuals have a heightened sensitivity to threat. Not only do anxious individuals detect threat stimuli more quickly than non-anxious individuals (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & Van Ijzendoorn, 2007; El Khoury-Malhame et al., 2011), but they also are more likely to appraise ambiguous and threatening stimuli as more threatening than they really are (Boddez et al., 2012; Britton, Lissek, Grillon, Norcross, & Pine, 2011; Dash & Davey, 2012).

Research has implicated specific neural circuitry, including the medial prefrontal cortex and amygdala in the detection of and response to threat-related stimuli in the environment (Davis, Walker, Miles, & Grillon, 2010). The amygdala is particularly important for threat-related information processing (Bishop, 2008; LeDoux, 2000) and plays a critical role in determining the extent to which stimuli are perceived as safe or dangerous (Tottenham & Sheridan, 2010). In the context of anxiety disorders, amygdalar responses have been found to be exaggerated (Stein & Nesse, 2011) and greater amygdala response to threat is positively correlated with the severity of anxiety symptoms (Fredrikson & Furmark, 2006; Phan, Fitzgerald, Nathan, & Tancer, 2006). In contrast, the prefrontal cortex plays a key role in the down-regulation of threat-related

responses once threats have passed (Milad & Quirk, 2002; Milad, Rauch, Pitman, & Quirk, 2006) and may be specifically crucial for modulating attention bias to threat (Bishop, 2007; Monk et al., 2008). Neuroimaging studies have demonstrated that the coordinated interaction between these two specific brain regions is negatively impacted in both clinical and non-clinical populations with high levels of anxiety (Bishop, 2007; Indovina, Robbins, Núñez-Elizalde, Dunn, & Bishop, 2011).

In addition to the heightened sensitivity to threat seen in highly anxious individuals, research has also demonstrated that the brain regions involved in processing threatening information can activate biological stress-response systems such as the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis (Dickerson & Kemeny, 2004; Mendes, Blascovich, Hunter, Lickel, & Jost, 2007). These same regions implicated in the triggering of stress-response systems are also responsible for regulating them.

Threat-related brain activity has been found to upregulate the sympathetic nervous system (SNS; Mendes, Major, McCoy, & Blascovich, 2008) while at the same time downregulating the parasympathetic nervous system (PNS; Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012; Thayer & Sternberg, 2009). A healthy ANS is characterized by high levels of adaptive variability (Friedman, 2007) which is regulated by a complex network of brain regions that coordinate autonomic, endocrine, and behavioral responses in adaption to changes in the environment (Thayer & Lane, 2000). Anxiety compromises the integrity of this network as sympathoexcitatory responses are unable to be inhibited, thus leaving the body in a state of prolonged physiological arousal (Newman & Llera, 2011). Similarly, this network can also be compromised by worry and hypervigilance,

which are features observed in all anxiety disorders (Bar-Haim et al., 2007). In turn, hyperactivity in autonomic responses can increase the expression of anxiety symptoms and interoception of body signals (Garfinkel, Eccles, & Critchley, 2015).

Anxiety: Cognitive Consequences

There have been several theories developed to try and explain the effects of anxiety on cognitive performance. From a broad standpoint, anxiety has been theorized to impair attention via its effects on attentional processing. The coordination of attentional processing has generally been distinguished by two attentional systems which have been defined as a goal-directed system and a stimulus-driven system (Corbetta & Shulman, 2002). The goal-directed system is involved in the top-down control of attention and is influenced by expectations, knowledge, and current goals. The stimulus-driven system is involved in bottom-up control of attention and is used during the detection of behaviorally relevant sensory events particularly when they are salient and unattended (Corbetta & Shulman). In the broadest sense of task performance, the efficiency of task completion relies on the ability of the goal-directed attentional system to override the stimulus-based attentional system (Corbetta & Shulman).

According to attentional control theory (Eysenck et al., 2007), anxiety disrupts the balance between the two attentional systems by reducing the influence of the goal-directed processes through biasing increased stimulus-driven processes (Dusek, Mergler, & Kermis, 1976; Markowitz, 1969; Shapiro & Lim, 1989; Williams, Tonymon, & Andersen, 1990). Specifically, attentional control theory posits that anxiety impairs the central executive (Derakshan & Eysenck, 2009; Eysenck, Derakshan, Santos, & Calvo, 2007) which is a component of working memory (Baddeley, 1986). More specifically, it

is theorized that anxiety impairs the central executive via its effects on one of its key functions, attentional control (Eysenck & Calvo, 1992). Attentional control has been conceptualized as the abilities to focus attention (i.e., maintain attentional engagement in the face of distractions) and to shift attention (i.e., execution of attentional disengagement in order to redirect attention away from distractions or toward new tasks; Derryberry & Reed, 2002; Miyake et al., 2000). The effects of anxiety on attentional control have largely been investigated through the perspective of task performance.

Task performance has generally been broken down into performance effectiveness and processing efficiency. Task effectiveness refers to the quality of performance or the ability to complete a task whereas efficiency is based on the relationship between effectiveness and the amount of effort and resources allocated to the achievement of said performance (Eysenck & Calvo, 1992). In most research studies, accuracy (i.e., number of errors committed) is regarded as the primary measure of performance effectiveness. In contrast, processing efficiency is assessed by measuring the amount of time and effort spent achieving a given level of performance. Although literature indicates that anxiety can impact performance effectiveness, the majority of research suggests there is a greater impact on processing efficiency (Derakshan & Eysenck, 2009). Further, research has demonstrated that the negative effects of anxiety on attentional control are observed in both visual and verbal information processing (Amir et al., 2005; Becker, Rinck, Margraf, & Roth, 2001; Derakshan, Ansari, Hansard, Shoker, & Eysenck, 2009; MacLeod, Mathews, & Tata, 1986; Spector, Pecknold, & Libman, 2003).

More in-depth assessment on task performance has involved analyzing inhibition and task switching as they have been identified as two functions of the central executive that are involved in both lower level functions and higher level functions such as goal-directed behavior (Miyake et al., 2000). Both central executive functions are crucial in completing tasks and as such, the level of performance on any given task is explicitly tied to the functioning of the central executive. Unfortunately, the functioning of the central executive is sensitive to an individual's mental state and research has demonstrated that high levels of anxiety negatively impact the central executive (Derakshan & Eysenck, 2009) as highly anxious individuals perform more poorly on tasks requiring executive control (Bishop, 2009). This may be due in part to recent findings that have confirmed the assumption (Eysenck & Derakshan, 1998) that anxiety is associated with reduced working memory capacity (Qi et al., 2014). The association between anxiety and attentional control is also evidenced in non-clinical populations with state anxiety (Richey, Keough, & Schmidt, 2012; Spada, Georgiou, & Wells, 2010) and trait anxiety (Derryberry & Reed, 2002; Judah, Grant, Mills, & Lechner, 2014).

Inhibition consists of two highly intercorrelated components which include prepotent response inhibition and resistance to distractor interference (Friedman & Miyake, 2004). Research has demonstrated that anxiety impairs the efficiency of prepotent response inhibition (Derakshan, Ansari, et al., 2009) and that anxious individuals are significantly more susceptible to distracting stimuli than non-anxious individuals (Calvo & Eysenck, 1996; Eysenck, 1992; Eysenck & Graydon, 1989). Further, the adverse effects of distractors on performance of anxious individuals are even greater when the distracting stimuli are threat-related rather than neutral (Egloff & Hock,

2001; Eysenck & Byrne, 1992). As previously mentioned, the tendency for anxious individuals to regard both ambiguous and threatening stimuli as more threatening than they really are (Boddez et al., 2012; Britton et al., 2011; Dash & Davey, 2012) increases the likelihood that their performance will be negatively impacted by any type of distracting stimuli. Neuroimaging studies have also demonstrated that there is a weaker recruitment of prefrontal control mechanisms in response to threat-related distractors in highly anxious clinical and non-clinical populations (Bishop, Duncan, Brett, & Lawrence, 2004; Bishop, Jenkins, & Lawrence, 2007; Shin et al., 2001).

Beyond compromised inhibitory processes exhibited by anxious individuals, research has demonstrated that there is a tendency for highly anxious individuals to exhibit attentional biases toward threat-related information (MacLeod et al., 1986), a tendency which is also seen in people with high trait anxiety who are not classified as clinically anxious individuals (Derryberry & Reed, 2002). Further, this threat-related attentional bias has also been implicated in the development and maintenance of psychiatric disorders including generalized anxiety disorder (GAD), obsessive compulsive disorder (OCD), post-traumatic stress disorder (PTSD), social anxiety disorder, panic disorder, and simple phobia (Daggleish et al., 2003; MacLeod & McLaughlin, 1995; Mathews, May, Mogg, & Eysenck, 1990). A meta-analytic review indicated that the magnitude of the attentional bias towards threat-related information was not significantly different between the disorders (Bar-Haim et al., 2007).

Another central executive task involved in attentional control is a process known as task switching which involves the performance of two tasks in rapid succession (Miyake et al., 2000). Task switching is associated with costs (e.g., increased errors

and/or reaction times) immediately following the switch (Monsell, 2003). Highly anxious individuals are significantly slower in a task-switching paradigm than in a single task control system (Derakshan, Smyth, & Eysenck, 2009) and the effects are compounded as task complexity increases (Derakshan, Smyth, et al., 2009). Further, highly anxious individuals are slower to disengage their attention from threatening stimuli (Derryberry & Reed, 2002; Lonigan & Vasey, 2009; Peers & Lawrence, 2009). The negative effects of anxiety are seen in decrements in both task switching efficiency (Goodwin & Sher, 1992; Orem, Petrac, & Bedwell, 2008; Wilson, Vine, & Wood, 2009) and effectiveness (Caselli, Reiman, Hentz, Osborne, & Alexander, 2004; Goodwin & Sher, 1992; Wilson et al., 2009).

Treatment

Medication and cognitive behavioral therapy (CBT) have been shown to be effective in the treatment of all anxiety disorders (Bandelow et al., 2017; Bandelow, Reitt, et al., 2015; Olatunji, Cisler, & Deacon, 2010). Comparisons between the efficacy of medication, psychotherapy, and combined treatment vary in terms of level of improvement but are nonetheless associated with high pre-post effect sizes (Bandelow et al., 2017; Bandelow, Reitt, et al., 2015; Crits-Christoph et al., 2011). While recent results indicate that medications are associated with significantly higher average pre-post effect size than psychotherapies (Bandelow et al., 2017), pharmacological interventions do have several drawbacks including adverse side effects (Bandelow, Lichte, Rudolf, Wiltink, & Beutel, 2015; Bandelow et al., 2012) as well as relevant drug interactions (Muscatello, Spina, Bandelow, & Baldwin, 2012) that must be closely monitored.

Although both medications and CBT have been shown to be effective during the short-term, analyses of long-term treatment effects (i.e., greater than 12 months post-treatment) have received less attention. In an analysis of eight randomized controlled trials of CBT for anxiety disorders, 48% of patients were still symptomatic after 2-14 years post-treatment (Durham et al., 2005). Relapse prevention studies examining long-term treatment effects (24-52 weeks) after discontinuation of medications found approximately 40% of patients had relapsed (Davidson et al., 2008; Katzman et al., 2011; Montgomery, Nil, Dürr-Pal, Loft, & Boulenger, 2005; Stein, Versiani, Hair, & Kumar, 2002; Stocchi et al., 2003).

While there is evidence supporting efficacy of other forms of psychotherapy such as short-term psychodynamic therapy, they are recommended as a second line of treatment due to the few number of controlled studies and generally less effective when compared to CBT (Bandelow, Lichte, Rudolf, Wiltink, & Beutel, 2014).

Cognitive Training

Despite the wide variety of treatments for anxiety disorders, the prevalence rates have remained largely stable for decades (De Graaf, Ten Have, Van Gool, & Van Dorsselaer, 2012). One of the reasons for this continuing trend in prevalence rates is the high relapse rates (Bruce et al., 2005; Bystritsky, 2006). As previously discussed, anxiety has been shown to have deleterious effects on both the visual and verbal information processing involved in attentional control and the central executive. Thus, it may be possible that the cognitive deficits seen in anxious individuals could be an underlying factor that medication and therapy are not specifically targeting. One intervention that is specifically aimed at targeting these cognitive deficits is known as cognitive training.

It is widely accepted that the brain is an adaptable organ that is capable of change across the lifespan (Kramer, Bherer, Colcombe, Dong, & Greenough, 2004) and that certain activities (e.g., cognitive training) are capable of improving a variety of cognitive processes in a range of medical conditions and populations (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010; Dunning, Holmes, & Gathercole, 2013) with gains that are sustained for one year (Dunning et al., 2013).

Researchers have recently begun investigating the effectiveness of computer based attentional control training interventions. This approach builds on the main assumptions of attention control theory (Derakshan & Eysenck, 2009; Eysenck et al., 2007) in that attentional control is impaired in highly anxious individuals. Researchers have posited that improving attentional control in anxious individuals may help reduce symptoms of anxiety (Berggren & Derakshan, 2013).

Attention bias modification (ABM) is one computer-based treatment that aims to reduce anxiety by reducing attention bias (AB) towards threat (MacLeod & Mathews, 2012; MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). Interestingly, different forms of CBT have been found to reduce threat-related cognitive-behavioral biases (Antoni et al., 2012; Smits, Julian, Rosenfield, & Powers, 2012). Despite encouraging results from initial studies, the overall efficacy of ABM is relatively weak (Mogg & Bradley, 2016) with some studies indicating it reduces both AB and anxiety, while others finding no effect on either outcome (Cristea, Kok, & Cuijpers, 2015).

Researchers have recently begun studying the effects of working memory training (WMT) to specifically improve attentional control in individuals with anxiety. Although there has been controversy over the duration of maintained working memory

improvements after WMT (Melby-Lervåg & Hulme, 2013) and whether or not improvements from WMT can be seen in areas outside the trained domain (i.e., far transfer effects; Melby-Lervåg, Redick, & Hulme, 2016) there is some positive findings in literature.

Recent findings indicated that working memory training improved attentional control in individuals with anxiety and the level of training-related improvement was associated with reductions in levels of trait anxiety (Sari, Koster, Pourtois, & Derakshan, 2016). Similarly, researchers in more recent study found that training-related improvements were associated with gains in working memory and worry symptoms in high worriers (Hotton, Derakshan, & Fox, 2018). In another study, researchers examining the effects of WMT and CBT in children with elevated anxiety found that both interventions were equally effective at increasing inhibitory control, reducing attentional biases to threat, and reducing anxiety symptoms (Hadwin & Richards, 2016).

Neurofeedback

Neurofeedback therapy, or electroencephalogram (EEG) biofeedback is a specific type of biofeedback that is a method based on operant learning mechanisms (Sherlin et al., 2011) which is thought to normalize deviant brain activity. Neurofeedback involves a noninvasive technique for measuring an individual's brain activity and translating the activity into signals that are fed back to the user in real time (Enriquez-Geppert, Huster, & Herrmann, 2017). The neurobiological abnormalities caused by anxiety can be seen in brain scans and functional differences that manifest in EEG changes (Mathersul, Williams, Hopkinson, & Kemp, 2008; McEwen, Eiland, Hunter, & Miller, 2012). Through this feedback, a user can learn how to self-regulate their own brain activity to

directly alter the underlying neural mechanism of cognition and behavior. This type of intervention can be appealing to individuals without a formal diagnosis as many other types of pharmacological and nonpharmacological treatments are often costly and require individuals to have formal diagnoses to receive financial assistance.

The first use of neurofeedback (utilizing voluntary control of EEG) for clinical applications began in the 1970s with the investigations of its use in epilepsy (Serman & Friar, 1972) and attention-deficit/hyperactivity disorder (ADHD; Lubar & Shouse, 1976). Since its early implementation, the benefits of neurofeedback have been observed in a variety of medical conditions including reducing seizure frequency (Tan et al., 2009), functional recovery after stroke (Rayegani et al., 2014), chronic insomnia (Hoedlmoser et al., 2008), and major depressive disorder (Choi et al., 2011). There is also some support for its effectiveness with anxiety disorders (Moore, 2000). A more recent study found that neurofeedback training in a PTSD population showed decreased self-rated anxiety (Walker, 2009). Similarly, results from our lab have shown that neurofeedback training is an effective treatment for improving attentional and response control in military veterans with PTSD, and that these improvements in attentional capacities are associated with significant improvements in overall wellbeing (McReynolds, Bell, & Lincourt, 2017). Other studies that have focused on the application of neurofeedback in a variety of non-clinical contexts have indicated that neurofeedback is an effective treatment for reducing anxiety (Hardt & Kamiya, 1978; Rice & Blanchard, 1982). A more recently developed type of neurofeedback using functional magnetic resonance imaging (fMRI) technique was found to have a facilitating effect on anxiety regulation in individuals with spider

phobia (Zilverstand, Sorger, Sarkheil, & Goebel, 2015) and in individuals with obsessive compulsive disorder (Scheinost et al., 2013).

The Current Study

It is apparent that anxiety disorders have substantial costs at the individual level and at the societal level (Olatunji et al., 2007; Wittchen et al., 2011). While anxiety can have negative effects at the physiological level, research indicates that anxiety compromises various neural structures and systems that are designed to regulate the physiological response to threat (Bandelow et al., 2017). Research has demonstrated that the main effects of anxiety on cognitive performance are via its deleterious effects on attentional control (e.g., Derakshan, Smyth, et al., 2009). Despite the availability of a variety of different treatments, prevalence rates have remained largely stable for decades (De Graaf et al., 2012). Based on attentional control theory (Derakshan & Eysenck, 2009; Eysenck et al., 2007), an emerging treatment for anxiety symptoms has focused on improving attentional processing via cognitive training in the hopes of reducing anxiety. Results from cognitive training have suggested that there is some support for associated improvements in attentional control and reductions in anxiety symptoms (Hadwin & Richards, 2016).

A more recent approach based on this theory is called neurofeedback treatment. While the benefits of neurofeedback on attentional processing have been observed in the literature, the vast majority of the research has been conducted in children with ADHD (Gevensleben et al., 2009; McReynolds, Villalpando, & Britt, 2018; Sherlin, Arns, Lubar, & Sokhadze, 2010). This study will examine the effects of a neurofeedback training program on attentional processing in adults with self-reported anxiety.

Aims and Hypotheses

The first aim of this study was to determine whether neurofeedback is an effective treatment for improving auditory and visual attention in individuals with self-reported anxiety. We hypothesized that there would be a significant increase in the global score for attention (Full Scale Attention Quotient) on the continuous performance test (CPT) for participants when comparing their performance before treatment to after 20 and 40 sessions of neurofeedback. We also hypothesized that there would be a significant increase in the global score for response control (Full Scale Response Control Quotient) on the CPT for participants when comparing their performance before treatment to after 20 and 40 sessions of neurofeedback.

The second aim of this study was to determine whether neurofeedback would significantly reduce self-reported symptoms of anxiety. We hypothesized that there would be a significant decrease in the self-reported symptoms of anxiety on the General Well-Being Schedule (GWBS) for participants when comparing their reported symptoms before treatment to after 40 sessions of neurofeedback.

CHAPTER TWO

METHODS

Participants

Data was extracted from an archival database of a sample of individuals who had previously received individual neurofeedback training at the California State University San Bernardino (CSUSB) clinic. Clinical neurofeedback services were provided to participants based on a sliding fee scale and because this was an archival study they were not compensated. This study was approved by the CSUSB Institutional Review Board.

Inclusion Criteria

Data were included if participants were 18 years or older at the time of the intervention, had completed at least 20 sessions of neurofeedback and provided valid response sets on visual and auditory Integrated Visual and Auditory-Version 2 (IVA-2) measures. Additionally, only participants who reported symptoms of anxiety during the intake interview with the licensed clinical psychologist were included.

Procedure

Every participant was administered and completed the IVA-2 CPT and the GWBS before beginning their first neurofeedback session. Testing was individually administered and scored in accordance with the specified test guidelines. The CPT was re-administered after the completion of 20 and 40 neurofeedback sessions. Following the last neurofeedback session, the GWBS rating scale was administered for the second time. The CPT data was analyzed comparing baseline test scores and the scores obtained after the 20th and 40th neurofeedback sessions. Analysis of the GWBS rating scale score compared pre-intervention baseline scores to scores obtained after the 40th session of treatment.

Neurofeedback Treatment Protocols

An individualized neurofeedback training plan was developed for each participant and clinically modified as necessary. Treatment was provided on a one-to-one basis in a private room. Therapeutic goals focused on improving auditory and/or visual attention and reducing any identified behavioral symptoms of anxiety. Training was completed using the SmartMind 3 artifact-corrected neurofeedback system with a two-channel EEG station (BrainTrain, Inc., North Chesterfield, Virginia) which continuously filters out both brief facial activities, as well as frequently occurring eye-blink and eye-movement artifacts in real time without interrupting the training program. As previously described (McReynolds, Bell, and Lincourt, 2017), neurofeedback exercises were provided in game-like format that utilized both visual and auditory reinforcement, as well as graphs and numerical scores to provide positive reinforcement. The first step in the training session was to collect participants' baseline EEG data to determine Z-score feedback goals for each participant. Based on each individual's performance, they were provided clinically relevant feedback and adjustments were made to the training protocol to optimize their performance. Sensors were attached and secured using 10-20 electrode paste and electrode sensors after the site locations were prepared. Impedance was checked to meet the manufacturer's requirements prior to the beginning of training. All EEG data was automatically deartifacted and recorded by the SmartMind 3 software.

Measures

Integrated Visual & Auditory 2 Continual Performance Test

The Integrated Visual and Auditory-Version 2 (Sandford & Sandford, 2014) CPT is a psychological test that enables clinicians to assess visual and auditory attention and

response control functioning. The IVA-2 CPT has been found to be a valid and reliable measure of both visual and auditory attention functioning in children and adults. The normative sample included 1,700 individuals ages 6 to 96, with approximately equal numbers of males and females (Maddux, 2010). All IVA-2 scale scores have a mean of 100 and a standard deviation of 15 (Sandford & Sandford, 2014).

As previously mentioned, anxiety affects both visual and verbal processing of information in both clinical and non-clinical populations. The IVA-2 test construction integrates both visual and auditory test stimuli simultaneously during both high and low demand conditions. Unlike many other traditional CPTs, the IVA-2 test design has inter-mixed stimuli (visual and auditory) within a single test condition (high or low demand). This design requires participants to pay close attention to each presented stimuli, thus preventing them from being able to learn to expect a non-target to appear after a series of targets within the same sensory modality, and vice versa for low demand conditions (Sandford & Sandford, 2014).

Although many of the IVA-2's primary scales have substantial associations with working memory indices of neuropsychological tests (Arble, Kuentzel, & Barnett, 2014), the Full Scale Attention Quotient and Full Scale Response Control Quotient scale scores were found to be the most strongly associated. The IVA-2 global and standard measures of attention used in this study are the Full Scale Attention Quotient (FAQ), Auditory Attention Quotient (AAQ), Visual Attention Quotient (VAQ), Full Scale Response Control Quotient (FRCQ), Auditory Response Control Quotient (ARCQ), and Visual Response Control Quotient (VRCQ).

The AAQ is a measure of auditory attention and is comprised of the three attention scales: Vigilance, Focus, and Speed. Vigilance is a measure of inattention as evidenced by omission errors. Speed provides a measure of reaction time for correct responses to visual and auditory stimuli targets. Focus reflects the total variability of reaction time for correct responses to targets.

The VAQ is a measure of visual attention and is comprised of the three attention scales: Vigilance, Focus, and Speed. Vigilance is a measure of inattention as evidenced by omission errors. Speed provides a measure of reaction time for correct responses to visual and auditory stimuli targets. Focus reflects the total variability of reaction time for correct responses to targets.

The FAQ is composed of equal measures of visual and auditory Vigilance, Focus, and Speed subscales. The FAQ represents a test-taker's overall ability to respond accurately and quickly while maintaining focus (attention; Sandford & Sandford, 2014).

The ARCQ is a measure of auditory response control is comprised of the three response control scales: Prudence, Consistency, and Stamina. Prudence is a measure of impulsivity and response inhibition as evidenced by commission errors. Consistency is a measure of the general reliability and variability of response times and is used to measure the ability of test-takers to stay on task. The Stamina scale compares the mean reaction times of correct responses between the first and second half of the test trials and is used to identify difficulties with fatigue and/or effort over time.

The VRCQ is a measure of visual response control is comprised of the three response control scales: Prudence, Consistency, and Stamina. Prudence is a measure of impulsivity and response inhibition as evidenced by commission errors. Consistency is a

measure of the general reliability and variability of response times and is used to measure the ability of test-takers to stay on task. The Stamina scale compares the mean reaction times of correct responses between the first and second half of the test trials and is used to identify difficulties with fatigue and/or effort over time.

The FRCQ is composed of equal measures of visual and auditory Prudence, Consistency, and Stamina subscales. The FRCQ represents a test-taker's overall ability to regulate responses and respond appropriately (response control; Sandford & Sandford, 2014). Factors that load on this scale include the ability to inhibit responses to non-targets, the ability to maintain mental processing speed, and the consistency of recognition reaction times throughout the duration of the test (Sandford & Sandford, 2014). Quotient scores for all IVA-2 scales are reported as standard scores ($M = 100$, $SD = 15$).

General Well-Being Schedule

The GWBS (Dupuy, 1978) is an 18-item questionnaire that is a self-report rating scale that measures a person's general sense of well-being. It incorporates six subscales of well-being including measures of anxiety, positive well-being, depression, vitality, general health, and self-control. The GWBS has been found to be both a valid and reliable measure of well-being for several ethnic groups including young Caucasian males (Fazio, 1977) as well as Japanese (Nakayama, Toyoda, Ohno, Yoshiike, & Futagami, 2000), Mexican-American (Poston et al., 1998), and African-American populations (Taylor et al., 2003).

Statistical Analyses

A one-way repeated-measures analysis of variance (ANOVA) using SPSS Version 20 was conducted to determine whether there were significant differences on FAQ (attention) scores between pre-intervention, after 20 neurofeedback sessions, and after 40 neurofeedback sessions. A second, one-way repeated-measures ANOVA using SPSS Version 20 was conducted to determine whether there were significant differences on FRCQ scores between pre-intervention, after 20 neurofeedback sessions, and after 40 neurofeedback sessions. The independent variable is time (baseline, after 20 sessions, after 40 sessions). The dependent variables are FAQ (attention) and FRCQ (response control).

To address the second aim, an exact sign test was conducted to determine whether the number of anxiety symptoms reported significantly decreased after neurofeedback treatment. The independent variable is time (baseline, after last neurofeedback session). The dependent variable is symptoms of anxiety as measured by the anxiety subscale of the GWBS. Of note, due to restricted access to the California State University, San Bernardino campus (due to COVID-19), only three participants' raw data for the GWBS score was available. As such, this analysis will only include this subset of our sample.

CHAPTER THREE

RESULTS

Demographics

Data from 38 participants evaluated between 2013 to 2020 completed at least 20 sessions of neurofeedback and reported symptoms of anxiety during an intake interview with a licensed clinical psychologist. Of the initial 38 participants, 37 (97%) provided complete and valid response sets on both measures of the continuous performance test of attention (IVA-2) and were included in the first set of analyses. Of the 37 participants who completed at least 20 sessions of neurofeedback, 14 went on to complete 40 sessions of neurofeedback. In the sample of 37 participants, 21 were male (56.8%) and the average age was 40.9 years ($SD = 17.0$). In the subsample of 14 participants, 10 were male (71.4%) and the average age was 39.6 years ($SD = 15.6$).

Paired-Samples t -Tests

A total of six paired samples t -tests were conducted to determine whether there were significant changes in IVA-2 scores from pre-intervention to after 20 sessions of neurofeedback. Means and standard deviations of all variables are reported in Table 1. To reduce the risk of Type I error for the multiple analyses of a single data set used in this study, a Bonferroni correction was used which resulted in adjusted alpha levels of .008 per test ($.05/6$).

Results of the paired samples t -tests indicated there was not a statistically significant mean change in participant FAQ scores from pre-intervention to after 20 sessions, $M = 4.69$, 95% CI [-1.11, 10.48], $t(34) = 1.65$, $p = .109$, $d = 0.278$ (See Table 1). There was a statistically significant mean increase in AAQ scores from pre-

intervention to after 20 sessions, $M = 8.27$, 95% CI [3.89, 12.66], $t(34) = 3.84$, $p = .001$, $d = 0.648$. Conversely, there was not a statistically significant mean change in VAQ scores from pre-intervention to after 20 sessions, $M = 4.38$, 95% CI [-1.16, 9.92], $t(31) = 1.61$, $p = .117$, $d = 0.285$.

Analysis of response control measures indicated there was a statistically significant mean increase in FRCQ scores when comparing performance before intervention to after 20 neurofeedback sessions, $M = 6.8$, 95% CI [2.44, 11.18], $t(35) = 3.16$, $p = .003$, $d = 0.527$. There was also a statistically significant mean increase in ARCQ scores from pre-intervention to after 20 sessions, $M = 8.13$, 95% CI [4.19, 12.07], $t(33) = 4.2$, $p < .001$, $d = 0.72$. In contrast, there was not a statistically significant mean change in VRCQ scores from pre-intervention to after 20 sessions, $M = 2.99$, 95% CI [-2.85, 8.82], $t(35) = 1.04$, $p = .306$, $d = 0.173$.

Table 1. Results of Paired Samples t-Test Comparing IVA-2 Quotient Scale Scores at Baseline and after 20 Sessions of Neurofeedback (N = 37)

IVA-2 Scales	Baseline		20 Sessions		<i>t</i>
	Mean	<i>SD</i>	Mean	<i>SD</i>	
FAQ	89.77	16.58	94.46	20.98	1.65
AAQ	89.65	14.94	97.92	15.41	3.84*
VAQ	91.81	3.65	96.19	18.59	1.61
FRCQ	84.64	18.94	91.44	19.00	3.16*
ARCQ	85.88	17.62	94.01	17.35	4.2**
VRCQ	88.69	21.51	91.68	19.71	1.04

Note. * $p < .005$; ** $p < .001$. FAQ = Full Scale Attention Quotient; AAQ = Auditory Attention Quotient; VAQ = Visual Attention Quotient; FRCQ = Full Scale Response Control Quotient; ARCQ = Auditory Response Control Quotient; VRCQ = Visual Response Control Quotient.

One-way Repeated Measures ANOVAs

A total of two, one-way repeated measures ANOVAs were conducted to test Hypotheses 1 and 2 to determine whether there were significant changes in IVA-2 scores from pre-intervention and post-intervention for the 14 participants who completed 40 sessions of neurofeedback. The first hypothesis predicted that there would be a significant increase on the global score for attention on the IVA-2 (FAQ) for participants when comparing their performance before treatment to after 20 and 40 sessions of neurofeedback. Results indicated there was a statistically significant change in FAQ scores over time, $F(2, 26) = 4.78, p = .017, \eta^2 = 0.269$ (See Table 2). Post hoc analysis with a Bonferroni adjustment revealed that there was a statistically significant mean increase in FAQ scores from pre-intervention to 40 sessions ($M = 16.68, 95\% \text{ CI } [1.44,$

31.92], $p = .03$), but not from pre-intervention to 20 sessions ($M = 10.76$, 95% CI [-5.73, 27.25], $p = .29$), nor from 20 to 40 sessions ($M = 5.92$, 95% CI [-7.23, 19.08], $p = .715$).

As part of an exploratory analysis, two additional one-way repeated measures ANOVAs were conducted to determine whether there were any statistically significant differences between timepoints in the AAQ (auditory attention) and VAQ (visual attention). Results indicated there was not a statistically significant change in AAQ scores over time, $F(2, 26) = 1.40$, $p = .264$, $\eta^2 = 0.097$. Conversely, there was a statistically significant change in VAQ scores over time, $F(2, 24) = 5.29$, $p = .013$, $\eta^2 = 0.306$. Post hoc analysis with a Bonferroni adjustment revealed that there was a statistically significant mean increase in VAQ scores from pre-intervention to 40 sessions ($M = 23.14$, 95% CI [5.73, 40.55], $p = .009$), but not from pre-intervention to 20 sessions ($M = 12.55$, 95% CI [-11.44, 36.53], $p = .515$), nor from 20 to 40 sessions ($M = 10.59$, 95% CI [-6.67, 27.86], $p = .342$).

The second hypothesis predicted that there would be a significant increase on the global score for response control on the IVA-2 (Full Scale Response Control Quotient) for participants when comparing their performance before treatment to after 20 and 40 sessions of neurofeedback. The second hypothesis was also partially supported as there was a statistically significant change in FRCQ score over time, $F(2, 26) = 5.45$, $p = .011$, $\eta^2 = 0.295$. Post hoc analysis with a Bonferroni adjustment revealed that there was a statistically significant mean increase in FRCQ scores from pre-intervention to 40 sessions ($M = 14.08$, 95% CI [.044, 28.11], $p = .049$), but not from pre-intervention to 20 sessions ($M = 6.39$, 95% CI [-3.04, 15.82], $p = .257$), nor from 20 to 40 sessions ($M = 7.69$, 95% CI [-3.57, 18.96], $p = .25$).

As part of an exploratory analysis, two additional one-way repeated measures ANOVAs were conducted to determine whether there were any statistically significant differences between timepoints in the ARCQ (auditory response control) and VRCQ (visual response control). Results indicated that there was not a statistically significant change in ARCQ scores over time $F(2, 26) = 2.45, p = .106, \eta^2 = 0.158$. Conversely, there was a statistically significant change in VRCQ scores over time, $F(2, 26) = 4.23, p = .026, \eta^2 = 0.246$. Post hoc analysis with a Bonferroni adjustment revealed that there was a statistically significant mean increase in VRCQ scores from pre-intervention to 40 sessions ($M = 12.51, 95\% \text{ CI } [1.88, 23.15], p = .02$), but not from pre-intervention to 20 sessions ($M = 7.81, 95\% \text{ CI } [-4.29, 19.91], p = .3$), nor from 20 to 40 sessions ($M = 4.7, 95\% \text{ CI } [-8.25, 17.66], p = .99$).

Table 2. Descriptive Statistics and Results of One-way Repeated Measures ANOVAs for IVA-2 Quotient Scores (N = 14)

	Baseline	20 Sessions	40 Sessions			
Measure	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>F</i>	<i>p</i>	η^2
FAQ	76.98 (20.82)	87.74 (21.54)	93.66 (20.20)	4.78	.017	0.269
AAQ	83.21 (16.20)	89.73 (19.25)	93.31 (25.54)	1.40	.264	0.097
VAQ	76.66 (27.20)	89.21 (26.59)	99.80 (12.65)	5.29	.013	0.306
FRCQ	78.92 (16.76)	85.31 (16.13)	93.00 (17.58)	5.45	.011	0.295
ARCQ	84.41 (19.35)	87.49 (18.59)	95.70 (14.98)	2.45	.106	0.158
VRCQ	79.45 (18.82)	87.26 (16.13)	91.96 (17.95)	4.23	.026	0.246

Note. FAQ = Full Scale Attention Quotient; AAQ = Auditory Attention Quotient; VAQ = Visual Attention Quotient; FRCQ = Full Scale Response Control Quotient; ARCQ = Auditory Response Control Quotient; VRCQ = Visual Response Control Quotient.

Exact Sign Test

An exact sign test was conducted to test H3 to determine the effects of neurofeedback training on anxiety. The third hypothesis predicted that there would be a significant improvement on the anxiety subscale on the General Well-being Schedule for participants when comparing their scores before treatment to after neurofeedback treatment. GWBS raw data of the three available participants was used to examine the changes over time in anxiety subscale scores from pre-intervention to post-intervention (20 sessions of neurofeedback). There was no statistically significant median improvement in anxiety subscale score (*Mdn* = 2 points) from pre-intervention (*Mdn* = 10 points) to post-intervention (*Mdn* = 14 points), $p = .25$. Similarly, there was no statistically significant median improvement in overall GWBS score (*Mdn* = 11 points) from pre-intervention (*Mdn* = 53.5 points) to post-intervention (*Mdn* = 70 points), $p = .5$.

Table 3. Descriptive Statistics and Results of Exact Sign Test for GWBS and Anxiety subscale (N = 3)

Measure	<i>M (SD)</i>	Min	Max	<i>Mdn</i>	<i>p</i>
ANX				2	.250
Baseline	11.67 (3.79)	9	16		
20 Sessions	14.33 (2.52)	12	17		
GWBS				11	.500
Baseline	50.67 (7.37)	45	59		
20 Sessions	65.67 (15.95)	48	79		

Note. GWBS = General Well-being Schedule; ANX = Anxiety subscale of GWBS. *Mdn* = median difference; higher scores indicate higher levels of well-being.

CHAPTER FOUR

DISCUSSION

IVA-2

Although the clinical use of neurofeedback therapy in neurology and psychiatry has increased over the past several decades (Arns, Conners, & Kraemer, 2013), there have only been a handful of studies examining the effects of neurofeedback on attentional processing in adults (Bresnahan & Barry, 2002; McReynolds et al., 2017; Schönenberg et al., 2017). As such, the purpose of this study was to investigate the effectiveness of neurofeedback training for reducing anxiety symptoms and improving attentional processing in adults with self-reported anxiety.

Findings from this study partially supported the first hypothesis such that scores on the Full Scale Attention Quotient significantly improved after 40 sessions of neurofeedback training. As a group, the 14 participants who completed 40 sessions of neurofeedback significantly increased their FAQ scores from a mean of 76.98 (Mildly to Moderately Impaired) at baseline to a mean of 93.66 (Average). While statistically significant changes were not observed after the completion of the first 20 sessions for these 14 individuals, the mean change from baseline to 20 sessions do indicate some clinical relevance (See Table A2). Because the normative mean quotient score of the IVA-2 test is 100 with a standard deviation of 15, any change or difference of eight or more points (one half standard deviation) is considered clinically significant. As a group, their FAQ scores increased after 20 sessions to a mean of 87.74 (Slightly Impaired) which represented a mean difference of 10.76 points.

Results from this study partially supported the second hypothesis such that scores on the Full Scale Response Control Quotient significantly improved after 40 sessions of neurofeedback training. As a group, the 14 participants who completed 40 sessions of neurofeedback significantly increased their FRCQ scores from a mean of 78.92 (Mildly to Moderately Impaired) at baseline to a mean of 93 (Average). Similar to the FAQ findings, there was not a statistically significant change observed from baseline to after 20 sessions. While their FRCQ scores improved to a mean of 85.31 (Slightly Impaired) after 20 sessions, the mean difference (6.39) failed to meet clinical significance.

The results from the current study are consistent with previous results from our lab that found significant improvements in IVA-2 quotient scores after 40 sessions of neurofeedback. Conversely, we did not find statistically significant changes in attentional processing after only 20 neurofeedback sessions unlike our lab's previous two papers which indicated that 20 sessions was sufficient to significantly improve IVA-2 performances (McReynolds et al., 2017, 2018). However, our initial set of analyses evaluating the effects of neurofeedback for individuals who completed at least 20 sessions indicated that there were statistically significant improvements in AAQ, FRCQ, and ARCQ scores from baseline to after 20 sessions. While this difference could be simply due to differences in sample size, it could also reflect potentially significant outcome differences between individuals who completed 20 sessions versus those individuals who went on to complete the 40 sessions. Unfortunately, the researcher was unable to access the physical files at the current time because of COVID-19 restrictions enforced on the CSUSB campus to determine the specific reasons for individuals who chose to discontinue after 20 sessions. Nonetheless, while the majority of research

typically use 30 or more sessions, there is evidence that significant improvements can be elicited within fewer than 20 sessions (Hillard, El-Baz, Sears, Tasman, & Sokhadze, 2013).

Results from the current study also revealed some noteworthy changes in IVA-2 scores between sensory modalities (i.e., auditory and visual) from baseline to after 40 neurofeedback sessions. Specifically, whereas the auditory subscales for response control and attention did not significantly change from baseline, both the visual subscales yielded statistically significant improvements after 40 sessions. To our knowledge, only one study has analyzed the specific effects of neurofeedback on different modalities. Hillard et al., 2013 examined the effects of 12 sessions of neurofeedback on attentional processing in children with ADHD. The researchers found statistically significant improvements in the VAQ and ARCQ but not the AAQ nor the VRCQ. The lack of literature in this area is likely due to not only the young age of this field of research, but also to the lack of any explicit interest in examining the differences between stimulus choice in attentional control studies (Roebuck, Freigang, & Barry, 2016). Unfortunately, the majority of research has used CPTs that only present visual test stimuli, which precludes the opportunity to detect any significant differences in inattention and impulsivity between auditory and visual stimuli. Research has indicated that there is indeed a difference between visual and auditory attentional control abilities with evidence supporting the notion of separate attentional capacities for different modalities (Noterdaeme, Amorosa, Mildenerger, Sitter, & Minow, 2001; Spaulding, Plante, & Vance, 2008). Research has also demonstrated differences between modality-specific processing speed, such that most people regardless of age respond faster to CPT visual

stimuli than to auditory stimuli (Sandford & Sandford, 2014). Further, the literature also suggests that it is important for researchers to also consider within-modality effects on attention as physical differences (e.g., loudness, brightness) between stimuli have different processing demands (Roebuck et al., 2016). It would be important for future research to consider these factors when examining how neurofeedback affects both visual and auditory attentional processing.

Results from the current study yielded small to medium effect sizes for IVA-2 scores after 20 sessions. These results are somewhat smaller when compared to previously published findings from our lab (McReynolds et al., 2017), which reported medium to large effect sizes (Cohen's $d = 0.5-0.7$). A meta-analysis of 15 studies evaluating children and adolescents with ADHD found that neurofeedback resulted in impressive efficacy (e.g., Cohen's $d > .08$) for attentional processing (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009).

Although this study did not have a control group, our findings suggest that the observed outcomes resulted from real change and not from practice effects, as prior research has shown that retesting individuals on the IVA-2 does not change their scores more than three to four points in either direction (Sandford & Sandford, 2014). While the current study did not include follow-up analyses of long-term effects post-training, other studies have shown that the positive benefits of neurofeedback can last from six months to two years for children with ADHD (Gani, Birbaumer, & Strehl, 2008; Gevensleben et al., 2010; Razoki, 2018). It is also important to note that there were several potential moderating factors (e.g., medication status) that were not included in the analyses for this

study due to the researcher being unable to access participant files because of COVID-19 restrictions enforced on the CSUSB campus.

GWBS Anxiety Subscale

As the findings from the current study did not reveal statistically significant changes in either overall GWBS scores nor anxiety subscale scores from baseline to post-intervention, the third hypothesis was not supported. However, the significantly small number of cases available at the time of this study was a severely restricting limitation in the analysis of this hypothesis. The second aim of this study was to determine whether the number of anxiety symptoms reported would significantly decrease from baseline to post-intervention. Contrary to the current study, previous research has shown neurofeedback to be an effective treatment for reducing anxiety related symptoms in adult populations with PTSD, OCD, and specific phobia (Scheinost et al., 2013; Walker, 2009; Zilverstand et al., 2015). Although research on the benefits of neurofeedback on attentional processing in the context of anxiety is limited, previous findings from our lab have shown that neurofeedback is an effective treatment for improving attentional processing in adults, and that these improvements in attentional capacities are associated with significant improvements in overall wellbeing (McReynolds et al., 2017). Researchers using cognitive training to improve attentional processing have found that gains in attentional control were associated with reductions in levels of anxiety and worry symptoms (Hadwin & Richards, 2016; Hotton et al., 2018; Sari et al., 2016).

Strengths and Limitations

The use of archival data in the current study is an important factor to consider as it led to several limitations. Firstly, the use of archival data limited how information was

collected and thus precluded the ability to use utilize other measures that could have been helpful in identifying potential confounding variables or providing additional information on self-reported levels of anxiety. Secondly, the use of archival data did not allow for any follow-up evaluations with participants. A third major limitation of this study was the lack of a control group. Given this design, it is impossible to attribute causal findings to the neurofeedback training which thus weakens internal validity. Despite these limitations, the use of archival data was also a benefit in that it provided uniform data collection over multiple years. Another strength of this study was the use of the IVA-2 CPT which allows for the assessment of both visual and auditory attentional processing. The findings from this study that neurofeedback had a more significant effect on the visual subscales, highlight the importance of examining both modalities and provide questions for future research.

Conclusions

Overall, findings from the present study demonstrated an improvement in attentional processing following neurofeedback training. Unlike most prior neurofeedback studies examining children, the current study demonstrated the effectiveness of neurofeedback training in adults with attentional processing difficulties and is consistent with the few studies that have also focused on adult populations (Bresnahan & Barry, 2002; McReynolds et al., 2017; Schönenberg et al., 2017).

Future research would benefit from including follow-up evaluations to determine whether the gains seen following training are maintained in the long-term. Similarly, future studies would benefit from including pre- and post-neuropsychological testing to assess the differences between visual and auditory attentional processing. Including a

more in-depth evaluation of participants' cognitive abilities could help assess the different subcomponents of attention across different modalities.

REFERENCES

- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Washington, DC: Author.
<https://doi.org/10.1176/appi.books.9780890425596>
- Amir, N., Klumpp, H., Elias, J., Bedwell, J. S., Yanasak, N., & Miller, L. S. (2005). Increased activation of the anterior cingulate cortex during processing of disgust faces in individuals with social phobia. *Biological Psychiatry*, *57*(9), 975–981.
<https://doi.org/10.1016/j.biopsych.2005.01.044>
- Antoni, M. H., Lutgendorf, S. K., Blomberg, B., Carver, C. S., Lechner, S., Diaz, A., ... Cole, S. W. (2012). Cognitive-behavioral stress management reverses anxiety-related leukocyte transcriptional dynamics. *Biological Psychiatry*, *71*(4), 366–372. <https://doi.org/10.1016/j.biopsych.2011.10.007>
- Arble, E., Kuentzel, J., & Barnett, D. (2014). Convergent validity of the integrated visual and auditory continuous performance test (IVA+Plus): Associations with working memory, processing speed, and behavioral ratings. *Archives of Clinical Neuropsychology*, *29*(3), 300–312. <https://doi.org/10.1093/arclin/acu006>
- Arns, M., Conners, C. K., & Kraemer, H. C. (2013). A decade of EEG theta/beta ratio research in ADHD: A meta-analysis. *Journal of Attention Disorders*, *17*(5), 374–383. <https://doi.org/10.1177/1087054712460087>
- Arns, M., de Ridder, S., Strehl, U., Breteler, M., & Coenen, A. (2009). Efficacy of neurofeedback treatment in ADHD: The effects on inattention, impulsivity, and hyperactivity: A meta-analysis. *Clinical EEG and Neuroscience*, *40*(3), 180–189. <https://doi.org/10.1177/155005940904000311>
- Baddeley, A. (1986). *Working Memory*. Clarendon Press.
- Bandelow, B., Lichte, T., Rudolf, S., Wiltink, J., & Beutel, M. E. (2014). The diagnosis of and treatment recommendations for anxiety disorders. *Deutsches Arzteblatt Online*, *111*(27–28), 473–480. <https://doi.org/10.3238/arztebl.2014.0473>
- Bandelow, B., Lichte, T., Rudolf, S., Wiltink, J., & Beutel, M. E. (2015). The German guidelines for the treatment of anxiety disorders. *European Archives of Psychiatry and Clinical Neuroscience*, *265*(5), 363–373. <https://doi.org/10.1007/s00406-014-0563-z>
- Bandelow, B., Michaelis, S., & Wedekind, D. (2017). Treatment of anxiety disorders. *Dialogues in Clinical Neuroscience*, *19*(2), 93–107. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/28867934>
- Bandelow, B., Reitt, M., Röver, C., Michaelis, S., Görlich, Y., & Wedekind, D. (2015). Efficacy of treatments for anxiety disorders: a meta-analysis. *International*

Clinical Psychopharmacology, 30(4), 183–192.
<https://doi.org/10.1097/YIC.0000000000000078>

- Bandelow, B., Sher, L., Bunevicius, R., Hollander, E., Kasper, S., Zohar, J., ... Vega, J. (2012). Guidelines for the pharmacological treatment of anxiety disorders, obsessive-compulsive disorder, and posttraumatic stress disorder in primary care. *International Journal of Psychiatry in Clinical Practice*, 16(2), 77–84.
<https://doi.org/10.3109/13651501.2012.667114>
- Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & Van Ijzendoorn, M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin*, 133(1), 1–24. <https://doi.org/10.1037/0033-2909.133.1.1>
- Baxter, A. J., Vos, T., Scott, K. M., Ferrari, A. J., & Whiteford, H. A. (2014). The global burden of anxiety disorders in 2010. *Psychological Medicine*, 44(11), 2363–2374.
<https://doi.org/10.1017/S0033291713003243>
- Beck, S. J., Hanson, C. A., Puffenberger, S. S., Benninger, K. L., & Benninger, W. B. (2010). A controlled trial of working memory training for children and adolescents with ADHD. *Journal of Clinical Child and Adolescent Psychology*, 39(6), 825–836. <https://doi.org/10.1080/15374416.2010.517162>
- Becker, E. S., Rinck, M., Margraf, J., & Roth, W. T. (2001). The emotional Stroop effect in anxiety disorders: General emotionality or disorder specificity? *Journal of Anxiety Disorders*, 15(3), 147–159. [https://doi.org/10.1016/S0887-6185\(01\)00055-X](https://doi.org/10.1016/S0887-6185(01)00055-X)
- Berggren, N., & Derakshan, N. (2013). Attentional control deficits in trait anxiety: Why you see them and why you don't. *Biological Psychology*, 92(3), 440–446.
<https://doi.org/10.1016/j.biopsycho.2012.03.007>
- Bishop, S., Duncan, J., Brett, M., & Lawrence, A. D. (2004). Prefrontal cortical function and anxiety: Controlling attention to threat-related stimuli. *Nature Neuroscience*, 7(2), 184–188. <https://doi.org/10.1038/nn1173>
- Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: an integrative account. *Trends in Cognitive Sciences*, 11(7), 307–316.
<https://doi.org/10.1016/j.tics.2007.05.008>
- Bishop, S. J. (2008). Neural mechanisms underlying selective attention to threat. *Annals of the New York Academy of Sciences*, 1129, 141–152.
<https://doi.org/10.1196/annals.1417.016>
- Bishop, S. J. (2009). Trait anxiety and impoverished prefrontal control of attention. *Nature Neuroscience*, 12(1), 92–98. <https://doi.org/10.1038/nn.2242>

- Bishop, S. J., Jenkins, R., & Lawrence, A. D. (2007). Neural processing of fearful faces: Effects of anxiety are gated by perceptual capacity limitations. *Cerebral Cortex*, *17*(7), 1595–1603. <https://doi.org/10.1093/cercor/bhl070>
- Blanchard, D. C., Griebel, G., Pobbe, R., & Blanchard, R. J. (2011). Risk assessment as an evolved threat detection and analysis process. *Neuroscience and Biobehavioral Reviews*, *35*(4), 991–998. <https://doi.org/10.1016/j.neubiorev.2010.10.016>
- Bloom, D. E., Cafiero, E. T., Jané-Llopis, E., Abrahams-Gessel, S., Bloom, L. R., Fathima, S., ... Weinstein, C. (2011). *The Global Economic Burden of Non-communicable Diseases*. Geneva: World Economic Forum.
- Boddez, Y., Vervliet, B., Baeyens, F., Lauwers, S., Hermans, D., & Beckers, T. (2012). Expectancy bias in a selective conditioning procedure: Trait anxiety increases the threat value of a blocked stimulus. *Journal of Behavior Therapy and Experimental Psychiatry*, *43*(2), 832–837. <https://doi.org/10.1016/j.jbtep.2011.11.005>
- Bresnahan, S. M., & Barry, R. J. (2002). Specificity of quantitative EEG analysis in adults with attention deficit hyperactivity disorder. *Psychiatry Research*, *112*(2), 133–144. [https://doi.org/10.1016/S0165-1781\(02\)00190-7](https://doi.org/10.1016/S0165-1781(02)00190-7)
- Britton, J. C., Lissek, S., Grillon, C., Norcross, M. A., & Pine, D. S. (2011). Development of anxiety: The role of threat appraisal and fear learning. *Depression and Anxiety*, *28*(1), 5–17. <https://doi.org/10.1002/da.20733>
- Bruce, S. E., Yonkers, K. A., Otto, M. W., Eisen, J. L., Weisberg, R. B., Pagano, M., ... Keller, M. B. (2005). Influence of psychiatric comorbidity on recovery and recurrence in generalized anxiety disorder, social phobia, and panic disorder: A 12-year prospective study. *American Journal of Psychiatry*, *162*(6), 1179–1187. <https://doi.org/10.1176/appi.ajp.162.6.1179>
- Bystritsky, A. (2006). Treatment-resistant anxiety disorders. *Molecular Psychiatry*, *11*(9), 805–814. <https://doi.org/10.1038/sj.mp.4001852>
- Calvo, M. G., & Eysenck, M. W. (1996). Phonological working memory and reading in test anxiety. *Memory (Hove, England)*, *4*(3), 289–305. <https://doi.org/10.1080/096582196388960>
- Caselli, R. J., Reiman, E. M., Hentz, J. G., Osborne, D., & Alexander, G. E. (2004). A distinctive interaction between chronic anxiety and problem solving in asymptomatic APOE e4 homozygotes. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *16*(3), 320–329. <https://doi.org/10.1176/jnp.16.3.320>
- Chalmers, J. A., Quintana, D. S., Abbott, M. J. A., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: A meta-analysis. *Frontiers in Psychiatry*, *5*(JUL), 1–11. <https://doi.org/10.3389/fpsy.2014.00080>

- Chisholm, D., Sweeny, K., Sheehan, P., Rasmussen, B., Smit, F., Cuijpers, P., & Saxena, S. (2016). Scaling-up treatment of depression and anxiety: a global return on investment analysis. *The Lancet. Psychiatry*, 3(5), 415–424. [https://doi.org/10.1016/S2215-0366\(16\)30024-4](https://doi.org/10.1016/S2215-0366(16)30024-4)
- Choi, S. W., Chi, S. E., Chung, S. Y., Kim, J. W., Ahn, C. Y., & Kim, H. T. (2011). Is alpha wave neurofeedback effective with randomized clinical trials in depression? A pilot study. *Neuropsychobiology*, 63(1), 43–51. <https://doi.org/10.1159/000322290>
- Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain. *Nature Reviews Neuroscience*, 3(3), 201–215. <https://doi.org/10.1038/nrn755>
- Cristea, I. A., Kok, R. N., & Cuijpers, P. (2015). Efficacy of cognitive bias modification interventions in anxiety and depression: meta-analysis. *British Journal of Psychiatry*, 206(1), 7–16. <https://doi.org/10.1192/bjp.bp.114.146761>
- Crits-Christoph, P., Newman, M. G., Rickels, K., Gallop, R., Gibbons, M. B. C., Hamilton, J. L., ... Pastva, A. M. (2011). Combined medication and cognitive therapy for generalized anxiety disorder. *Journal of Anxiety Disorders*, 25(8), 1087–1094. <https://doi.org/10.1016/j.janxdis.2011.07.007>
- Dalgleish, T., Taghavi, R., Neshat-Doost, H., Moradi, A., Canterbury, R., & Yule, W. (2003). Patterns of processing bias for emotional information across clinical disorders: A comparison of attention, memory, and prospective cognition in children and adolescents with depression, generalized anxiety, and posttraumatic stress disorder. *Journal of Clinical Child and Adolescent Psychology*, 32(1), 10–21. <https://doi.org/10.1207/15374420360533022>
- Dash, S. R., & Davey, G. C. L. (2012). An experimental investigation of the role of negative mood in worry: The role of appraisals that facilitate systematic information processing. *Journal of Behavior Therapy and Experimental Psychiatry*, 43(2), 823–831. <https://doi.org/10.1016/j.jbtep.2011.12.002>
- Davidson, J. R. T., Wittchen, H.-U., Llorca, P.-M., Erickson, J., Detke, M., Ball, S. G., & Russell, J. M. (2008). Duloxetine treatment for relapse prevention in adults with generalized anxiety disorder: A double-blind placebo-controlled trial. *European Neuropsychopharmacology*, 18(9), 673–681. <https://doi.org/10.1016/j.euroneuro.2008.05.002>
- Davis, M., Walker, D. L., Miles, L., & Grillon, C. (2010). Phasic vs sustained fear in rats and humans: Role of the extended amygdala in fear vs anxiety. *Neuropsychopharmacology*, 35(1), 105–135. <https://doi.org/10.1038/npp.2009.109>

- De Graaf, R., Ten Have, M., Van Gool, C., & Van Dorsselaer, S. (2012). Prevalence of mental disorders and trends from 1996 to 2009. Results from the Netherlands Mental Health Survey and Incidence Study-2. *Social Psychiatry and Psychiatric Epidemiology*, *47*(2), 203–213. <https://doi.org/10.1007/s00127-010-0334-8>
- Dekker, J. M., Crow, R. S., Folsom, A. R., Hannan, P. J., Liao, D., Swenne, C. A., & Schouten, E. G. (2000). Low heart rate variability in a 2-minute rhythm strip predicts risk of coronary heart disease and mortality from several causes: The ARIC study. *Circulation*, *102*(11), 1239–1244. <https://doi.org/10.1161/01.CIR.102.11.1239>
- Derakshan, N., Ansari, T. L., Hansard, M., Shoker, L., & Eysenck, M. W. (2009). Anxiety, inhibition, efficiency, and effectiveness: an investigation using the antisaccade task. *Experimental Psychology*, *56*(1), 48–55. <https://doi.org/10.1027/1618-3169.56.1.48>
- Derakshan, N., & Eysenck, M. W. (2009). Anxiety, processing efficiency, and cognitive performance. *European Psychologist*, *14*(2), 168–176. <https://doi.org/10.1027/1016-9040.14.2.168>
- Derakshan, N., Smyth, S., & Eysenck, M. W. (2009). Effects of state anxiety on performance using a task-switching paradigm: An investigation of attentional control theory. *Psychonomic Bulletin & Review*, *16*(6), 1112–1117. <https://doi.org/10.3758/PBR.16.6.1112>
- Derryberry, D., & Reed, M. A. (2002). Anxiety-related attentional biases and their regulation by attentional control. *Journal of Abnormal Psychology*, *111*(2), 225–236. <https://doi.org/10.1037/0021-843X.111.2.225>
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*(3), 355–391. <https://doi.org/10.1037/0033-2909.130.3.355>
- Dunning, D. L., Holmes, J., & Gathercole, S. E. (2013). Does working memory training lead to generalized improvements in children with low working memory? A randomized controlled trial. *Developmental Science*, *16*(6), 915–925. <https://doi.org/10.1111/desc.12068>
- Dupuy, H. (1978). Self-representations of general psychological well-being of American adults. *Paper presented at the American Public Health Association Meeting, Los Angeles, CA*. https://doi.org/10.1007/978-0-387-79948-3_1939
- Durham, R., Chambers, J., Power, K., Sharp, D., Macdonald, R., Major, K., ... Gumley, A. (2005). Long-term outcome of cognitive behaviour therapy clinical trials in central Scotland. *Health Technology Assessment*, *9*(42), 1–128. <https://doi.org/10.3310/hta9420>

- Dusek, J. B., Mergler, N. L., & Kermis, M. D. (1976). Attention, encoding, and information processing in low- and high-test-anxious children. *Child Development, 47*(1), 201. <https://doi.org/10.2307/1128300>
- Egloff, B., & Hock, M. (2001). Interactive effects of state anxiety and trait anxiety on emotional Stroop interference. *Personality and Individual Differences, 31*(6), 875–882. [https://doi.org/10.1016/S0191-8869\(00\)00188-4](https://doi.org/10.1016/S0191-8869(00)00188-4)
- El Khoury-Malhame, M., Reynaud, E., Soriano, A., Michael, K., Salgado-Pineda, P., Zendjidjian, X., ... Khalfa, S. (2011). Amygdala activity correlates with attentional bias in PTSD. *Neuropsychologia, 49*(7), 1969–1973. <https://doi.org/10.1016/j.neuropsychologia.2011.03.025>
- Enriquez-Geppert, S., Huster, R. J., & Herrmann, C. S. (2017). EEG-neurofeedback as a tool to modulate cognition and behavior: A review tutorial. *Frontiers in Human Neuroscience, 11*(February), 1–19. <https://doi.org/10.3389/fnhum.2017.00051>
- Eysenck, M. W. (1992). *Anxiety: The cognitive perspective*. Hove: Lawrence Erlbaum. Retrieved from <https://www.taylorfrancis.com/books/9780203775677>
- Eysenck, M. W., & Byrne, A. (1992). Anxiety and susceptibility to distraction. *Personality and Individual Differences, 13*(7), 793–798. [https://doi.org/10.1016/0191-8869\(92\)90052-Q](https://doi.org/10.1016/0191-8869(92)90052-Q)
- Eysenck, M. W., & Calvo, M. G. (1992). Anxiety and performance: The processing efficiency theory. *Cognition & Emotion, 6*(6), 409–434. <https://doi.org/10.1080/02699939208409696>
- Eysenck, M. W., & Derakshan, N. (1998). Working memory capacity in high trait-anxious and repressor groups. *Cognition & Emotion, 12*(5), 697–713. <https://doi.org/10.1080/026999398379501>
- Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336–353. <https://doi.org/10.1037/1528-3542.7.2.336>
- Eysenck, M. W., & Graydon, J. (1989). Susceptibility to distraction as a function of personality. *Personality and Individual Differences, 10*(6), 681–687. [https://doi.org/10.1016/0191-8869\(89\)90227-4](https://doi.org/10.1016/0191-8869(89)90227-4)
- Fawcett, J. (2013). Suicide and anxiety in DSM-5. *Depression and Anxiety, 30*(10), 898–901. <https://doi.org/10.1002/da.22058>
- Fazio, A. F. (1977). A concurrent validation study of the NCHS General Well-Being Schedule. *Vital and Health Statistics. Series 2, Data Evaluation and Methods Research, (73)*, 1–53. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/610049>

- Fredrikson, M., & Furmark, T. (2006). Amygdaloid regional cerebral blood flow and subjective fear during symptom provocation in anxiety disorders. *Annals of the New York Academy of Sciences*, 985(1), 341–347. <https://doi.org/10.1111/j.1749-6632.2003.tb07092.x>
- Friedman, B. H. (2007). An autonomic flexibility-neurovisceral integration model of anxiety and cardiac vagal tone. *Biological Psychology*, 74(2), 185–199. <https://doi.org/10.1016/j.biopsycho.2005.08.009>
- Friedman, N., & Miyake, A. (2004). The relations among inhibition and interference control functions: A latent-variable analysis. *Journal of Experimental Psychology: General*, 133(1), 101–135. <https://doi.org/10.1037/0096-3445.133.1.101>
- Frijda, N. H. (1988). The laws of emotion. *American Psychologist*, 43(5), 349–358. <https://doi.org/10.1037/0003-066X.43.5.349>
- Gani, C., Birbaumer, N., & Strehl, U. (2008). Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with attentiondeficit/hyperactivity disorder (ADHD). *Int J Bioelectromagn*, 10(4), 209–232.
- Garfinkel, S. N., Eccles, J. A., & Critchley, H. D. (2015). The heart, the brain, and the regulation of emotion. *JAMA Psychiatry*, 72(11), 1071–1072. <https://doi.org/10.1001/jamapsychiatry.2015.1493>
- Gevensleben, H., Holl, B., Albrecht, B., Schlamp, D., Kratz, O., Studer, P., ... Heinrich, H. (2010). Neurofeedback training in children with ADHD: 6-month follow-up of a randomised controlled trial. *European Child and Adolescent Psychiatry*, 19(9), 715–724. <https://doi.org/10.1007/s00787-010-0109-5>
- Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., ... Heinrich, H. (2009). Is neurofeedback an efficacious treatment for ADHD? A randomised controlled clinical trial. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 50(7), 780–789. <https://doi.org/10.1111/j.1469-7610.2008.02033.x>
- Goodwin, A. H., & Sher, K. J. (1992). Deficits in set-shifting ability in nonclinical compulsive checkers. *Journal of Psychopathology and Behavioral Assessment*, 14(1), 81–92. <https://doi.org/10.1007/BF00960093>
- Greenberg, P. E., Sisitsky, T., Kessler, R. C., Finkelstein, S. N., Berndt, E. R., Davidson, J. R., ... Fyer, A. J. (1999). The economic burden of anxiety disorders in the 1990s. *The Journal of Clinical Psychiatry*, 60(7), 427–435. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10453795>
- Gustavsson, A., Svensson, M., Jacobi, F., Allgulander, C., Alonso, J., Beghi, E., ... CDBE2010Study Group. (2011). Cost of disorders of the brain in Europe 2010.

European Neuropsychopharmacology : The Journal of the European College of Neuropsychopharmacology, 21(10), 718–779.
<https://doi.org/10.1016/j.euroneuro.2011.08.008>

- Hadwin, J. A., & Richards, H. J. (2016). Working memory training and CBT reduces anxiety symptoms and attentional biases to threat: A preliminary study. *Frontiers in Psychology*, 7(February), 1–12. <https://doi.org/10.3389/fpsyg.2016.00047>
- Hardt, J., & Kamiya, J. (1978). Anxiety change through electroencephalographic alpha feedback seen only in high anxiety subjects. *Science*, 201(4350), 79–81.
<https://doi.org/10.1126/science.663641>
- Härter, M. C., Conway, K. P., & Merikangas, K. R. (2003). Associations between anxiety disorders and physical illness. *European Archives of Psychiatry and Clinical Neuroscience*, 253(6), 313–320. <https://doi.org/10.1007/s00406-003-0449-y>
- Hillard, B., El-Baz, A. S., Sears, L., Tasman, A., & Sokhadze, E. M. (2013). Neurofeedback training aimed to improve focused attention and alertness in children with ADHD. *Clinical EEG and Neuroscience*, 44(3), 193–202.
<https://doi.org/10.1177/1550059412458262>
- Hoedlmoser, K., Pecherstorfer, T., Gruber, G., Anderer, P., Doppelmayr, M., Klimesch, W., & Schabus, M. (2008). Instrumental conditioning of human sensorimotor rhythm (12-15 Hz) and its impact on sleep as well as declarative learning. *Sleep*, 31(10), 1401–1408. <https://doi.org/10.5665/sleep/31.10.1401>
- Hoffman, D. L., Dukes, E. M., & Wittchen, H.-U. (2008). Human and economic burden of generalized anxiety disorder. *Depression and Anxiety*, 25(1), 72–90.
<https://doi.org/10.1002/da.20257>
- Hotton, M., Derakshan, N., & Fox, E. (2018). A randomised controlled trial investigating the benefits of adaptive working memory training for working memory capacity and attentional control in high worriers. *Behaviour Research and Therapy*, 100(November 2017), 67–77. <https://doi.org/10.1016/j.brat.2017.10.011>
- Indovina, I., Robbins, T. W., Núñez-Elizalde, A. O., Dunn, B. D., & Bishop, S. J. (2011). Fear-conditioning mechanisms associated with trait vulnerability to anxiety in humans. *Neuron*, 69(3), 563–571. <https://doi.org/10.1016/j.neuron.2010.12.034>
- Janszky, I., Ahnve, S., Lundberg, I., & Hemmingsson, T. (2010). Early-onset depression, anxiety, and risk of subsequent coronary heart disease. 37-year follow-up of 49,321 young Swedish men. *Journal of the American College of Cardiology*, 56(1), 31–37. <https://doi.org/10.1016/j.jacc.2010.03.033>
- Judah, M. R., Grant, D. M. M., Mills, A. C., & Lechner, W. V. (2014). Factor structure and validation of the attentional control scale. *Cognition and Emotion*, 28(3), 433–451. <https://doi.org/10.1080/02699931.2013.835254>

- Katzman, M. A., Brawman-Mintzer, O., Reyes, E. B., Olausson, B., Liu, S., & Eriksson, H. (2011). Extended-release quetiapine fumarate (quetiapine XR) monotherapy as maintenance treatment for generalized anxiety disorder. *International Clinical Psychopharmacology*, 26(1), 11–24. <https://doi.org/10.1097/YIC.0b013e32833e34d9>
- Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: Insights from the study of heart rate variability. *International Journal of Psychophysiology*, 89(3), 288–296. <https://doi.org/10.1016/j.ijpsycho.2013.06.018>
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, 62(6), 593. <https://doi.org/10.1001/archpsyc.62.6.593>
- Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H.-U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research*, 21(3), 169–184. <https://doi.org/10.1002/mpr.1359>
- Kessler, R. C., Ruscio, A. M., Shear, K., & Wittchen, H.-U. (2010). Epidemiology of anxiety disorders. *Current Topics in Behavioral Neurosciences*, 2, 21–35. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/21309104>
- Kramer, A. F., Bherer, L., Colcombe, S. J., Dong, W., & Greenough, W. T. (2004). Environmental influences on cognitive and brain plasticity during aging. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences*, 59(9), M940-57. <https://doi.org/10.1093/gerona/59.9.m940>
- LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience*, 23(1), 155–184. <https://doi.org/10.1146/annurev.neuro.23.1.155>
- Leon, A. C., Portera, L., & Weissman, M. M. (1995). The social costs of anxiety disorders. *The British Journal of Psychiatry. Supplement*, (27), 19–22. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/7794589>
- Levenson, R. W. (1988). Emotion and the autonomic nervous system: A prospectus for research on autonomic specificity. *Social Psychophysiology and Emotion: Theory and Clinical Applications*, 17–42.
- Li, C., Barker, L., Ford, E. S., Zhang, X., Strine, T. W., & Mokdad, A. H. (2008). Diabetes and anxiety in US adults: Findings from the 2006 behavioral risk factor surveillance system. *Diabetic Medicine*, 25(7), 878–881. <https://doi.org/10.1111/j.1464-5491.2008.02477.x>

- Lonigan, C. J., & Vasey, M. W. (2009). Negative affectivity, effortful control, and attention to threat-relevant stimuli. *Journal of Abnormal Child Psychology*, *37*(3), 387–399. <https://doi.org/10.1007/s10802-008-9284-y>
- Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR). *Biofeedback and Self-Regulation*, *1*(3), 293–306. <https://doi.org/10.1007/BF01001170>
- MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology*, *8*(1), 189–217. <https://doi.org/10.1146/annurev-clinpsy-032511-143052>
- MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*(1), 15–20. <https://doi.org/10.1037/0021-843X.95.1.15>
- MacLeod, C., & McLaughlin, K. (1995). Implicit and explicit memory bias in anxiety: A conceptual replication. *Behaviour Research and Therapy*, *33*(1), 1–14. [https://doi.org/10.1016/0005-7967\(94\)E0004-3](https://doi.org/10.1016/0005-7967(94)E0004-3)
- MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of Abnormal Psychology*, *111*(1), 107–123. <https://doi.org/10.1037/0021-843X.111.1.107>
- Markowitz, A. (1969). Influence of the repression-sensitization dimension, affect value, and ego threat on incidental learning. *Journal of Personality and Social Psychology*, *11*(4), 374–380. <https://doi.org/10.1037/h0027258>
- Martens, E. J., de Jonge, P., Na, B., Cohen, B. E., Lett, H., & Whooley, M. A. (2010). Scared to death? Generalized anxiety disorder and cardiovascular events in patients with stable coronary heart disease: The Heart and Soul Study. *Archives of General Psychiatry*, *67*(7), 750–758. <https://doi.org/10.1001/archgenpsychiatry.2010.74>
- Mathersul, D., Williams, L. M., Hopkinson, P. J., & Kemp, A. H. (2008). Investigating models of affect: Relationships among eeg alpha asymmetry, depression, and anxiety. *Emotion*, *8*(4), 560–572. <https://doi.org/10.1037/a0012811>
- Mathews, A., May, J., Mogg, K., & Eysenck, M. (1990). Attentional bias in anxiety: Selective search or defective filtering? *Journal of Abnormal Psychology*, *99*(2), 166–173. <https://doi.org/10.1037/0021-843X.99.2.166>
- McEwen, B. S., Eiland, L., Hunter, R. G., & Miller, M. M. (2012). Stress and anxiety: Structural plasticity and epigenetic regulation as a consequence of stress.

Neuropharmacology, 62(1), 3–12.
<https://doi.org/10.1016/j.neuropharm.2011.07.014>

- McReynolds, C., Bell, J., & Lincourt, T. (2017). Neurofeedback: A noninvasive treatment for symptoms of posttraumatic stress disorder in veterans. *NeuroRegulation*, 4(3–4), 114–124. <https://doi.org/10.15540/nr.4.3-4.114>
- McReynolds, C., Villalpando, L., & Britt, C. (2018). Using neurofeedback to improve ADHD symptoms in school-aged children. *NeuroRegulation*, 5(4), 109–128. <https://doi.org/10.15540/nr.5.4.109>
- Melby-Lervåg, M., & Hulme, C. (2013). Is working memory training effective? A meta-analytic review. *Developmental Psychology*, 49(2), 270–291. <https://doi.org/10.1037/a0028228>
- Melby-Lervåg, M., Redick, T. S., & Hulme, C. (2016). Working memory training does not improve performance on measures of intelligence or other measures of “far transfer”: Evidence from a meta-analytic review. *Perspectives on Psychological Science*, 11(4), 512–534. <https://doi.org/10.1177/1745691616635612>
- Mendes, W. B., Blascovich, J., Hunter, S. B., Lickel, B., & Jost, J. T. (2007). Threatened by the unexpected: Physiological responses during social interactions with expectancy-violating partners. *Journal of Personality and Social Psychology*, 92(4), 698–716. <https://doi.org/10.1037/0022-3514.92.4.698>
- Mendes, W. B., Major, B., McCoy, S., & Blascovich, J. (2008). How attributional ambiguity shapes physiological and emotional responses to social rejection and acceptance. *Journal of Personality and Social Psychology*, 94(2), 278–291. <https://doi.org/10.1037/0022-3514.94.2.278>
- Milad, M. R., & Quirk, G. J. (2002). Neurons in medial prefrontal cortex signal memory for fear extinction. *Nature*, 420(6911), 70–74. <https://doi.org/10.1038/nature01138>
- Milad, M. R., Rauch, S. L., Pitman, R. K., & Quirk, G. J. (2006). Fear extinction in rats: Implications for human brain imaging and anxiety disorders. *Biological Psychology*, 73(1), 61–71. <https://doi.org/10.1016/j.biopsycho.2006.01.008>
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*, 41(1), 49–100. <https://doi.org/10.1006/cogp.1999.0734>
- Mogg, K., & Bradley, B. P. (2016). Anxiety and attention to threat: Cognitive mechanisms and treatment with attention bias modification. *Behaviour Research and Therapy*, 87, 76–108. <https://doi.org/10.1016/j.brat.2016.08.001>

- Monk, C. S., Telzer, E. H., Mogg, K., Bradley, B. P., Mai, X., Louro, H. M. C., ... Pine, D. S. (2008). Amygdala and ventrolateral prefrontal cortex activation to masked angry faces in children and adolescents with generalized anxiety disorder. *Archives of General Psychiatry*, *65*(5), 568–576. <https://doi.org/10.1001/archpsyc.65.5.568>
- Monsell, S. (2003). Task switching. *Trends in Cognitive Sciences*, *7*(3), 134–140. [https://doi.org/10.1016/S1364-6613\(03\)00028-7](https://doi.org/10.1016/S1364-6613(03)00028-7)
- Montgomery, S. A., Nil, R., Dürr-Pal, N., Loft, H., & Boulenger, J.-P. (2005). A 24-week randomized, double-blind, placebo-controlled study of escitalopram for the prevention of generalized social anxiety disorder. *The Journal of Clinical Psychiatry*, *66*(10), 1270–1278. <https://doi.org/10.4088/JCP.v66n1009>
- Moore, N. C. (2000). A review of EEG biofeedback treatment of anxiety disorders. *Clinical EEG and Neuroscience*, *31*(1), 1–6. <https://doi.org/10.1177/155005940003100105>
- Muscatello, M. R., Spina, E., Bandelow, B., & Baldwin, D. S. (2012). Clinically relevant drug interactions in anxiety disorders. *Human Psychopharmacology: Clinical and Experimental*, *27*(3), 239–253. <https://doi.org/10.1002/hup.2217>
- Nakayama, T., Toyoda, H., Ohno, K., Yoshiike, N., & Futagami, T. (2000). Validity, reliability and acceptability of the Japanese version of the General Well-Being Schedule (GWBS). *Quality of Life Research*, *9*(5), 529–539. <https://doi.org/10.1023/A:1008940902849>
- Newman, M. G., & Llera, S. J. (2011). A novel theory of experiential avoidance in generalized anxiety disorder: A review and synthesis of research supporting a contrast avoidance model of worry. *Clinical Psychology Review*, *31*(3), 371–382. <https://doi.org/10.1016/j.cpr.2011.01.008>
- Noterdaeme, M., Amorosa, H., Mildenerger, K., Sitter, S., & Minow, F. (2001). Evaluation of attention problems in children with autism and children with a specific language disorder. *European Child & Adolescent Psychiatry*, *10*(1), 58–66. <https://doi.org/10.1007/s007870170048>
- O'Donovan, A., Slavich, G. M., Epel, E. S., & Neylan, T. C. (2013). Exaggerated neurobiological sensitivity to threat as a mechanism linking anxiety with increased risk for diseases of aging. *Neuroscience & Biobehavioral Reviews*, *37*(1), 96–108. <https://doi.org/10.1016/j.neubiorev.2012.10.013>
- Olatunji, B. O., Cisler, J. M., & Deacon, B. J. (2010). Efficacy of cognitive behavioral therapy for anxiety disorders: A review of meta-analytic findings. *Psychiatric Clinics of North America*, *33*(3), 557–577. <https://doi.org/10.1016/j.psc.2010.04.002>

- Olatunji, B. O., Cisler, J. M., & Tolin, D. F. (2007). Quality of life in the anxiety disorders: A meta-analytic review. *Clinical Psychology Review, 27*(5), 572–581. <https://doi.org/10.1016/j.cpr.2007.01.015>
- Orem, D. M., Petrac, D. C., & Bedwell, J. S. (2008). Chronic self-perceived stress and set-shifting performance in undergraduate students. *Stress, 11*(1), 73–78. <https://doi.org/10.1080/10253890701535103>
- Peers, P. V., & Lawrence, A. D. (2009). Attentional control of emotional distraction in rapid serial visual presentation. *Emotion, 9*(1), 140–145. <https://doi.org/10.1037/a0014507>
- Phan, K. L., Fitzgerald, D. A., Nathan, P. J., & Tancer, M. E. (2006). Association between amygdala hyperactivity to harsh faces and severity of social anxiety in generalized social phobia. *Biological Psychiatry, 59*(5), 424–429. <https://doi.org/10.1016/j.biopsych.2005.08.012>
- Poston, W. S. C., Olvera, N. E., Yanez, C., Haddock, C. K., Dunn, J. K., Hanis, C. L., & Foreyt, J. P. (1998). Evaluation of the factor structure and psychometric characteristics of the General Well-Being Schedule (GWB) with Mexican American Women. *Women & Health, 27*(3), 51–64. https://doi.org/10.1300/J013v27n03_04
- Qi, S., Chen, J., Hitchman, G., Zeng, Q., Ding, C., Li, H., & Hu, W. (2014). Reduced representations capacity in visual working memory in trait anxiety. *Biological Psychology, 103*, 92–99. <https://doi.org/10.1016/j.biopsycho.2014.08.010>
- Rayegani, S. M., Raeissadat, S. A., Sedighipour, L., Mohammad Rezazadeh, I., Bahrami, M. H., Eliaspour, D., & Khosrawi, S. (2014). Effect of neurofeedback and electromyographic-biofeedback therapy on improving hand function in stroke patients. *Topics in Stroke Rehabilitation, 21*(2), 137–151. <https://doi.org/10.1310/tsr2102-137>
- Razoki, B. (2018). Neurofeedback versus psychostimulants in the treatment of children and adolescents with attention-deficit/hyperactivity disorder: A systematic review. *Neuropsychiatric Disease and Treatment, 14*, 2905–2913. <https://doi.org/10.2147/NDT.S178839>
- Rice, K. M., & Blanchard, E. B. (1982). Biofeedback in the treatment of anxiety disorders. *Clinical Psychology Review, 2*(4), 557–577. [https://doi.org/10.1016/0272-7358\(82\)90030-7](https://doi.org/10.1016/0272-7358(82)90030-7)
- Richey, J. A., Keough, M. E., & Schmidt, N. B. (2012). Attentional control moderates fearful responding to a 35% CO₂ challenge. *Behavior Therapy, 43*(2), 285–299. <https://doi.org/10.1016/j.beth.2011.06.004>

- Roebuck, H., Freigang, C., & Barry, J. G. (2016). Continuous performance tasks: Not just about sustaining attention. *Journal of Speech, Language, and Hearing Research*, 59(3), 501–510. https://doi.org/10.1044/2015_JSLHR-L-15-0068
- Roest, A. M., Martens, E. J., de Jonge, P., & Denollet, J. (2010). Anxiety and risk of incident coronary heart disease. A meta-analysis. *Journal of the American College of Cardiology*, 56(1), 38–46. <https://doi.org/10.1016/j.jacc.2010.03.034>
- Roy-Byrne, P. P., Davidson, K. W., Kessler, R. C., Asmundson, G. J. G., Goodwin, R. D., Kubzansky, L., ... Stein, M. B. (2008). Anxiety disorders and comorbid medical illness. *General Hospital Psychiatry*, 30(3), 208–225. <https://doi.org/10.1016/j.genhosppsych.2007.12.006>
- Sandford, J. A., & Sandford, S. E. (2014). IVA-2: Integrated Visual and Auditory Continuous Performance Test Manual. North Chesterfield: VA: Brain Train Inc.
- Sandford, J. A., & Sandford, S. E. (2015). *IVA-2 Integrated Visual and Auditory Continuous Performance Test Manual*. North Chesterfield, VA: BrainTrain, Inc.
- Sari, B. A., Koster, E. H. W., Pourtois, G., & Derakshan, N. (2016). Training working memory to improve attentional control in anxiety: A proof-of-principle study using behavioral and electrophysiological measures. *Biological Psychology*, 121(Pt B), 203–212. <https://doi.org/10.1016/j.biopsycho.2015.09.008>
- Scheinost, D., Stoica, T., Saksa, J., Papademetris, X., Constable, R. T., Pittenger, C., & Hampson, M. (2013). Orbitofrontal cortex neurofeedback produces lasting changes in contamination anxiety and resting-state connectivity. *Translational Psychiatry*, 3(4), e250-6. <https://doi.org/10.1038/tp.2013.24>
- Schönenberg, M., Wiedemann, E., Schneidt, A., Scheeff, J., Logemann, A., Keune, P. M., & Hautzinger, M. (2017). Neurofeedback, sham neurofeedback, and cognitive-behavioural group therapy in adults with attention-deficit hyperactivity disorder: a triple-blind, randomised, controlled trial. *The Lancet Psychiatry*, 4(9), 673–684. [https://doi.org/10.1016/S2215-0366\(17\)30291-2](https://doi.org/10.1016/S2215-0366(17)30291-2)
- Scott, K. M. (2014). Depression, anxiety and incident cardiometabolic diseases. *Current Opinion in Psychiatry*, 27(4), 289–293. <https://doi.org/10.1097/YCO.0000000000000067>
- Shapiro, K. L., & Lim, A. (1989). The impact of anxiety on visual attention to central and peripheral events. *Behaviour Research and Therapy*, 27(4), 345–351. [https://doi.org/10.1016/0005-7967\(89\)90004-1](https://doi.org/10.1016/0005-7967(89)90004-1)
- Sherlin, L., Arns, M., Lubar, J., & Sokhadze, E. (2010). A position paper on neurofeedback for the treatment of ADHD. *Journal of Neurotherapy*, 14(2), 66–78. <https://doi.org/10.1080/10874201003773880>

- Sherlin, L. H., Arns, M., Lubar, J., Heinrich, H., Kerson, C., Strehl, U., & Sterman, M. B. (2011). Neurofeedback and basic learning theory: Implications for research and practice. *Journal of Neurotherapy*, *15*(4), 292–304. <https://doi.org/10.1080/10874208.2011.623089>
- Shibeshi, W. A., Young-Xu, Y., & Blatt, C. M. (2007). Anxiety worsens prognosis in patients with coronary artery disease. *Journal of the American College of Cardiology*, *49*(20), 2021–2027. <https://doi.org/10.1016/j.jacc.2007.03.007>
- Shin, L. M., Whalen, P. J., Pitman, R. K., Bush, G., Macklin, M. L., Lasko, N. B., ... Rauch, S. L. (2001). An fMRI study of anterior cingulate function in posttraumatic stress disorder. *Biological Psychiatry*, *50*(12), 932–942. [https://doi.org/10.1016/S0006-3223\(01\)01215-X](https://doi.org/10.1016/S0006-3223(01)01215-X)
- Smith, P. J., & Blumenthal, J. A. (2011). Psychiatric and behavioral aspects of cardiovascular disease: epidemiology, mechanisms, and treatment. *Revista Española de Cardiología*, *64*(10), 924–933. <https://doi.org/10.1016/j.recesp.2011.06.003>
- Smits, J. A. J., Julian, K., Rosenfield, D., & Powers, M. B. (2012). Threat reappraisal as a mediator of symptom change in cognitive-behavioral treatment of anxiety disorders: A systematic review. *Journal of Consulting and Clinical Psychology*, *80*(4), 624–635. <https://doi.org/10.1037/a0028957>
- Spada, M. M., Georgiou, G. A., & Wells, A. (2010). The relationship among metacognitions, attentional control, and state anxiety. *Cognitive Behaviour Therapy*, *39*(1), 64–71. <https://doi.org/10.1080/16506070902991791>
- Spaulding, T. J., Plante, E., & Vance, R. (2008). Sustained selective attention skills of preschool children with specific language impairment: Evidence for separate attentional capacities. *Journal of Speech, Language, and Hearing Research*, *51*(1), 16–34. [https://doi.org/10.1044/1092-4388\(2008/002\)](https://doi.org/10.1044/1092-4388(2008/002))
- Spector, I. P., Pecknold, J. C., & Libman, E. (2003). Selective attentional bias related to the noticeability aspect of anxiety symptoms in generalized social phobia. *Journal of Anxiety Disorders*, *17*(5), 517–531. [https://doi.org/10.1016/S0887-6185\(02\)00232-3](https://doi.org/10.1016/S0887-6185(02)00232-3)
- Spitzer, C., Barnow, S., Völzke, H., John, U., Freyberger, H. J., & Grabe, H. J. (2009). Trauma, posttraumatic stress disorder, and physical illness: findings from the general population. *Psychosomatic Medicine*, *71*(9), 1012–1017. <https://doi.org/10.1097/PSY.0b013e3181bc76b5>
- Stein, D. J., & Nesse, R. M. (2011). Threat detection, precautionary responses, and anxiety disorders. *Neuroscience and Biobehavioral Reviews*, *35*(4), 1075–1079. <https://doi.org/10.1016/j.neubiorev.2010.11.012>

- Stein, D. J., Scott, K. M., Jonge, P. de, & Kessler, R. C. (2017). Epidemiology of anxiety disorders: From surveys to nosology and back. *Dialogues in Clinical Neuroscience, 19*(2), 127–136.
- Stein, D. J., Versiani, M., Hair, T., & Kumar, R. (2002). Efficacy of paroxetine for relapse prevention in social anxiety disorder: A 24-week study. *Archives of General Psychiatry, 59*(12), 1111–1118.
<https://doi.org/10.1001/archpsyc.59.12.1111>
- Sterman, M. ., & Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalography and Clinical Neurophysiology, 33*(1), 89–95. [https://doi.org/10.1016/0013-4694\(72\)90028-4](https://doi.org/10.1016/0013-4694(72)90028-4)
- Stocchi, F., Nordera, G., Jokinen, R. H., Lepola, U. M., Hewett, K., Bryson, H., & Iyengar, M. K. (2003). Efficacy and tolerability of paroxetine for the long-term treatment of generalized anxiety disorder. *The Journal of Clinical Psychiatry, 64*(3), 250–258. <https://doi.org/10.4088/JCP.v64n0305>
- Suliman, S., Stein, D. J., Myer, L., Williams, D. R., & Seedat, S. (2010). Disability and treatment of psychiatric and physical disorders in South Africa. *Journal of Nervous and Mental Disease, 198*(1), 8–15.
<https://doi.org/10.1097/NMD.0b013e3181c81708>
- Tan, G., Thornby, J., Hammond, D. C., Strehl, U., Canady, B., Arnemann, K., & Kaiser, D. A. (2009). Meta-analysis of EEG biofeedback in treating epilepsy. *Clinical EEG and Neuroscience, 40*(3), 173–179.
<https://doi.org/10.1177/155005940904000310>
- Taylor, J. E., Poston, W. S. C., Haddock, C. K., Blackburn, G. L., Heber, D., Heymsfield, S. B., & Foreyt, J. P. (2003). Psychometric characteristics of the General Well-Being Schedule (GWB) with African-American women. *Quality of Life Research, 12*(1), 31–39. <https://doi.org/10.1023/A:1022052804109>
- Thayer, J. F., Åhs, F., Fredrikson, M., Sollers, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience and Biobehavioral Reviews, 36*(2), 747–756. <https://doi.org/10.1016/j.neubiorev.2011.11.009>
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders, 61*(3), 201–216.
[https://doi.org/10.1016/S0165-0327\(00\)00338-4](https://doi.org/10.1016/S0165-0327(00)00338-4)
- Thayer, J. F., & Sternberg, E. M. (2009). Neural concomitants of immunity—Focus on the vagus nerve. *NeuroImage, 47*(3), 908–910.
<https://doi.org/10.1016/j.neuroimage.2009.05.058>

- Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology*, *141*(2), 122–131. <https://doi.org/10.1016/j.ijcard.2009.09.543>
- Tottenham, N., & Sheridan, M. A. (2010). A review of adversity, the amygdala and the hippocampus: A consideration of developmental timing. *Frontiers in Human Neuroscience*, *3*(JAN), 1–18. <https://doi.org/10.3389/neuro.09.068.2009>
- Vogelzangs, N., Seldenrijk, A., Beekman, A. T. F., van Hout, H. P. J., de Jonge, P., & Penninx, B. W. J. H. (2010). Cardiovascular disease in persons with depressive and anxiety disorders. *Journal of Affective Disorders*, *125*(1–3), 241–248. <https://doi.org/10.1016/j.jad.2010.02.112>
- Walker, J. E. (2009). Anxiety associated with post-traumatic stress disorder—the role of quantitative electro-encephalograph in diagnosis and in guiding neurofeedback training to remediate the anxiety. *Biofeedback*, *37*(2), 67–70. <https://doi.org/10.5298/1081-5937-37.2.67>
- Williams, J. M., Tonymon, P., & Andersen, M. B. (1990). Effects of life-event stress on anxiety and peripheral narrowing. *Behavioral Medicine*, *16*(4), 174–181. <https://doi.org/10.1080/08964289.1990.9934606>
- Wilson, M. R., Vine, S. J., & Wood, G. (2009). The influence of anxiety on visual attentional control in basketball free throw shooting. *Journal of Sport and Exercise Psychology*, *31*(2), 152–168. <https://doi.org/10.1123/jsep.31.2.152>
- Wittchen, H.-U., Kessler, R. C., Beesdo, K., Krause, P., Höfler, M., & Hoyer, J. (2002). Generalized anxiety and depression in primary care: prevalence, recognition, and management. *The Journal of Clinical Psychiatry*, *63 Suppl 8*, 24–34. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/12044105>
- Wittchen, H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., Jönsson, B., ... Steinhausen, H. C. (2011). The size and burden of mental disorders and other disorders of the brain in Europe 2010. *European Neuropsychopharmacology*, *21*(9), 655–679. <https://doi.org/10.1016/j.euroneuro.2011.07.018>
- Woody, E. Z., & Szechtman, H. (2011). Adaptation to potential threat: The evolution, neurobiology, and psychopathology of the security motivation system. *Neuroscience and Biobehavioral Reviews*, *35*(4), 1019–1033. <https://doi.org/10.1016/j.neubiorev.2010.08.003>
- Zilverstand, A., Sorger, B., Sarkheil, P., & Goebel, R. (2015). fMRI neurofeedback facilitates anxiety regulation in females with spider phobia. *Frontiers in Behavioral Neuroscience*, *9*(June), 1–12. <https://doi.org/10.3389/fnbeh.2015.00148>

APPENDIX A

Table A1. Description of IVA-2 Global Quotient Scores

IVA-2 Measure	Description
FAQ	Composite scale which comprises equal weights (not an average) of the AAQ and VAQ scales
AAQ	Measures auditory inattention, loss of focus, and slow processing speed
VAQ	Measures visual inattention, loss of focus, and slow processing speed
FRCQ	Composite scale which comprises equal weights (not an average) of the AAQ and VAQ scales
ARCQ	Measures auditory response inhibition, sustaining effort, and consistent responses
VRCQ	Measures visual response inhibition, sustaining effort, and consistent responses

Table A2. Descriptive Labels for IVA-2 Standard Quotient Scores

Descriptive Label	Standard Quotient Score Range ^a
Exceptional	≥ 130
Superior	120-129
Above Average	110-119
Average	90-109
Slightly Impaired	85-89
Mildly Impaired	80-84
Mildly to Moderately Impaired	76-79
Moderately Impaired	72-75
Moderately to Severely Impaired	68-71
Severely Impaired	61-67
Extremely Impaired	≤ 60

Note. Descriptive labels provided in the IVA-2 manual. ^aScores reported in standard scores ($M = 100$, $SD = 15$).

Table A3. IVA-2 Manual Recommended Quotient Score Change Classifications

Quotient Point Differences	Difference Description
< 8	No Significant Change
8-10	Slight Change
11-18	Mild Change
19-27	Moderate Change
≥ 28	Major Change

Note. Descriptive labels provided in the IVA-2 manual.

Table A4. Descriptive Labels for General Well-being Schedule

Descriptive Label	Raw Score Range
Positive well-being	81-110
Low positive well-being	76-80
Marginal	71-75
Stress problem	56-70
Distress	41-55
Serious	26-40
Severe	0-25

Note. Descriptions provided by the National Center for Health Statistics