ANXIETY, DEPRESSION, AND SLEEP DISORDERS: THEIR RELATIONSHIP AND REDUCTION WITH NEUROTHERAPY

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This study investigated the relationship among anxiety, depression, and sleep disturbances and the treatment of these three disorders through neurotherapy. Research suggests that these conditions commonly co-occur in the general population and that central nervous system (CNS) arousal may play a primary role in the development and maintenance of these disorders. Several recent studies suggested that neurotherapy, a biofeedback-based treatment for CNS dysregulation, might be an effective treatment for comorbid conditions, particularly the ones of interest here, depression, anxiety, and sleep disturbances. This investigation used a clinical case-series design to assess pre/post neurotherapy changes on objective measures of anxiety, depression, and sleep and to determine whether changes in anxiety and depression then predict improvements in sleep quality. Data for 23 participants (10 males) were obtained from files of adults (M_{age} = 40.22 years, *SD* = 16.20) who received at least 15 neurotherapy sessions (*M* = 47.83 sessions, *SD* = 22.23) the University of North Texas Neurotherapy Lab. Matched pair *t*-tests revealed that symptoms of sleep disturbance, depression, and anxiety showed significant improvements following neurotherapy. Neurotherapy treatment effect sizes generally ranged from moderate to large (*d* = .414 – .849). Multiple regression analysis found that changes in self-reported anxiety symptoms, but not depressive symptoms, predicted observed improvements in sleep quality (adjusted R^2 = .26). Last, the implications and limitations were discussed in relation to neurotherapy practice and the associated research.

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CHAPTER I

BACKGROUND

Epidemiology and Definition of Sleep Disturbances

Insomnia as a common form of sleep disturbance represents a significant total estimated annual economic cost in the United States (Walsh & Engelhardt, 1999) and in other countries, such as Canada (Daley et al., 2009; Morin et al., 2006) and Switzerland (Buysse et al., 2008). In 1995 (i.e., the latest figures available), healthcare costs related to sleep disturbance in United States were estimated to be around 30 to 35 billion dollars (Walsh & Engelhardt, 1999). In Quebec Province of Canada, the economic burden of sleep disorders consumed approximately 1% of the gross national product (Daley et al., 2009).

When the symptoms of poor sleep interfere with an individual's life functioning, healthcare professionals may diagnose this as "Insomnia" using the following definition from the American Psychiatric Association's (2000) *Diagnostic and Statistical Manual of Mental Disorders-IV-TR*:

A. "The predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month.

B. "The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. "The sleep disturbance does not occur exclusively during the course of Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhythm Sleep Disorder, or a Parasomnia.

D. “The disturbance does not occur exclusively during the course of another mental disorder (e.g., Major Depressive Disorder, Generalized Anxiety Disorder, a delirium).

E. “The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.” (p. 604)

Disturbed sleep symptoms represented common complaints in the general population (Ohayon, 2002). Prevalence estimates varied from study to study due to non-standardized (Ohayon, 2002) or different operational definitions (Roth et al., 2006; Taylor et al., 2005) with prevalence estimates as wide ranging as 5% to 50% (Roth et al., 2006). However, a common figure reported in the literature was that approximately 33% of adults in the United States reported sleep disturbances (Ohayon, 2002, 2009; Roth et al., 2006) and another 10% to 15% met *DSM-IV* diagnostic criteria for insomnia (Roth et al., 2006) though the latter is not without dispute (Ohayon, 2002).

Insomnia prevalence rates decreased to approximately 16% to 21% when respondents report symptoms based on frequency (e.g., number of symptoms per week, etc.) and ranged from 10% to 28% when asked to describe the severity of the sleep symptoms (e.g., being bothered a lot, etc.). When daytime consequences are included in the criteria, prevalence rates dropped to approximately 10% (Ohayon, 2002). Prevalence rates of actual insomnia diagnosis (primary insomnia; insomnia disorder related to another mental disorder; sleep disorder due to a medical condition, insomnia type) ranged from 4.4% to 6.4% (Ohayon, 2002).

Sociodemographic Prevalence of Sleep Disturbance

Ohayon (2002) provided an overview of insomnia related prevalence rates across sociodemographic variables, including gender, age, marital status, income, education, and

employment. Women were 1.4 to 1.7 times more likely to report insomnia symptoms than men, with increased age associated with more problematic sleep. Additionally, women were twice as likely to receive a diagnosis of insomnia. Married couples had fewer insomnia symptoms; conversely, those who are separated, divorced, or widowed, especially if they are women, reported more sleep disturbances on average. A consistent finding in the research was that the prevalence of insomnia increased with age (Ancoli-Israel & Kripke, 1991; Ohayon, 2002; Sivertsen, Krokstad, Overland, & Mykletun, 2009), especially for respondents over 65, when the rate reached nearly 50%. However, the data was mixed regarding the stability of this finding when daytime consequences and sleep dissatisfaction ratings were taken into account (Ohayon, 2002). Data also suggested that less educated and lower income persons had more disturbed sleep. Ohayon pointed out though that these poverty prevalence rates are questionable, mainly due to limitations of research paradigms, and that these differential rates may be more related to older age than to greater poverty.

Beaulieu-Bonneau et al.'s (2007) cross sectional data suggested a possible heritability factor in predisposition for insomnia as participants, especially women, with current or past sleep disturbances were more likely to report a family history of insomnia. However, the authors admitted that additional longitudinal studies with stringent confound controls need to be conducted to make a more accurate determination about possible heritability of insomnia.

Sleep Disturbance and Co-Morbid Mental Disorders

Sleep disturbances, including insomnia, in the general population were widely reported in the scientific literature to co-occur with mental disorders (Ohaynon, 2002; Roth et al., 2006; Sateia, 2009; Sivertsen, Krokstad, Overland, & Mykletun, 2009), particularly with major depression and anxiety (Ohaynon, 2002, 2009; Riemann, 2007; Roth et al., 2006; Taylor,

Lichstein, & Durrence, 2003; Taylor et al., 2005). The National Comorbidity Survey Replication (NCS-R) (Roth et al., 2006), a survey of more than 9,200 adults, resulted in a wealth of data that pertains to sleep problems and to comorbid mental disorders. Although components of *DSM-IV* diagnostic criteria were used in the questionnaires, a unique aspect of this particular research was its focus on general sleep disturbance rather than the specific diagnostic category of just insomnia. Their survey instrument specifically investigated multiple difficulties, including difficulty initiating sleep (DIS), difficulty maintaining sleep (DMS), and early morning awakening (EMA). Non-restorative sleep (NRS), a 4th item in the survey and an apparent measure of sleep quality, queried participants about retrospective reports of trouble over the past year waking-up in the morning, waking up feeling tired, feeling the need to sleep more despite adequate sleep time in bed, and not feeling refreshed after sleep.

Overall, 36.3% of NCS-R respondents reported at least 1 sleep problem over the past 12 months. Additionally, in this study 12-month prevalence rates for DIS, DMS, EMA, and NRS were estimated at 16.4%, 19.9%, 16.7%, and 25%, respectively, and 25% of participants had 1 or more of these symptoms for an average of 22 to 29 weeks (dependent on the time frame assessed). Not surprisingly, those who reported all 4 sleep problems also had the highest mean duration of sleep disturbance. Age emerged as a significant factor with persons 18 to 29 years old 2 to 3 times more likely to report DIS and NRS than older persons, while those 60 years or higher were 1.3 to 1.7 times likely to have trouble with DMS and EMA than younger persons (Roth et al., 2006).

Further, Roth et al. (2006) found that poor sleep was highly correlated with a variety of psychological disturbances in the NCS-R. Seventeen *DSM-IV* diagnoses were investigated that included anxiety, mood, impulse-control, and substance use disorders. Approximately 50% of

persons with sleep disturbance also met criteria for at least 1 *DSM-IV* diagnostic category, and sleep problems were significantly associated with each of the 17 *DSM-IV* disorders. For example, respondents with symptoms of generalized anxiety disorder (GAD) were significantly more likely to report concomitant DIS (*Odds Ratio [OR]* = 2.9), DMS (*OR* = 3.0), EMA (*OR* = 3.2), and NRS (*OR* = 6.1), while those with a depressed mood were significantly at risk for concurrent DIS (*OR* = 3.5), DMS (*OR* = 3.9), EMA (*OR* = 2.9), and NRS (*OR* = 6.1). Sleep disturbances, particularly NRS, significantly affected the quantity or quality of work at their place of employment (Roth et al., 2006).

A relatively recent publication, the Nord-Trøndelag Health Study-2 (HUNT-2), examined associations between insomnia and physical and mental health (Sivertsen, Krokstad, Overland, & Mykletun, 2009). Approximately 48,000 (53.9% female) Norwegians aged 20 to 89 years agreed to participate. A majority of the participants were married or living with a partner (61.8%), obtained at least a high school degree (78.8%), and were employed (55.1%). All participants completed an insomnia questionnaire, a physical examination to assess for somatic conditions, chronic pain, and pain conditions with an uncertain etiology, and The Hospital Anxiety and Depression Scale. The authors noted that they investigated components of insomnia rather than insomnia per se due to the lack of information on middle-of-the night awakenings and day impairment. Hence, similar to the National Comorbidity Survey Replication, the HUNT-2 study may be viewed as an investigation into symptoms of sleep disturbance.

HUNT-2 researchers (Sivertsen, Krokstad, Overland, & Mykletun, 2009) found that an average of 13.5% of the participants reported symptoms of insomnia. Significant gender and age interactions surfaced with more sleep disturbances among women and older participants. Persons with a lower educational status reported sleep problems nearly twice as often. In the

final model, symptoms of insomnia were significantly more likely to co-occur for all physical and psychological disorders examined, except for allergies and obesity; and, of special relevance to the current investigation, psychological disorders had the strongest association with insomnia. Persons with depression were nearly twice as likely to experience poor sleep and those with anxiety were 2.5 times more likely to have sleep disturbances. Of all physical health variables assessed, only fibromyalgia had a higher association with sleep disturbances (OR = 2.68) than depression and anxiety.

That depression and anxiety were associated with poor sleep was not a conclusion unique to the HUNT-2 and the National Comorbidity Survey Replication. Taylor et al. (2005) used a cross-sectional survey design with a community based sample of 772 people. After extensive control of potential confounds, Taylor et al. estimated that participants with symptoms of insomnia were 9.82 times more likely to present with depression and 17.35 times more likely to have anxiety. Additionally, Beck Depression Inventory and State Trait Anxiety Inventory scores significantly increased coincident with more sleep complaints. Similar to the HUNT-2 study, Taylor et al. also reported gender and age differences with women and older participants reporting more frequent insomnia.

Ohayon (2002) reported that 33% of persons with insomnia complaints and dissatisfaction with sleep quality received a co-morbid mental disorder diagnosis. Even in a presumably well-educated and intelligent population of medical students, poor sleep was associated with minor psychiatric disorders, such as depressive and anxious moods and depressive thoughts (Hidalgo & Caumo, 2002).

Objective Psychological Characteristics of Poor Sleepers and the MMPI-2

Only a handful of studies that detail the psychological characteristics of poor sleepers using Minnesota Multiphasic Personality Inventory (MMPI) have been published in the past 20 years (Hauri, 1983; Kalogjer-Sackellares & Cartwright, 1997; Shirayama et al., 2003; Tsushima & Ingolfsdottir, 2004; Vgontzas et al., 2008), and only 2 of these used the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) (Tsushima & Ingolfsdottir, 2004; Vgontzas et al., 2008). The remainder of the studies utilized the original Minnesota Multiphasic Personality Inventory.

Tsushima and Ingolfdottir (2004) administered the MMPI-2 to a group of 55 males and 49 females with insomnia whose ages ranged from 18 to 87 years (M_{age} = 49 years). No clinically elevated scores (t > 64) or gender or age interactions were found on the average MMPI-2 group profile using K-corrected t-scores. However, researchers noted that a relatively high percentage of participants with clinically elevated scales. Depression (Scale 2, 41.3%), hypochondrias (Scale 1, 37.5%), hysteria (Scale 3, 37.5%), and psychasthenia (Scale 7, 33.6%) were the four most frequently occurring elevations.

Vgontzas et al. (2008) investigated the role of emotional stress in sleep disturbance and insomnia in a group of 1300 obese people that included 561 men and 739 women. All participants completed physical examination, sleep outcome measures, and the MMPI-2. Judging by the researcher's graph as no numeric data was provided, Scale 1 (hypochondrias) was the only clinically elevated Scale (t = 65) for obese persons with insomnia. All other scales for those with insomnia and sleep difficulty fell in the sub-clinical range. However, on average, obese persons with insomnia/sleep disturbance had significant elevations on each MMPI-2 clinical scale when compared to persons with similar weight related issues and no sleep problems. The four highest elevated clinical scales for those with insomnia or sleep difficulty

were hypochondrias, depression, hysteria, and psychasthenia. When the authors looked at the relation of self-reported sleep duration and stress, they found that "both BMI [body mass index] and average MMPI-2 scores were significant predictors of sleep duration. In the main effects model, the effect of MMPI-2 score was stronger than that of BMI in terms of reduction of sleep duration per 1 standard deviation increase of MMPI-2 or BMI, as well as of the p-values. No significant interaction between MMPI-2 and gender was observed." (p. 804) Obese persons with sleep complaints had higher levels of stress and commensurate increases on clinical scales within the MMPI-2 profiles.

In summary, the 2 published MMPI-2 studies that detailed the emotional characteristics of persons with insomnia and sleep disturbance appeared to converge on elevated MMPI-2 Scales 1, 2, 3, and 7. In both studies, Scales 1, 2, 3, and 7 were the 4 highest scales. More specifically, Tsushima and Ingolfdottir (2004) found that meaningful percentages of their participants had *t*-scores over 65 on these 4 MMPI-2 clinical scales. Vgontzas et al. (2008) reported 1 clinically elevated scale (Scale 1) and statistically significant differences (albeit mostly subclinical ones) on all MMPI-2 clinical scales, including Scales 1, 2, 3, and 7, between obese persons with sleep complaints and those with adequate sleep.

Theoretical Relationship between Sleep Disturbance and Mental Health

Hyperarousal Model of Insomnia

The hyperarousal model of insomnia provides a biopsychosocial or integrative approach to sleep disturbance that has received much interest and attention as of late (Bonnet & Arand, 2010; Riemann et al., 2010). The hyperarousal model of insomnia proposed an interaction among behavioral, social, psychological, biological, and pharmacological factors that play predisposing, precipitating, and perpetuating roles in sleep disturbance symptoms (Riemann et

al., 2010). At the heart of this model was the concept that the sleep system and the central nervous system (CNS) are in conflict (Bonnet & Arand, 2010). For example, researchers have directly recorded heightened cognitive activity, over-activation of cortical electrophysiology, and enhanced cognitive processing during sleep states in persons with reported poor sleep (Bonnet & Arand, 2010; Riemann et al., 2010). Riemann et al. (2010) stated that increased fast brainwave activity, especially in the beta and gamma frequency bands, during awake and/or asleep, are reported in the literature, though there were some contradictory findings.

Riemann et al. (2010) and Bonnet and Arand (2010) provided a comprehensive overview of empirical evidence for the hyperarousal theory of insomnia that covered published research from molecular genetics, brain imaging, including electroencephalography (EEG), quantitative electroencephalography (QEEG), single photon emission computed tomography (SPECT), positron emission tomography (PET), and functional magnetic resonance imaging (fMRI), as well as autonomic nervous systems, neuroendocrine systems, and neuroimmunological systems. A summary of the components of hyperarousal model of insomnia relevant to the current investigation follows below.

In the hyperarousal model of insomnia, acute insomnia was viewed to be the result of some threat in the environment, such as changes in social or medical status, which resolves for most people upon termination of the negative situation. Rumination, worry, and a focus on the symptoms of disturbed sleep may lead to chronic sleep difficulties that go on to become independent of the original stressor(s) (Riemann et al., 2010). Maladaptive behaviors may follow, such as daytime napping, substance use, and poor bed hygiene along with negative classical conditioning to the bedroom environment (i.e., bed/bedroom elicit arousal instead of de-arousal), which further perpetuates poor sleep (Riemann et al., 2010). As a result, clinically

significant psychopathology can develop over the course of extended sleep difficulties, especially anxiety and depression. Hyperarousal model of insomnia attributed the development of depression to the learned helplessness model of depression whereby a person feels helpless to make meaningful changes in sleep behaviors due to a lack of relief from attempts to improve sleep or standard therapies. This can result in dysphoria, adaptation of maladaptive behaviors, such as substance abuse, and the belief that a situation may be without possible resolution (Riemann et al., 2010).

An additional important concept in the hyperarousal model of insomnia was that short-term and long-term sleep disturbances are accompanied by excessive input from the autonomic and somatic nervous systems to cortical and subcortical brain centers that regulate sleep (Riemann et al., 2010). Persons with insomnia are reported to have excessive sympathetic activity, including increased heart rate and skin resistance, reduced parasympathetic activity, and may be also predisposed to physiological activation. When these factors are combined with environmental stressors, disturbed sleep can more easily follow (Bonnet & Arand, 2010). Moreover, CNS physiological activation (i.e., sympathetic) tended to increase with age, physical status, and medical problems (Bonnet & Arand, 2010). This observation might explain research detailed herein that sleep quality generally decreases with age.

Transdiagnostic Perspective of Psychopathology

Harvey (2008) argued that insomnia might best be viewed from a transdiagnostic perspective that was not inconsistent with the hyperarousal model of insomnia. Harvey noted that insomnia is often viewed to be epiphenomenal to or a byproduct of the primary mental disorders. However, the author concluded after a review of current research that insomnia may be a primary cause (both onset and relapse) and contributor to the maintenance of mental health disturbances,

as well as a common process (transdiagnostic) in most psychiatric disorders/diagnoses. Specifically, the author pointed out that disturbed sleep played an important role in psychiatric disorders and part of the diagnostic criterion for separation anxiety disorder; major depressive disorder; dysthymic disorder; bipolar disorder; posttraumatic stress disorder; acute stress disorder; generalized anxiety disorder; many substance withdrawals, including alcohol, amphetamine, caffeine intoxication, cocaine, nicotine, opioids, sedative-hypnotics, and anxiolytics; and, of course, for numerous sleep disorders. The author also cited research that suggested that sleep played an important role in quality of life, mood regulation, and cognitive functioning, including speed of processing and memory. Harvey stated that profound changes in public health might occur if researchers and clinicians adapt transdiagnostic approaches to research and treatment, as opposed to the current focus on single disorders.

Bioelectric Arousal Theory of Psychopathology

Othmer (2007) advocated what can best be described as a neuronal bioelectric network model of psychopathology with a strong emphasis on functional connectivity (i.e., neural frequency, amplitude, and timing), especially with thalamocortical connections, and the role of the brainstem in overall cortical arousal, timing, and frequency. This model was consistent with and, in some ways, extended the hyperarousal model and transdiagnostic perspectives of insomnia. A noteworthy observation was that the brainstem, a prominent influential structure in the neuronal bioelectric network model of psychopathology (Othmer, 2007), also played a dominant role in general arousal and sleep (Waxman, 2009).

The high degree of symptom and diagnostic overlap among disturbances of sleep and mood (anxiety and depression) suggested a common underlying biological/physiological process. As advocated in Othmer's bioelectric neural network model of psychopathology (2007), deviant

brainwave activity may play an important role, if not a unifying one, in the development and/or maintenance of psychopathology and co-morbid conditions. Said another way, psychological conditions such as insomnia, depression, and anxiety may be primarily disorders of central nervous system arousal, which has been noted elsewhere (Arns, Gunkelman, Bretleler, & Spronk, 2008; Gunkelman, 2006; Johnston, Gunkelman, & Lunt, 2005; Othmer, 2007). As is discussed below in the Introduction to Electroencephalography section, specific brainwave frequencies play an important role in cognitive, emotional, and behavioral functioning and arousal. Thus, dysfunctional electrophysiological states may create a conflict between the sleep system and central nervous system believed to be at the heart of sleep disturbances, as advocated by Bonnet and Arand (2010), given that the human brain is a functionally connected network (Othmer, 2007). The review below specifically addresses anxiety and depression in terms of abnormal EEG amplitude/frequency. Prior to this review, however, a brief explanation of basic terms used in electroencepahology will be provided.

Introduction to EEG Terminology

Brainwave activity is classified in cycles per second, or hertz (Hz). Brainwave frequencies are categorized into bandwidths that roughly coincide with subjective states of consciousness, including delta (0 to 4 Hz), theta (4 to 8 Hz), alpha (8 to 12 Hz), SMR (12 to 15 Hz), and beta (13 to 40 Hz). Briefly, slower wave forms are associated with ever increasing states of drowsiness and faster brainwaves with increased alertness and vigilance. Delta is the primary characteristic of sleep and theta can reflect a trance-like states at the low end of the theta frequency band or semi-awake, dream-like, spacey states at the higher end of the theta frequency band. Alpha is associated with states of relaxation and cortical idling. SMR is a frequency with a specific morphology that occurs over the sensori-motor cortex and is associated with a mentally

alert, yet physically calm state. Beta appears as small, fast, desynchronized brainwaves that reflect intellectual activity and perception of the environment (Hammond, 2007). Beta greater than 20 Hz (high beta) up to 30 Hz or 35 Hz is thought to be associated with anxiety, rumination, and hyper-vigilance (Demos, 2005).

EEG Patterns in Anxiety and Depression

Some of the earliest quantitative electroencephalography (QEEG) studies that investigated "EEG subtypes" of psychopathology reported abnormal electroencephalography (EEG) profiles (John, Prichep, & Almas, 1992; Prichep & John, 1992). Since that time, the EEG characteristics of anxiety and depression have been described extensively in the literature. Excessive beta, particularly high beta, in the frontal and central regions, was commonly reported to represent symptoms of anxiety in the research literature (Gunkelman, 2006; Hammond, 2005a; Johnston, Gunkelman, & Lunt, 2005) and in neurofeedback textbooks (Demos, 2005; Kropotov, 2008; Thompson & Thompson, 2003). The presence of excessive beta correlates with hyperarousal of the central nervous system, including worry, apprehension, and rumination, which triggers the flight-or-fight response of the sympathetic nervous system. Slow-wave frontal lobe disturbances, especially increased theta and alpha, are also believed to play a primary role in anxiety disorders (Hammond, 2005), such as generalized anxiety disorder and obsessive-compulsive disorder (Johnston, Gunkelman, & Lunt, 2005).

Asymmetrical EEG patterns are reported to play a role in anxiety (Heller et al., 1997; Mathersul, Williams, Hopkinson, & Kemp, 2008) and depression (Mathersul, Williams, Hopkinson, & Kemp, 2008; Yatsenko, Baas, Ponomarev, & Kropotov, 2010), and can predict development of clinically significant anxiety symptoms up to 1 year in the future (Blackhart, Minnix, & Kline, 2006) or emotional response to negative stimuli (Allen, Harmon-Jones,

Cavender, 2001). However, much attention has been given to a specific asymmetrical EEG pattern believed to underlie many depressive states, often referred to as the alpha asymmetry of depression. Alpha-asymmetry is a construct that describes a physiological state where the left frontal hemisphere's tendency for positive emotion and memories and approach/motivation characteristics are placed "on hold" (i.e., under-activated) due to excessive amounts of alpha; consequently, the right hemisphere's tendency for negative emotion and withdrawal are over-activated (Davidson, 1992; Davidson, 1995, Davidson, 1998a, Davidson, 1998b; Hammond, 2005a). From an arousal perspective, this may represent a mixed hypo- and hyper-activation with depressed mood, behavioral lethargy, and rumination.

A recent study (Yatsenko, Baas, Ponomarev, & Kropotov, 2010) that compared 111 medication-free adults with early stage depressive symptoms to 526 non-depressant participants provided extensive information about the EEG characteristics of depression. For example, participants with depressive symptoms exhibited statistically significant increased beta (14 to 20 Hz) in the left hemisphere (especially in the left sensorimotor area), increased bilateral theta (4 to 7.5 Hz), increased bilateral occipital alpha (7.5 to 14 Hz) activity, and increased alpha in the right parietal-temporal region. The authors failed to find the classic alpha-asymmetry profile described above, but stated that differences in patient populations, such as varied comorbid conditions, substance use, and medications, may account for this non-finding. Nonetheless, Yatsenko, Baas, Ponomarev, and Kropotov's research described depression as a mixed hypo- and hyper-activation state characterized by increased slow brainwaves (theta and alpha) thought to reflect typical depressive symptoms and increased fast brainwave activity believed to reflect ongoing anxiety.

The above research provided good evidence supporting the explanatory value of the hyperarousal and transdiagnostic models of sleep disturbance and associated conditions such as

anxiety and depression. If all three conditions represent instances of CNS hyperarousal or dysregulation, treatment approaches designed to reduce CNS arousal or improve dysregulated neurological patterns, such as biofeedback or neurofeedback, would seem to be logical clinical choices for treating these disorders.

The Theoretical and Practical Applications of Neurofeedback

Numerous published books and peer reviewed articles are available that describe the theoretical and practical application of neurofeedback in clinical practice (Budzynski, Budzynski, Evans, & Abarbanel, 2008; Demos, 2005; Evans, 2007; Evans & Abarbanel, 2007; Gunkelman & Johnstone, 2005; Hammond, 2007; Hammond & Kirk, 2007; Kropotov, 2008; Masterpasqua & Healey, 2003; Swingle, 2008; Thompson & Thompson, 2003). Neurofeedback, also known as EEG-biofeedback, is an approach to treating various psychological, psychiatric, and developmental disorders by training participants to actively self-regulate brainwave activity through the use of operant conditioning principles (Hammond, 2007). There is a history of research that demonstrates that people as well as animals are capable of changing their EEG pattern when given proper rewards (i.e., visual and/or auditory feedback) (Hammond, 2007).

A fundamental property of cells in the central nervous system is that all cells generate electricity through action potentials, post-synaptic inhibitory potentials, and post-synaptic excitatory potentials for neuronal communication and regulation. The brain interconnectivity of various neuronal networks mean that changes in 1 networked system can elicit changes in others (Othmer, 2007). An additional fundamental concept is that all cognitive, emotional, and physical states (adaptive or maladaptive) have cerebral electrical correlates (noting an exception of certain reflexive activity, particularly those that originate from the spinal column). Therefore, neurofeedback is based on the view that psychopathology results in part (or is at least associated)

from dysregulated neurological activity. This activity may involve deficient or excessive brainwave electrical power (magnitude or amplitude) in specific frequencies or problems in inter-cerebral communication or timing (coherence, synchrony, and phase).

In short, neurofeedback attempts to modify abnormal brainwave activity to change associated thoughts, emotions, and behaviors. The power of neurofeedback lies in its ability to establish self-directed homeostasis in a dysregulated system with the potential to ameliorate multiple or complex symptom presentations (Othmer, 2007) through gradual shaping of desired EEG patterns over successive learning trials. Of course, none of this matters if the benefits erode after neurofeedback ends; consequently, participants often attend 30 or more half-hour or longer neurofeedback sessions to over learn the desired EEG changes; thus, promoting long-term maintenance of gains.

A Description of a Typical Neurofeedback Session

A typical neurofeedback session begins when sensors are placed on a patient's head to detect faint electrical signals that are emitted from the client's brain. The brain's electrical signals are then sent to an amplifier, which literally boosts the signal, and sends it on to a neurofeedback software program for further algorithmic processing to transform the EEG signal into usable information (Hammond, 2007). After this initial set up, clients begin their neurofeedback session and may watch a graphic feedback display, play video games, listen to music, or even watch their favorite DVDs for approximately 20 to 30 minutes. The selection of a reward/feedback modality (i.e., music, video, etc.) is often based on the one that best helps the client learn how to regulate his or her brainwaves, as well as therapist and client preferences. Positive reinforcement of desired brainwave states occurs when the client fully meets performance criteria set by the neurotherapist. During positive reinforcement phases, auditory

and/or visual feedback (i.e., music, DVD, etc.) plays uninterrupted; conversely, auditory and visual feedback is withdrawn when the patient fails to meet criterion. Participants attend these sessions 2 to 5 times per week for at least 30 sessions with more severe or complex symptoms taking 40 to 100 sessions.

Research on Neurofeedback for Sleep Disturbance

Three recent studies specifically investigated the impact of neurofeedback on disturbed sleep. Cortoos et al. (2010) reported that neurofeedback training led to significant improvements in sleep for participants with insomnia. The sample represented a relatively pure insomnia group as all psychiatric or medical disorders were excluded. Participants with dysthymia or generalized anxiety were included if clearly related to their sleep disturbance. A design that integrated random assignment and blinded treatment conditions (to the participants) allowed for a neurofeedback group and an electromyography (EMG) biofeedback group to be compared to a healthy control group. Medication free participants in the neurofeedback group with 3 women and 6 men (M_{age} = 41 years) and biofeedback group with 3 women and 5 men (M_{age} = 43 years) received approximately 20 sessions of training. Twelve healthy participants made up the control group. Participants in the biofeedback group received relaxation training via reduction of EMG paced at the center of the forehead (i.e., electrode placement Fpz) along with a 50 Hz reference at the top of the head in the center (electrode placement Cz), while those in the neurofeedback group received a SMR training protocol that enhances 12 Hz to 15 Hz (SMR) and inhibits 4 Hz to 8 Hz (theta) and 20 Hz to 30 Hz (high beta) at electrode site Cz. Participants completed a number of pre/post subjective and objective assessments, such as sleep logs, bedtime diaries, EEGs (i.e., polysomnography), Pittsburgh Sleep Quality Index, Athens Insomnia Scale, Epworth Sleepiness Scale, State Trait Anxiety Index, Beck Depression Inventory, Presleep Arousal Scale

with a somatic and a cognitive subscale, and the Mini International Neuropsychiatric Interview. Total sleep time, sleep latency, wake after sleep onset, sleep efficiency, and time in bed were selected as additional outcome measures (Cortoos et al., 2010).

Cortoos et al. (2010) found that participants in the neurofeedback group reported statistically significant improvements in sleep latency (39%) and waking after sleep onset (53%) in a within-group pre/post analysis. While participants in the biofeedback condition also reported reductions in sleep latency (44%) and waking after sleep onset (13%), only participants who received neurofeedback realized additional significant improvements in time in bed, rapid eye movement (REM) sleep, and total sleep time. Small to medium effect sizes were achieved; however, modest effect sizes were not unexpected given the very small sample size for the neurofeedback group (i.e., 9 participants). Larger effect sizes could be reasonably expected with a bigger sample size (Cortoos et al., 2010)

Cortoos et al. (2010) concluded that: "Applying a NFB protocol intervening on the level of cognitive processing, may thus have had an influence on cortical arousal and information processing during sleep, resulting in an increase of TST [total sleep time]. On a more concrete level, by inhibiting high beta (20 to 30 Hz) during NFB [neurofeedback] training we tried to intervene directly on the reactivity to stress and arousal. Furthermore, Sterman et al. [1970] showed that the increase of SMR resulted in a facilitation of sleep spindle bursts and quiet sleep. This in turn might have had an influence on the consolidation of sleep in our subjects and might explain the positive impact on sleep duration that we observed." (p. 18-19)

Hoedlmoser et al. (2008) found that 10 sessions of SMR (12 to 15 Hz) neurofeedback significantly improved sleep and declarative memory in a randomized, parallel group design. The researchers created a unique study design (described in more detail below) that allowed the

experimental and control groups to receive nearly identical treatment conditions (to overcome a common criticism of many neurofeedback studies), except, of course, only 1 group received an actual treatment. A number of primary outcome measures were utilized, such as sleep onset latency times, sleep diaries, mood measures, declarative memory tasks, and polygraphic sleep recordings with sleep spindle analysis.

Hoedlmoser et al. (2008) randomly assigned 13 male and 14 female healthy subjects (M_{age} = 23.63 years; *SD* = 2.69) to receive SMR neurofeedback (experimental group) or randomized frequency neurofeedback (control group). The use of randomized frequency neurofeedback insured equal treatment of participants by exposing persons in the control group to the exact same conditions as the experimental group, except that they received reinforcement with a different EEG frequency band (excluding 12 to 15 Hz) at each session. Learning theory predicts that random reinforcement will produce little to no acquisition of the target behaviors (Skinner, 1953). Ten 1 hour neurofeedback sessions were completed over 10 days (1 per day). The neurofeedback protocol included 8, 3 minute blocks with each block containing a 3 second baseline (to calculate participant specific SMR amplitudes) followed by audio and visual reinforcement when SMR amplitudes exceeded threshold for 250ms (milliseconds) or longer (Hoedlmoser et al., 2008).

Hoedlmoser et al. (2008) found that participants benefited from SMR neurofeedback on numerous sleep and memory related outcome measures. Specifically, participants who received targeted 12 to 15 Hz neurofeedback exhibited significantly enhanced sleep spindles, increased SMR amplitudes (which demonstrates that conditioned learning occurred), decreased sleep latency onset time (effect size [*d*] =.7), and improved memory on learning exercises at post-assessment (*d* = .9) when compared to the control group. These are impressive results given that

participants received only a limited number of neurofeedback sessions (i.e., 10). The researchers concluded that neurofeedback should be considered a possible alternative treatment for primary insomnia.

A recent unpublished study by Johnson and Bodenhamer-Davis (2009) examined the impact of neurofeedback on adult sleep patterns. The authors utilized a time-series analysis to analyze archival data from 74 male and female adult participants who sought treatment for a wide range of psychological and/or behavioral conditions. Data was derived from the pre-session questionnaire which participants completed prior to each session at the University of North Texas (UNT) Neurotherapy Lab.

The pre-session questionnaire contained 17 self-report variables grouped in the following domains: sleep, mood, attention, somatic stress areas, side effects (irritability, headache), dietary intake (protein, caffeine, sugar, alcohol, medication), and relaxation efforts. Specific items related to sleep included, "How many hours did you sleep last night?", "How long did it take you to fall asleep?" and "How many times did you wake up during the night?" Participants were asked to endorse their subjective rating of these domains on a Likert scale format. Questions that pertained to sleep correlated with measures of sleep onset latency (SOL), number of awakenings/wake time after sleep onset (WASO) and total sleep time (TST).

Johnson and Bodenhamer-Davis (2009) reported findings that examined the impact of neurofeedback on sleep quality. On average, total sleep time significantly improved for most participants - even those that did not have sleep complaints at intake. Specifically, participants not reporting sleep related concerns at intake increased their average hours of sleep from approximately 6.5 hours to 8.5 hours. Participants initially presenting with sleep complaints had an increase in their average hours of sleep from approximately 6 hours to 8 hours or more. A

survival analysis related to the times series further yielded notable trends that showed that participants experienced the most gains in total sleep time during sessions 15 to 30 and that sessions beyond 50 sessions produced only minimal gains or decreases. Although Johnson and Bodenhamer-Davis did not specifically investigate changes in psychopathology, they reported that those participants who completed neurofeedback the fastest reported significant increased energy, which loaded on the Mental State construct derived from factor analysis. This suggested that changes in at least 1 factor related to psychological well-being may have been related to changes in sleep quality and ultimate treatment outcome.

In summary, neurofeedback appeared to be a relatively effective treatment for symptoms of insomnia as demonstrated in 2 controlled (Cortoos et al., 2010; Hoedlmoser et al., 2008) and 1 uncontrolled study (Johnson & Bodenhamer-Davis, 2009). Moreover, these promising results were obtained with small sample sizes (Cortoos et al., 2010; Hoedlmoser et al., 2008) and limited neurofeedback sessions (Cortoos et al., 2010). It seemed reasonable to conclude that neurofeedback may be a relatively powerful treatment for symptoms of insomnia as statistically significant improvements were obtained despite the use of small sample sizes and limited treatment sessions - factors known to limit statistical power. In addition, Johnson and Bodenhamer-Davis (2009) demonstrated that, on average, all participants' sleep improved following neurofeedback - even those without initial sleep complaints - and that changes in mental energy might play a role or at least correlate with gains in total hours slept.

Research on Neurofeedback for Depression and Anxiety

There are no published controlled outcome studies for neurofeedback for depression, and this void has been noted elsewhere (Walker, 2007; Yucha & Montgomery, 2008). Thus, the Association for Applied Psychophysiology (AAPB) rated neurofeedback for depression as

"Level 2: Possibly Efficacious" based on currently available case studies (Yucha & Montgomery, 2008). Neurofeedback for depression case studies have been reviewed by Hammond (2005a, 2005b) and Walker (2007) with generally positive outcomes reported by Baehr, Rosenfeld, & Baehr (1997), Baehr, Rosenfeld, & Baehr (2001), Baehr, Miller, Rosenfeld, & Baehr, (2004), and Baehr, Rosenfeld, Baehr, & Earnest (1999). Nonetheless, the lack of controlled outcome studies for depression was particularly surprising given the extensive amount of EEG basic research on the alpha-asymmetry (i.e., differential activation of the frontal lobes) characteristics of depression (Davidson, 1992; Davidson, 1995, Davidson, 1998a, Davidson, 1998b; Hammond, 2005a).

Researchers have published a number of controlled outcome studies for neurofeedback for anxiety-related disorders. In addition, several uncontrolled clinical case studies of neurofeedback/anxiety have been reported in the literature with generally positive outcomes (Hammond, 2005a; Hammond, 2005b; Kerson, Sherman, and Kozlowski, 2009; Moore, 2000; Moore, 2005; Walker, 2009). Neurofeedback for anxiety received an efficacy rating of "Level 4: Efficacious" (Yucha & Montgomery, 2008) by AAPB. A summary of controlled outcome studies for neurofeedback for anxiety and related co-morbid psychopathology is provided below.

Agnihotri, Paul, and Singh Sandhu (2007) randomly assigned 21 males and 24 females (age range: 18 - 30 years) with generalized anxiety disorder to 1 of 3 groups (15 participants per group): electromyography (EMG) frontalis biofeedback, EEG-biofeedback, or no treatment control. Participants in both biofeedback groups trained for 35 minutes per day for 12 days. The researchers carefully screened participants for generalized anxiety disorder via strict guidelines set by the *DSM-IV-TR*. Participants were excluded if they previously practiced relaxation techniques or took anxiolytic medication. The State Trait Anxiety Inventory (STAI) and galvanic

skin response served as the primary anxiety outcome measures. The researchers re-administered STAI and galvanic skin response measures 2 weeks after treatment in the follow-up phase of the study. The researchers reported that participants in both biofeedback groups evidenced significant improvements on measures of galvanic skin resistance and state and trait scores for anxiety. Participants in the control group exhibited no changes on any of these outcome measures. STAI state anxiety scores decreased by 34.70% and 27.91% for EMG biofeedback and EEG biofeedback, respectively, and STAI trait anxiety scores dropped by 24.34% and 17.08% for participants in the EMG biofeedback and EEG biofeedback groups, respectively. A 2 week follow-up revealed that persons in the EEG biofeedback group maintained gains in galvanic skin response and trait scores, while persons in the EMG biofeedback group retained improvements in trait scores. These results for both biofeedback groups are impressive given the relatively short treatment duration (i.e., 12 days) since generalized anxiety disorder is known to take a severe, chronic, and treatment refractory course (Brown, O'Leary, & Barlow, 2001).

Vanathy, Sharma, and Kumar (1998) randomly assigned 14 male and 4 female medication-free participants (M_{age} = 32 years) with chronic anxiety (M_{years} = 8.61) to either an EEG alpha/beta group, EEG biofeedback theta/beta group, or a wait-list control group. Not to be confused with alpha-theta neurofeedback described below, the protocols called for either increased alpha (8.5 to 12 Hz) or theta (4 to 8 Hz) with a reduction of beta (12.5 to 16 Hz) in the occipital region across 15, 30-minute neurofeedback sessions over 4 weeks. Inclusion criteria called for relatively pure cases of generalized anxiety disorder as persons with co-morbid conditions, such as obsessive-compulsive disorder, panic attacks, and substance abuse, were excluded from this study. Outcome measures included self-report measures in the State-Trait Anxiety Inventory (STAI) and Global Quality of Life Questionnaire (GQL), blinded observer

anxiety ratings via the Hamilton Anxiety Rating Scale (HARS), and pre/post EEG spectral analysis.

Vanathy, Sharma, & Humar (1998) reported significant reductions of self-reported state anxiety (STAI) and observer-reported manifest anxiety (HARS) for both EEG biofeedback groups in a between group comparison. Participants in the EEG biofeedback theta/beta group also evidenced significant improvement on the quality of life measure (GQL). No differences were found in trait anxiety on the STAI. In within-group comparisons, persons in the EEG alpha/beta group realized significant improvements in self-reported trait anxiety (STAI) and observer-reported manifest anxiety (HARS), while those in the EEG biofeedback theta/beta biofeedback group reported significantly decreased state anxiety (STAI) and observer-reported manifest anxiety (HARS). Participants in the wait-list control group evidenced statistically significant increased state anxiety. The researchers concluded that, "The results of the present investigation indicated clear cut effects of alpha and theta neurofeedback training in subjects with GAD." (p. 141)

Sarkar, Rathee, and Neera (1999) examined the effectiveness of EEG biofeedback and pharmacotherapy to treat symptoms of generalized anxiety disorder. Thirty eight male and 12 female participants (age range: 20 to 55 years) were randomly assigned to an EEG biofeedback group or a pharmacotherapy group with a total of 25 participants per group. The Hamilton Anxiety Rating Scale (HARS) served as a primary objective outcome measure (along with other projective assessment measures). Sarkar, Rathee, & Neera only reported within-group statistical results. Participants in both groups realized statistically significant decreases in their HARS scores. HARS scores decreased from 15.76 to 5.53, on average, for those who received EEG

biofeedback, while those who took medication reported average HARS score reductions from 20.44 to 5.2.

A number of clinical trials, often with random assignment, comparison groups, and partial or complete blind conditions, have been published in the field of substance abuse that involve neurofeedback for co-morbid psychological conditions, including anxiety and depression (Burkett, Cummins, Dickson, & Skolnick, 2005; Callaway, & Bodenhamer-Davis, 2008; deBeus, 2007; Peniston & Kulkosky, 1989; Peniston & Kulkosky, 1990; Peniston & Kulkosky, 1991; Peniston, Marrinan, Deming, & Kulkosky, 1993; Scott & Kaiser, 1998; Scott, Kaiser, Othmer, & Sideroff, 2005). Although specific sleep measures were not reported in these studies, it is known that persons with post-traumatic stress disorder (PTSD), crack-cocaine dependence, alcoholism and other serious mental disorders often experience poor sleep, especially since many of these diagnostic labels include disturbed sleep as 1 criterion. In fact, *DSM-IV-TR* (2000) states that potential symptoms of PTSD include distressing dreams and difficulty falling or staying asleep and that substance abuse disorders, such as alcohol and cocaine withdrawal and caffeine intoxication, include insomnia or other sleep related symptoms.

Eugene Peniston conducted a now classic series of neurofeedback studies with Vietnam War veterans diagnosed with alcohol abuse and co-morbid post-traumatic stress disorder (PTSD) (Peniston & Kulkosky, 1989; Peniston & Kulkosky, 1990; Peniston & Kulkosky, 1991; Peniston, Marrinan, Deming, & Kulkosky, 1993). Peniston and Kulkosky (1989) randomly selected 20 participants from a Veterans' Administration alcohol treatment unit, and randomly assigned them to 1 of 2 groups: EEG biofeedback group or traditional alcohol treatment control group. Ten additional non-alcoholic participants were selected randomly from the same VA medical center to serve as another control group. All participants completed a variety of outcome measures that

included the Beck Depression Inventory (BDI) and Million Clinical Multiaxial Inventory (MCMI). Peniston and Kulkosky published positive results after a 15 session alpha-theta biofeedback treatment protocol. Only persons in the EEG-biofeedback group experienced statistically significant reductions on the Beck Depression Inventory (in addition to positive changes in substance abuse behaviors). In fact, participants who completed neurofeedback realized a 50% decrease in BDI scores and their post-treatment scores were comparable with the non-alcoholic group.

In 1990, Peniston and Kulkosky presented additional data from 1989 study not previously published pertaining to pre-post MCMI scores. The researchers found that those who presented with substance abuse had co-morbid psychopathology at pre-assessment as evidenced by base rate scores over 70 on the Drug Abuse, Alcohol Abuse, Anxiety, and Dysthymia clinical scales at pre-assessment. They reported that persons in the EEG-biofeedback group showed statistically significant reductions on 13 scales, including the Dysthymia, Psychotic Depression, and Anxiety. Those in the traditional alcohol treatment group had significant improvements on the Avoidant and Psychotic Thinking Scales and a significant negative increase on the Compulsive Scale. Peniston and Kulkosky stated that participants had "fundamental changes in alcoholic personality variables following alpha-theta EEG biofeedback" (p. 37).

In 1991, Peniston and Kulkosky published the results of a separate, but similar neurofeedback for anxiety study at the same VA facility that involved 29 Vietnam War veterans diagnosed with PTSD and ongoing nightmares and/or flashbacks. Participants were randomly assigned to an EEG biofeedback group or a traditional medical care group (medication plus individual and group therapy). A neurofeedback protocol nearly identical to that used in their 1989 study was employed. The Minnesota Multiphasic Personality Inventory (MMPI) served as

the primary outcome measure, and the study included a 30 month follow-up period. Participants in the EEG biofeedback group achieved statistically significant reductions on the MMPI clinical scales that included a 35 point *t*-score reduction on the Depression Scale and a reduction of approximately 17 *t*-score points on the Psychasthenia Scale (i.e., commonly associated with anxiety or excessive worry), as well as on numerous other clinical scales. Those in the traditional medical treatment group showed decreases only on the Schizophrenia Scale. At 30-month follow-up, all persons (14 out of 14) in the traditional medical treatment group relapsed compared to 3 of 15 in the EEG biofeedback group.

Several years later, Scott and Kaiser (1998) discovered that participants with poly-substance (i.e., cocaine and methamphetamine) at an in-patient facility demonstrated marked improvement when an SMR-beta protocol (i.e., 2 to 7 Hz inhibit; 12 to 15 Hz enhance, and 22 to 30 Hz inhibit) was added to the traditional alpha/theta protocol advocated by Peniston and Kulkosky (1989). Their modified protocol became known as the "Scott and Kaiser Modification of the Peniston Protocol." Scott, Kaiser, Othmer, and Sideroff (2005) subsequently published results of this modified alpha-theta neurofeedback protocol with a group of poly-substance abusers that included pre/post changes on the MMPI. All test administrators were blinded to the participants' group status. One hundred twenty volunteers were randomly assigned to an EEG-biofeedback plus conventional treatment group (60 participants) (experimental group) or conventional treatment only group (60 participants) (control group). Nearly 50% of participants in the control group dropped from treatment compared to 25% percent in the experimental group. The experimental group, compared to the control group, had statistically significant changes on the MMPI clinical scales, including Depression, Hypochondriasis, Hysteria, Schizophrenia, and Social Introversion. A 12-month follow up of treatment completers revealed a 77% abstinence

rate for those who received EEG biofeedback compared to a 44% abstinence rate for those who received conventional treatment only.

Burkett, Cummins, Dickson, and Skolnick (2005) used the Scott and Kaiser Modifications of the Peniston Protocol at a faith-based inpatient treatment facility for homeless persons with crack cocaine dependency. Although the participants were specifically treated for substance abuse, statistically significant and clinically meaningful pre/post changes emerged on the Beck Depression Index (BDI) and Clinical Anxiety Scale (CAS) at treatment completion and at a 12-month follow-up. Specifically, BDI scores improved 65% at treatment completion and 70% at 12-month follow-up and the CAS improved 54% at treatment completion and 43% at 12-month follow-up. The researchers reported 49% total abstinence from crack cocaine, 40% partial relapse (crack cocaine used 1 to 9 times), and 10% full relapse after 1 year. Additionally, significant improvements in treatment retention rates, establishment and maintenance of a regular residence, and holding a steady job or attending school, and reduced re-arrest rates were found.

DeBeus (2007) randomly assigned 7 participants to a QEEG-guided neurofeedback group, 6 participants to a researched "symptom-based neurofeedback" protocol (i.e., Scott and Kaiser Modifications of the Peniston Protocol or "Scott/Peniston"), and 6 participants to a wait list-control group. All participants were diagnosed with substance abuse. Co-occurring diagnoses found in these groups of participants included anxiety disorders (PTSD, obsessive compulsive disorder [OCD], and social anxiety disorder) and major depression. The Personality Assessment Inventory (PAI) served as 1 of many primary outcome measures that were administered at pre/post intervals by a therapist blinded to participants' assigned group. Participants in both the QEEG-guided neurofeedback group and the Scott/Peniston group

realized statistically significant improvements on the PAI Anxiety Scale (along with significant improvement on substance abuse scales) with large effect sizes of 1.07 and .80, respectively, compared to the wait-list controls.

The deBeus study (2007) was particularly applicable to the current investigation because it included a similar population of heterogonous participants with co-morbid psychopathology, especially sleep disturbance, anxiety, and depression, along with some complaints of substance abuse. deBeus utilized a similar QEEG-guided neurofeedback approach to treatment used at UNT Neurotherapy Lab; hence, the results of this study provide some insight into the treatment effect sizes that might be expected in the current proposed investigation. The fact that large effect sizes were achieved using extremely small groups (i.e., 6 to 7 participants) suggests that QEEG-guided neurofeedback may be a powerful treatment for these co-morbid psychopathologies. Moreover, it seemed reasonable to conclude that QEEG-guided neurofeedback might significantly impact co-morbid depression with a larger sample size given that participants in the deBeus study realized a .80 standard deviation improvement.

In summary, neurofeedback has been shown to be an effective treatment of depression and anxiety that occur in the presence of other co-morbid conditions in a variety of treatment settings using valid and reliable objective instruments, including the BDI and MMPI-2. In many of the studies described above, participants with known treatment refractory conditions (i.e., PTSD, substance abuse, crack cocaine dependence, etc.) benefitted from EEG biofeedback when compared to a control group or to standard medical treatment.

Rationale for Using Neurotherapy for Sleep Disorders, Depression, and Anxiety

Neurofeedback represents not only a promising treatment for sleep disorders, but especially for co-morbid symptom presentations. This is because neurofeedback treatments

generally, and QEEG-guided neurofeedback in particular, aim to correct imbalances or abnormalities in brain electrical activity identified through statistical comparisons to a normative EEG database. Since the treatment tends to affect multiple interconnected brain systems, improvements may be seen across a number of different symptom measures following treatment, as described in the research above. In short, neurofeedback tends to establish greater homeostasis in connected cerebral networks, producing transdiagnostic cognitive, emotional, and behavioral changes.

Additionally, neurotherapists often combine other forms of (peripheral) biofeedback, such as heart-rate variability (HRV) or temperature biofeedback, with EEG biofeedback, along with some supportive counseling for at least a few sessions. Many clients receive this multi-modal biofeedback treatment throughout their therapy. Each form of biofeedback in this treatment model has the effect of reducing excessive sympathetic nervous system, or flight or fight, activity. A multi-modal approach to treatment using 1 or more peripheral forms of biofeedback plus a central form (EEG biofeedback) may be collectively referred to as neurotherapy.

Purpose of the Current Investigation

The purpose of this study was to investigate the relationship of anxiety and depression to sleep disorder symptoms and the outcomes of neurotherapy for these conditions with outpatient adult clients of UNT Neurotherapy Lab. This investigation expanded on Johnson and Bodenhamer-Davis' (2009) neurofeedback/sleep study to include pre/post changes in sleep quality using an objective and reliable measure in the Pittsburg Sleep Quality Index (PSQI) (Buysse et al., 1989). At the time of the original Johnson and Bodenhamer-Davis study, the PSQI was not regularly administered at post-assessment intervals at the out-patient facility.

Hence, too few participants completed the PSQI to allow for a more objective measure of sleep outcomes. Since that time, however, more clients completed the PSQI at pre- and post-assessment phases. In the current study, the few clients with PSQIs from 2000-2008 (i.e., used in the original study) were combined with more recent ones.

This investigation further assessed pre-post changes in psychological symptoms of adult participants with disturbed sleep using self-report objective measures (Beck Depression Inventory-II, Beck Anxiety Inventory, Beck Hopelessness Scale, and Minnesota Multiphasic Personality Inventory-2). Published research left little doubt that poor sleep may be a risk factor for, contributor to, or consequence of other psychological conditions, particularly anxiety and depression (Harvey, 2008; Roth et al., 2006; Sateia, 2009; Taylor et al., 2005).

Additionally, the current study investigated whether significant pre/post changes on 4 MMPI-2 clinical scales (Scale 1 [Hypochondrias], Scale 2 [Depression], Scale 3 [Hysteria], and Scale 7 [Psychasthenia]) predicted post-assessment changes in PSQI scores. One possibility implied, but not directly addressed in Johnson and Bodenhamer-Davis' (2009) research due to a limitation of the design, was that the increased energy reported by clients resulted from positive changes in mood (e.g., remittance of depression, anxiety, etc.) which facilitated improved sleep quality. Hence, this study attempted to determine whether changes in objectively measured psychopathology via the MMPI-2 predicted changes in sleep quality in participants who receive neurotherapy.

Last, the current study investigated symptoms of insomnia (sleep disorder) rather than insomnia as a diagnostic category. Diagnostic labeling of clients was not routinely practiced in this clinic as neurofeedback generally does not rely on diagnosis for treatment. Nonetheless, the

PSQI captured many symptoms required to diagnose Insomnia as listed (see page 1 of this paper) in the *DSM-IV-TR* (American Psychiatric Association, 2000).

Research Hypotheses

Hypothesis 1. Post-treatment scores on the Pittsburgh Sleep Quality Index will be significantly lower ($p \leq .05$) than the pre-treatment scores in a matched-pairs comparison.

Hypothesis 2. Post-treatment mean scores on the Beck Depression Inventory-II will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 3. Post-treatment mean scores on the Beck Anxiety Inventory will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 4. Post-treatment mean scores on the Beck Hopelessness Scale will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 5. Post-treatment mean scores on the MMPI-2 Clinical Scale 1 will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 6. Post-treatment mean scores on the MMPI-2 Clinical Scale 2 will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 7. Post-treatment mean scores on the MMPI-2 Clinical Scale 3 will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 8. Post-treatment mean scores on the MMPI-2 Clinical Scale 7 will be significantly lower ($p \leq .05$) than pre-treatment mean scores in a matched-pairs comparison.

Hypothesis 9. Statistically significant changes on MMPI-2 Clinical Scales 1 (Hypochondrias), 2 (Depression), 3 (Hysteria), and 7 (Psychasthenia) will predict as a group of predictors statistically meaningful changes in sleep quality as measured by the Pittsburgh Sleep Quality Index.

CHAPTER II

METHODS

Participants

G*Power (Faul, Erdfelder, Buchner, & Lang, 2009; Faul, Erdfelder, Lang, & Buchner, 2007) power analysis with a matched-pairs *t*-test suggested that 27 participants were required to achieve 80% experimental power assuming a medium (d = .5) treatment effect size at the .05 significance level.

Participant inclusion criteria included individuals of any ethnicity or gender who are age 18 or older with:

1. At least 15 neurofeedback sessions completed
2. Pre-treatment Pittsburgh Sleep Quality Index with a score greater than 5
3. Post-treatment Pittsburgh Sleep Quality Index
4. Pre and post-treatment Minnesota Multiphasic Personality Inventory-2

Participant exclusion criteria were:

1. Any person under the age of 18
2. Any person who received more than 5 cranial electrotherapy sessions during the course of their treatment
3. Any person that did not sign a consent to allow their treatment information to be used for research or educational purposes (i.e., standard form presented to clients who start therapy at the UNT Neurotherapy Lab)

Data for 23 participants (10 males) were obtained from client files of adults (M_{age} = 40.22 years, SD = 16.20) who received neurotherapy at UNT Neurotherapy Lab between 1995 and 2010. Participants completed an average of 47.83 (SD = 22.23) neurotherapy sessions.

Approximately 96% of participants (22 of 23) reported at least 2 or more clinically meaningful symptoms and 11 of 23 (47%) participants described some form of sleep disturbance. Frequency of presenting symptoms were varied with primary anxiety (14), or anxiety related disorders, such as panic attacks (2) and post-traumatic stress disorder (PTSD) (1), the most frequently reported followed by depression (12), sleep (11), attention/concentration (11), and cognitive issues (i.e., slowed speed of processing and memory) (7). See Table 1 for a complete list of all reported symptoms.

Apparatus and Materials

Outcome Measures

The Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989) is a valid, reliable, and frequently used (Backhaus et al., 2002; Okun et al., 2009) brief objective self-report measure of overall sleep quality and good versus bad sleepers (Okun et al., 2009). The PSQI is a 9-question, 19-item self-report instrument designed to measure sleep quality and disturbance over a 1 month period (Carpenter & Andrykowski, 1998). PSQI questions 1 to 4 request specific respondent information that is filled in by hand, such as customary bed time and length of time to fall asleep. PSQI Questions 5 to 8 are answered on a 0-3 scale with 0 indicating no symptom presence and 3 representing symptom presence 3 or more times the past week. Question 9 is answered on a 0-3 scale with 0 meaning very good and 3 representing very bad (Carpenter & Andrykowski, 1998). All scores are combined according to the scoring criteria included with the form to produce a Global PSQI Score. Scores above 5 indicate clinically relevant levels of disturbed sleep. Cronbach's alpha coefficient produced an average internal consistency reliability estimate of .80 for the Global PSQI Score across numerous patient populations with a variety of different physical ailments (Carpenter & Andrykowski, 1998). Additionally, the PSQI is more highly

correlated with sleep problems (r = .69 - .77) than with different constructs (i.e., discriminant validity), such as mood symptoms and depression (r = .22 - .65) (Carpenter & Andrykowski, 1998). Backhaus et al. (2002) reported an average Global PSQI Score test-retest reliability correlation coefficient of .87 in a group of 80 participants with primary insomnia over a test-retest interval of 2 days to 2 weeks. Additionally, a Global PSQI Score above 5 resulted in a sensitivity of 98.7% and specificity of 84.4% to persons with sleep disturbances versus controls (Backhaus et al., 2002). It is noteworthy that PSQI captures almost all of *Diagnostic and Statistical Manual of Mental Disorders-IV-TR (DSM-IV-TR)* (American Psychiatric Association, 2000) diagnostic criteria for insomnia described on page 1. However, data available for the current archival study did not permit verification that all diagnostic criteria were met, especially D and E.

The Minnesota Multiphasic Personality Inventory-2 (MMPI-2) was first published in 1989 and consists of 567 forced choice true/false items (Graham, 2000). The MMPI-2 is the most widely used clinical objective adult personality inventory that consists of 10 clinical scales, 7 validity scales, 15 content scales, and a number of various supplementary scales with a focus on psychiatric, psychological, neurological, and or physical symptoms (Groth-Marnat, 2003). The MMPI-2 normative sample consisted of a diverse group of 1138 men and 1462 women living in the United States and demographic factors, including for minority groups, paralleled the 1980 census data (Graham, 2000).

The MMPI-2 is generally accepted as a valid and reliable objective measure of personality. One week test-retest estimates for men range from .77 to .84 for the validity scales (L, F, and K) and .67 to .92 for the clinical scales, while test-retest estimates for women range from .69 to .81 for the validity scales (L, F, and K) and .58 to .91 for the clinical scales. The

majority of test-retest reliability estimates fell in the .7 to .8 range. For men and women, Scale 6 (Paranoia) exhibited the lowest test-retest reliability, while Scale 10 (Social Introversion) obtained the highest score (Graham, 2000). Internal consistency estimates for the validity and clinical scales range from low to acceptable for both men (.34 - .85) and women (.37 - .87). Graham acknowledges that some scales have low internal consistency, but also points out that this is consistent with the MMPI's original empirical keying design and that little concern was given to internal consistency. Graham reported acceptable convergent and discriminate validity for the MMPI-2 clinical scales in his review of 2 studies that correlated partner's behavioral ratings and the clinical scales, and psychologists and psychiatrists' ratings after patient observation and interview and the clinical scales.

The Beck Anxiety Inventory (BAI) is a brief (5 to 10 minutes) 21-item adult (ages 17 or older) self-report instrument designed to measure common symptoms of anxiety that have occurred over the past week. All questions are answered on a scale of 0-4, with 0 indicating no symptom presence and 4 representing severe levels (*I could barely stand it*) of the symptom. The BAI provides 4 qualitative criteria of anxiety based the participant's total score that include minimal (0 - 7), mild (8 - 15), moderate (16 - 25), and severe (26 - 63). One week test-retest stability estimates revealed that the BAI possesses acceptable reliability (.75). Estimates of internal consistency using Cronbach coefficient alpha ranged from .92 - .94 (Beck & Steer, 1990).

The Beck Depression Inventory-II (BDI-II) is a 21-item self-report adolescent (ages 13 or older) and adult instrument designed to measure common symptoms of depression that have occurred over the past 2 weeks, such as anhedonia, fatigue, guilt, and pessimism. Administration requires approximately 5 to 10 minutes. All questions are answered on a scale of 0-3, with 0

indicating no symptom presentation and 3 representing frequent symptom occurrence. The BDI-II provides 4 qualitative criteria of depression based the participant's total score that include minimal (0 - 13), mild (14 - 19), moderate (20 - 28), and severe (29 - 63). One week test-retest stability estimates conducted with a small sample of out-participants suggested that the BDI-II is a highly reliable instrument (.93). Estimates of internal consistency using Cronbach coefficient alpha ranged from .92 to .93 (Beck, Steer, & Brown, 1996).

The Beck Hopelessness Scale (BHS) is a brief 20-item true-false objective measure of pessimism and attitudes toward the future. Items are scored 0 or 1 with a maximum of 20 points (Beck, Weissman, Lester, & Trexler, 1974). The BHS possessed a reliability coefficient of .93 as determined by a coefficient alpha (KR-20) analyses. The reliability sample consisted of a population of 294 hospitalized participants with recent suicide attempts. Item-total correlation coefficients ranged from .39 to .76 for all items on the BHS. Concurrent validity using clinician ratings correlated with total BHS scores in 2 different samples resulted in estimates of .74 with out-patients in a general medical practice and .62 with an attempted suicide population. The BHS was found to moderately correlate with other measures of depression (i.e., Semantic Differential Test and Beck Depression Inventory) with estimates in the .60's (Beck, Weissman, Lester, & Trexler, 1974).

Procedure

Study Design

This investigation was an uncontrolled clinical case series using archival data and intended to examine the relationship of anxiety and depression to sleep disorder symptoms and the outcomes of neurotherapy for these conditions with outpatient adult clients of UNT Neurotherapy Lab. The study design included multiple pre-test/post-test matched-pairs analyses

and 1 forward stepwise multiple regression. I tested the predictions that significant changes on self-report measures of sleep quality (PSQI), anxiety (BAI), depression (BDI-II), hopelessness (BHS), and psychopathology (MMPI-2) will occur after at least 15 sessions of neurotherapy. I also tested the hypothesis that statistically significant improvements on select scales of the MMPI-2 predicted changes in sleep quality. Additionally, although a control condition was not possible, it was thought that this clinical case series might provide data to answer the question of whether there is a relationship between clinical conditions (i.e., anxiety, depression, and sleep disorders) that can be classified as central nervous system dysregulation syndromes and whether these conditions might therefore respond as a group to psychophysiological self-regulation methods such as neurotherapy.

Statistical Analyses

Prior to analysis, all data were entered into SPSS 17 for Windows (SPSS Inc., 2008) and examined for missing values, extreme values, and overall accuracy. Relevant variables were converted into a histogram to visually confirm normality. Descriptive statistics were presented for all relevant demographic variables including participants' age, gender, and number of neurofeedback sessions.

As can be concluded from the review of neurofeedback research above, only a handful of studies provided adequate data to allow for specific effect size predictions in this current investigation. Cortoos et al. (2010) reported small to medium effect sizes in a very small group (n = 9) of participants that received neurofeedback for sleep disturbance, while Hoedlmoser et al. (2008) reported medium to large effect sizes for reduction in sleep latency onset time. deBeus (2007) reported large effect sizes for changes in anxiety in subjects who abused substances (n = 19) and who received 2 forms of neurofeedback.

In light of the research reviewed herein, the expectation for at least moderate treatment effect sizes for all variables under investigation seemed reasonable since the treatment outcomes described above are for variables similar to those in this investigation. Thus, a moderate effect size (Cohen's d = .50) was used for all predictions.

To test hypotheses 1 through 8, matched-pairs *t*-tests using an a-priori .05 significance level were run to determine whether significant pre/post changes occurred following neurotherapy (independent variable) on measures of sleep (PSQI), depression (BDI), anxiety (BAI), hopelessness (BHS), and select MMPI-2 Scales (dependent variables). Ninety-five percent confidence intervals and Cohen's d effect sizes were provided for all matched-pairs *t*-tests. To test Hypothesis 9, a forward stepwise multiple regression (Mertler & Vannatta, 2005) was run with all statistically significant (i.e., determined from the matched-pairs *t*-tests) MMPI-2 Scales as the independent variables (IVs) and PSQI scores as the dependent variable (DV). Difference scores obtained between pre- and post-intervals on relevant scales were specifically utilized in the forward stepwise regression analysis. Forward stepwise regression was considered a more stringent procedure than backward regression due to its reliance on the exclusive use of statistically significant variables (p = .05) (Meyers, Gamst, & Guarino, 2006). Bivariate correlations were calculated among the IV's and the DV. The IV with the highest correlation with the DV was entered first into the analysis and its contribution (i.e., R^2) to the DV was assessed. The next variable entered was the IV that contributed most to the prediction of the DV after the effects of the first variable were partialed out. This effect of the second variable was measured by the increase in R^2. Additional variables were entered until at some point they failed to make significant contributions to the prediction of the DV (Mertler & Vannatta, 2005).

Tolerance parameters (i.e., colinearity and multi-colinearity) and suppressor variables were assessed for their impact on the multiple regression model (Meyers, Gamst, & Guarino, 2006).

Additionally, in accordance with Erceg-Hurn & Mirosevich (2008), robust statistical methods, particularly non-parametric analyses, were employed as appropriate when data failed to meet the underlying statistical assumptions (e.g., normality, homogeneity of variance, and linearity). Additionally, effect size estimates and confidence intervals were provided when appropriate.

CHAPTER III

RESULTS

Data Inspection

Matched-Pairs t-*Tests*

An initial visual inspection of the Beck Anxiety Index (BAI), Beck Depression Index, and Beck Hopelessness Scale (BHS) data identified the presence of possible outlier variables on each measure with the potential to undermine the assumption of a normal distribution (see Figures 1, 2, and 3). Formal statistical testing using the Shapiro-Wilk test ($p \leq .05$) confirmed visual analysis of non-normal distributions for these measures. Thus, additional non-parametric analyses using the related samples Wilcoxon signed ranks test ($p \leq .05$) were conducted for each of these outcome measures to control for the observed non-normal distributions and are discussed in more detail below. The Pittsburgh Sleep Quality Index (PSQI) appeared to satisfy underlying statistical assumptions, including normality of distribution, as evidenced visual examination (Figure 4) and statistical confirmation via the Shapiro-Wilk analysis. As a result, non-parametric statistical methods were not employed for this analysis. An initial visual inspection of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) Scales 1, 2, 3, and 4 suggested the possibility non-normal distributions for each scale. However, the Shapiro-Wilk test ($p \leq .05$) found all scales, except post-scale 3, to have statistically normal distributions. Thus, additional non-parametric analysis using the related samples Wilcoxon signed ranks test ($p \leq .05$) was conducted for MMPI-2 Scale 3.

Further inspection of the Beck Depression Inventory Scales found that 3 of the pre-test forms and 4 of the post-test forms were completed using the original Beck Depression Inventory (BDI-I). Beck, Steer, Ball, and Ranieri (1996) compared the BDI-I to the BDI-II in a sample of

psychiatric outpatients. Each scale contains 21 items with total score range of 0 to 63. Questions on each scale are very similar with generally moderate to strong correlations with specific exception to 3 new items introduced in the BDI-II. Beck, Steer, Ball, and Ranieri reported a .93 correlation between the total score of the BDI-I and BDI-II which equated to a 2.01 mean total score difference. Based on this statistical data, it was decided to combine BDI-I and BDI-II scores for the matched-pairs *t*-test since both scales contain the same number of items with identical score ranges combined with a relatively high correlation between total scores. Nonetheless, an additional statistical analysis using only the BDI-II was completed to provide an unbiased estimate of pre/post changes on this measure.

Multiple Regression

An initial inspection of the data (i.e., difference scores) related to the multiple regression analysis revealed that the data reasonably conformed to normality, linearity, and colinearity assumptions as discussed by Mertler and Vannatta (2005). As a result, non-parametric statistical methods were not employed for this analysis.

Although all original hypotheses made uni-directional predictions, all statistical procedures utilized 2-way analyses to allow for the possibility that participants' symptoms may have worsen over the course of treatment, as well as to provide a more conservative and less biased estimate of treatment effects.

Data Analysis

Hypothesis 1

The average post-treatment score on the PSQI was predicted to be significantly lower ($p < .05$) than the average pre-treatment score in a matched-pairs comparison. A paired sample *t*-test was conducted to test this hypothesis. Results indicated that the mean post-PSQI score ($M =$

6.96, *SD* = 3.24) was significantly lower than the mean pre-PSQI score (*M* = 9.65, *SD* = 3.11), *t*(22) = 3.43, *p* = .002, 95% CI [1.07, 4.33], *d* = .85.

Hypothesis 2

The average post-treatment score on the combined Beck Depression Inventory I and Beck Depression Inventory II (BDI-I/II) was predicted to be significantly lower ($p < .05$) than the average pre-treatment mean score in a matched-pairs comparison. A paired sample *t*-test was conducted to test this hypothesis. Results indicated that mean post-BDI-I/II score (*M* = 6.95, *SD* = 8.24) was significantly lower than the mean pre-BDI-I/II score (*M* = 15.05, *SD* = 10.69), *t*(19) = 4.34, $p < .001$, 95% CI [4.19, 12.01], *d* = .849. Non-parametric analysis using the related samples Wilcoxon signed ranks test revealed that median differences between post-BDI-I/II and pre-BDI-I/II scores were significant (*p* = .001).

Additional paired sample *t*-test was conducted to test the above hypothesis utilizing only BDI-II measures. Results indicated that mean post-BDI-II score (*M* = 7.67, *SD* = 8.84) was significantly lower than the mean pre-BDI-II score (*M* = 15.00, *SD* = 11.00), *t*(14) = 3.84, $p < .002$, 95% CI [3.23, 11.43], *d* = .74. Non-parametric analysis utilizing only BDI-II measures with the related samples Wilcoxon signed ranks test found that median differences between post-BDI-II and pre-BDI-II scores were significant (*p* = .006). Thus, the BDI-II (alone) or BDI-I/II (combined) produced statistically significant results, and will be collectively referred to hereafter as BDI-II.

Hypothesis 3

The average post-treatment mean score on the BAI was predicted to be significantly lower ($p < .05$) than the average pre-treatment mean score in a matched-pairs comparison. A paired sample *t*-test was conducted to test this hypothesis. Results indicated that the mean post-

BAI score (M = 8.62, SD = 6.31) was not significantly lower than mean pre-BAI score (M = 11.71, SD = 8.31), $t(20) = 1.66$, $p = .112$), 95% CI [-.79, 6.98], $d = .419$. Non-parametric analysis using the related samples Wilcoxon signed ranks test found that median differences between post-BAI and pre-BAI scores were not significant ($p = .152$).

Hypothesis 4

The average post-treatment mean score on the Beck Hopelessness Scale was predicted to be significantly lower ($p < .05$) than the average pre-treatment mean score in a matched-pairs comparison. A paired sample t-test was conducted to test this hypothesis. Results indicated that the mean post-BHS score (M = 4.00, SD = 4.80) was significantly lower than the mean pre-BHS score (M = 6.18, SD = 4.85), $t(10) = 3.88$, $p = .003$, 95% CI [.91, 3.45], $d = .452$. Non-parametric analysis using the related samples Wilcoxon signed ranks test found that median differences between post-BHS and pre-BHS scores were significant ($p = .007$).

Hypothesis 5

The average post-treatment score on the MMPI-2 Clinical Scale 1 was predicted to be significantly lower ($p < .05$) than the average pre-treatment mean score in a matched-pairs comparison. A paired sample t-test was conducted to test this hypothesis. Results indicated that the post-MMPI-2 Scale 1 score (M = 55.74, SD = 18.28) was significantly lower than the pre-MMPI-2 Scale 1 score (M = 62.30, SD = 10.35), $t(22) = 2.08$, $p = .050$, 95% CI [.01, 13.11], d = .44.

Hypothesis 6

The average post-treatment score on the MMPI-2 Clinical Scale 2 was predicted to be significantly lower ($p < .05$) than the average pre-treatment mean score in a matched-pairs group comparison. A paired sample t-test was conducted to test this hypothesis. Results indicated that

the post-MMPI-2 Scale 2 score (M = 58.52, SD = 11.96) was significantly lower than pre-MMPI-2 Scale 2 score (M = 68.30, SD = 13.16), $t(22) = 4.84$, $p < .001$, 95% CI [5.59, 13.97], $d = .78$.

Hypothesis 7

The average post-treatment mean score on the MMPI-2 Clinical Scale 3 was predicted to be significantly lower ($p < .05$) than the average pre-treatment score in a matched-pairs comparison. A paired sample t-test was conducted to test this hypothesis. Results indicated that the post-MMPI-2 Scale 3 score (M = 56.87, SD = 14.53) was not significantly lower than the pre-MMPI-2 Scale 3 score (M = 62.13, SD = 10.55), $t(22) = 1.91$, $p = .070$, 95% CI [-.46, 10.98], $d = .41$. However, non-parametric analysis using the related samples Wilcoxon signed ranks test found that median differences between post-MMPI-2 Clinical Scale 3 and pre-Clinical Scale 3 scores were significant ($p = .039$).

Hypothesis 8

The average post-treatment mean score on the MMPI-2 Clinical Scale 7 was predicted to be significantly lower ($p < .05$) than the average pre-treatment mean score in a matched-pairs comparison. A paired sample t-test score was conducted to test this hypothesis. Results indicated that the mean post-MMPI-2 Scale 7 score (M = 56.78, SD = 13.34) was significantly lower than pre-MMPI-2 Scale 7 score (M = 67.00, SD = 11.49), $t(22) = 4.71$, $p < .001$, 95% CI [5.72, 14.72], $d = .82$.

Hypothesis 9

A forward stepwise regression analysis was run to determine whether changes in pre- and post-MMPI-2 Scales 1, 2, 3, and 7 were predictive factors of change in pre- and post-PSQI. The variable entered in step 1 was the change between pre- and post-Scale 7, which obtained a multiple R of .54, $F(1, 21) = 8.61$, $p = .008$ and an adjusted R^2 of .26. The variable entered into

step 2 was the change between pre- and post-Scale 3, step 3 contained the change between pre- and post-Scale 1, and step 4 contained the change between pre- and post-Scale 2. In the model, change in MMPI-2 pre- and post-Scale 7 (B = .20, p = .008, 95% CI [.06, .33]) was a significant predictor of change in pre- and post- PSQI. However, changes in pre- and post- Scale 3 (B = -.15, p = .512), pre- and post-Scale 1 (B = -.14, p = .51), and pre- and post-Scale 2 (B = -.30, p = .117) scores were not significant predictors of change in pre- and post- PSQI scores.

Figures 5 and 6 provide a graphical summary of mean change from pre- to post-intervals for each measure.

CHAPTER IV

DISCUSSION

Summary

In summary, 23 participants (10 males) who received an average of 48 neurotherapy sessions reported, on average, statistically significant symptom changes on numerous self-report psychological measures that included Pittsburgh Sleep Quality Index (PSQI), Beck Depression Inventory-II (BDI-II), Beck Hopelessness Scale (BHS), and Minnesota Multiphasic Personality Inventory-2 (MMPI-2) Scales 1 (Hypochondrias), 2 (Depression), and 7 (Psychasthenia). Thus, Hypotheses 1, 2, 4, 5, 6, and 8 were supported (discussed in more detail below). MMPI-2 Scale 3 showed statistically significant improvement in the non-parametric analysis, but not the parametric analysis. Thus, Hypothesis 7 was partially supported. Treatment effect sizes across multiple measures generally ranged from moderate to large, which was similar to published research by Hoedlmoser et al. (2008) and deBeus (2007). A multiple regression analysis significantly predicted changes on the PSQI using select scales of the MMPI-2 following neurotherapy, which supported Hypothesis 9 (discussed in more detail below).

General Implications

The current results provided support to Johnson and Bodenhamer-Davis' (2009) findings that neurofeedback significantly and positively impacted sleep with at least 15 neurofeedback sessions. Johnson and Bodenhamer-Davis also reported changes on a Mental State construct (i.e., increased energy) following neurofeedback. Although their Mental State construct was based on an original pre-treatment questionnaire and not part of the current study, it seemed reasonable to conclude that increased energy might follow the remittance or reduction of depression and anxiety. In the current investigation, symptoms of depression and anxiety

evidenced significant reductions at the group level during post-treatment assessment; thus, this suggested that improved mental energy may be related to treatment outcome. However, this deduction reflects only a preliminary hypothesis and no causal determination of this relationship, if any, can be made due to limitations of this study design.

Participants with disturbed sleep and apparent co-morbid psychopathology, particularly anxiety and depression as measured by the MMPI-2, responded at the group level to psychophysiological self-regulation treatment methods collectively referred to as neurotherapy. This positive response to treatment gave added support to existing neurofeedback research discussed earlier that suggested that many common mental disorders may be related to underlying central nervous system dysregulation. The current findings also provided indirect evidence for Othmer's (2007) neuronal bioelectric network model of psychopathology which states that deviant brainwave activity plays a primary role in the development and/or maintenance of psychopathology. Othmer also believed that therapies which target cortico-electrical dysregulation may have the potential to elicit changes in multiple symptom presentations due to cortical and subcortical functional connectivity, and, the limitations of the current study design notwithstanding, significant improvement across a number of symptom domains was observed in this study.

The current findings provided support to the substantial body of research (Ohaynon, 2002; Ohaynon, 2009; Roth et al., 2006, Riemann, 2007; Roth et al., 2006; Sateia, 2009; Sivertsen, Krokstad, Overland, & Mykletun; Taylor et al., 2003; Taylor et al., 2005) that reported a high rate of comorbidity of poor sleep quality and symptoms of anxiety and depression as there was relatively high percentage of participants with disturbed sleep that was accompanied by clinically significant depression (15 of 23 or 65%) and anxiety (13 of 23 or

56%) (as measured by the MMPI-2). Moreover, a review of pre-treatment mean scores on MMPI-2 Scales 1, 2, 3, and 7 found that only the primary depression (Scale 2, $t = 68.30$) and general anxiety (Scale 7, $t = 67.00$) scales were clinically elevated ($t > 65$). Other self-report measures of anxiety and depression (Beck Depression Inventory-II, Beck Anxiety Index, and Beck Hopelessness Scale) also evidenced significant, clinically meaningful mean scores at pre-treatment.

Implications Related to Specific Outcome Measures

Sleep

Although significant changes in the pre/post-PSQI mean score (Hypothesis 1) were numerically small ($M_{diff} = 2.7$), this represented a greater than one-half standard deviation improvement as compared to the original PSQI normative sample (Buysse et. al, 1989) and a medium to large treatment effect size. Observed statistical changes on the PSQI may have the potential to translate into clinically meaningful and observable changes in client self-reported sleep behaviors and sleep quality. This point is further reinforced by the fact that post-PSQI mean score of 6.96 was less than 1 point away from the normal sleep criterion (i.e., less than 6). In summary, this finding suggested that neurotherapy significantly and positively impacted participants' overall sleep quality.

Depression

Mean pre-treatment depression scores on BDI-II, BHS, and MMPI-2 Scale 2 collectively documented the presence of statistically and clinically meaningful depression in this participant sample. Participants reported significant improvements in depressive symptoms as measured by changes in the mean pre/post-scores on the BDI-II (supported Hypothesis 2), BHS (supported Hypothesis 4), and MMPI-2 Scale 2 (supported Hypothesis 6). Changes on MMPI-2 Scale 2

reflected an almost 1 standard deviation of improvement and a moderate to large treatment effect size. Similar improvement was found on the BDI-II with a moderate to large treatment effect size, while BHS scores reflected a small to medium treatment effect size. Participants, on average, completed neurotherapy with non-significant (i.e., MMPI-2) and minimal (i.e., BDI-II) levels of depressive symptoms. As such, participants generally entered treatment with significant depressive symptoms and ended treatment asymptomatic.

Anxiety

Neurotherapy's impact on measures of anxiety produced mixed results. Significant reductions in symptoms of anxiety following treatment were found on MMPI-2 Scales 1 and 7 (supported Hypotheses 5 and 8, respectively), as well as on Scale 3 (non-parametric analysis only; partially supported Hypothesis 7); however, no statistical improvements were achieved with the BAI (failed to support Hypothesis 3). A closer examination of the BAI and MMPI-2 Scale 7 was performed in an effort to reconcile these seemingly disparate findings on the 2 most similar measures of general anxiety. The BAI contains a high number questions (15 of 21) related to physical/bodily components of anxiety. Scale 7, on the other hand, focuses on psychological turmoil, such as anxiety, worry, fears, apprehension, and difficulty concentrating, though it also contains some items related to physical functioning. Perhaps differences in outcome on MMPI-2 Scale 7 and BAI occurred because they measure somewhat different components of anxiety. Additionally, BAI mean pre-treatment scores were qualitatively rated as mild. This suggested a low degree of initial distress at pre-treatment as measured by this construct, and thus more limited changes took place due to a restricted range. Another primary difference between the BAI and MMPI-2 is that the BAI is an "obvious" measure of anxiety, while the MMPI-2 is a "subtle" one. A major strength of the MMPI-2 is that it does not rely on

question content to score the clinical scales (called empirical keying), which allows for the subtle detection of unreported, over-reported, or under-reported symptoms. In this scenario, participants in the current investigation may have rated symptoms of distress higher at post-treatment when using a questionnaire with obvious content (BAI). Last, many biofeedback treatment modalities specifically train clients to become more sensitive and attentive to changes in autonomic nervous system and psychomotor functions. Participants may have reported a higher degree of physical symptoms (versus emotional and cognitive) at the BAI post-assessment phase due to heightened awareness of their body as a result of biofeedback training.

Prediction of Changes in Sleep Quality Based on Changes in Mental Status

Three of 4 (1, 2, and 7) hypothesized MMPI-2 scales documented statistically significant improvements on parametric matched-pairs analyses and were eligible for inclusion into the multiple regression analysis. In addition, MMPI-2 Scale 3 evidenced statistically significant changes on the non-parametric related samples analysis, and was therefore also included in the multiple regression analysis. Only MMPI-2 Scale 7 remained, however, in the predictive model after forward stepwise procedures. Thus, select MMPI-2 scales as a group of predictors provided support for Hypothesis 9. MMPI-2 Scale 7 accounted for a relatively robust 25% of variance observed in mean pre/post changes in PSQI scores. As a result, an important finding of this study is that change in mental state specific to reduced anxiety predicted changes in sleep quality.

An interesting observation was that of all outcomes measures examined in this study, MMPI-2 Scale 7 content most closely resembles the explanations for disturbed sleep as advocated by the hyperarousal theory of insomnia (Bonnet & Arand, 2010; Riemann et al., 2010). As previously discussed, the hyperarousal theory of insomnia poses a biopsychosocial process whereby normal sleep is thought to be a largely automatic process whereby the sleep

system and the central nervous system (CNS) are in conflict during insomnia episodes. Acute sleep difficulties are thought to be triggered by situational stressors that result in increased cognitive activity, such as an intense focus on symptoms, rumination, worry, and autonomic arousal. These factors, combined with classical conditioning, poor sleep hygiene, and other maladaptive behaviors, as well as a biological predisposition toward insomnia, lead to chronic sleep difficulties. Many items of MMPI-2 Scale 7 capture similar problem areas as presented by the hyperarousal theory of insomnia that include psychological turmoil, anxiety, tension, uncontrolled thoughts, fatigue, insomnia, and physical complaints. That only change on MMPI-2 Scale 7 predicted improvement in sleep may provide some support for the hyperarousal theory of insomnia. The hyperarousal theory of insomnia and transdiagnostic theories (Harvey, 2008) of insomnia also predict the presence of depression in persons with poor sleep, and that is what was found in this study as evidenced by the clinical elevations on multiple measures of depression. However, that only MMPI-2 Scale 7 predicted changes on the PSQI after neurotherapy suggested that clinicians should first focus their treatments on resolving symptoms of anxiety if sleep is a primary presenting concern.

Implications for Neurofeedback

The hyperarousal theory of insomnia posits that heightened cortical activation seen in faster frequency bands, including beta and gamma, was common to persons with disturbed sleep. As mentioned previously, beta reflected cognitive activity and perception of the environment (Hammond, 2007), while beta greater than 20 hertz (Hz) (called high beta) up to 30 Hz or 35 Hz was thought to be associated with anxiety, rumination, and hyper-vigilance (Demos, 2005). A noteworthy observation based on my clinical experience and years of work at the UNT Neurotherapy Lab was that this clinic routinely adds a 20 to 30 Hz inhibit to many treatment

protocols unless clinically contraindicated. As discussed by Cortoos et al. (2010), a high-beta inhibit might turn out to be a primary contributor to the significantly reduced anxiety and subsequent gains in sleep quality.

Future Studies

The current study investigated select MMPI-2 clinical scales that were derived from a review of existing published data. Future studies may be able to derive a more comprehensive prediction model using additional MMPI-2 clinical scales. In addition, there were surprisingly few MMPI-2 studies that detailed the psychological characteristics of persons with insomnia. Future studies are needed to build common psychological profiles of persons with disturbed sleep. Doing so may also help practitioners refine therapeutic approaches to patients with primary sleep complaints by guiding the selection of appropriate treatments.

Randomized controlled investigations are needed in general and to specifically determine which components of the multi-modal treatment approach commonly used in biofeedback/neurofeedback clinics and private practices contribute to observed changes in sleep, depression, and anxiety. An additional valuable contribution of controlled outcome studies would be to determine whether quantitative electroencephalography (QEEG)-guided neurofeedback versus symptom-based neurofeedback, namely sensori-motor rhythm (SMR)-neurofeedback, provides the best treatment efficacy for depression, anxiety, and sleep disturbances. From a technical perspective, comparison of QEEG-guided neurofeedback with and without 20 to 30 Hz inhibits may allow for further refinement of treatment protocols and better illuminate the mechanism of action in observed changes following neurotherapy.

Limitations and Confounds

The primary benefit of a clinical case series is to examine treatments as actually utilized in clinical practice, but these designs also suffer from a number of confounds. History and maturation effects represent significant confounds in the current investigation as the lack of a control group makes it impossible to determine if participants improved due to treatment or simply to the passage of time (i.e., regression to the mean, the ability to self-heal, historical events, etc.) Next, although all participants received neurofeedback as a part of a multi-modal treatment (neurotherapy), no conclusions can be reached regarding causality of observed improvements or which component of treatment (neurofeedback, peripheral biofeedback, talk therapy, etc.) accounted for symptom changes.

This study suffered from a selection bias due to inclusion criteria that called for participants who completed at least 15 sessions. This essentially eliminated treatment drop-outs, presumably due to non- or limited-treatment response or other factors. Thus, outcomes of this study should only be generalized to those who complete at least 15 neurotherapy sessions.

An additional possible confound was exclusive reliance on the PSQI as the sole indicator of sleep disturbance. Although this measure exhibited excellent sensitivity and specificity in published research (Buysse et al., 1989), the possibility existed that some clients with significant sleep disturbance were omitted from analysis due to a low PSQI score.

The current study included a relatively small number of participants, which limited its generalization to other populations and decreased overall statistical power. Despite the probable reduction in statistical power, participants in this study realized significant symptom improvements across a number of domains. Small sample sizes are also prone to non-normal distributions, and this was found on the BDI, BAI, and BHS. Nonetheless, distribution free, non-

parametric analysis provided increased confidence in the parametric analyses as significant findings were once again obtained for several scales. One statistical limitation in this study was that multiple-regression analysis was restricted to only 4 of 10 available clinical scales on the MMPI-2. Inclusion of other scales may have significantly impacted the final multiple-regression model. Last, I did not personally collect data from participants, and although all scores were verified for accuracy, I relied on the data as administered and collected by the original therapists.

Table 1

Self-Report Symptoms in a Sample of 23 Participants at an Outpatient Neurotherapy Clinic

Symptom Classification	**Frequency**	**Percent**
Anxiety / Irritable / Rumination	14	61%
Depression	12	52%
Disturbed Sleep	11	48%
Attention / Concentration	11	48%
Cognitive (Speed of Processing / Memory)	7	30%
Substance Abuse	6	26%
Social Problems	3	13%
Fatigue	3	13%
Panic Attacks	2	9%
Traumatic Brain Injury	2	9%
Migraine / Headache	2	9%
Anger	1	4%
Binge Eating	1	4%
COPD	1	4%
Fibromyalgia	1	4%
Immune Disorder	1	4%
Mood Swings	1	4%
Post-Traumatic Stress Disorder	1	4%
Prostate Cancer	1	4%
Sexual Addiction	1	4%
Sexual Identity Issues	1	4%
Note: Total number of symptoms exceeds actual sample size due to co-morbid symptom presentations.		

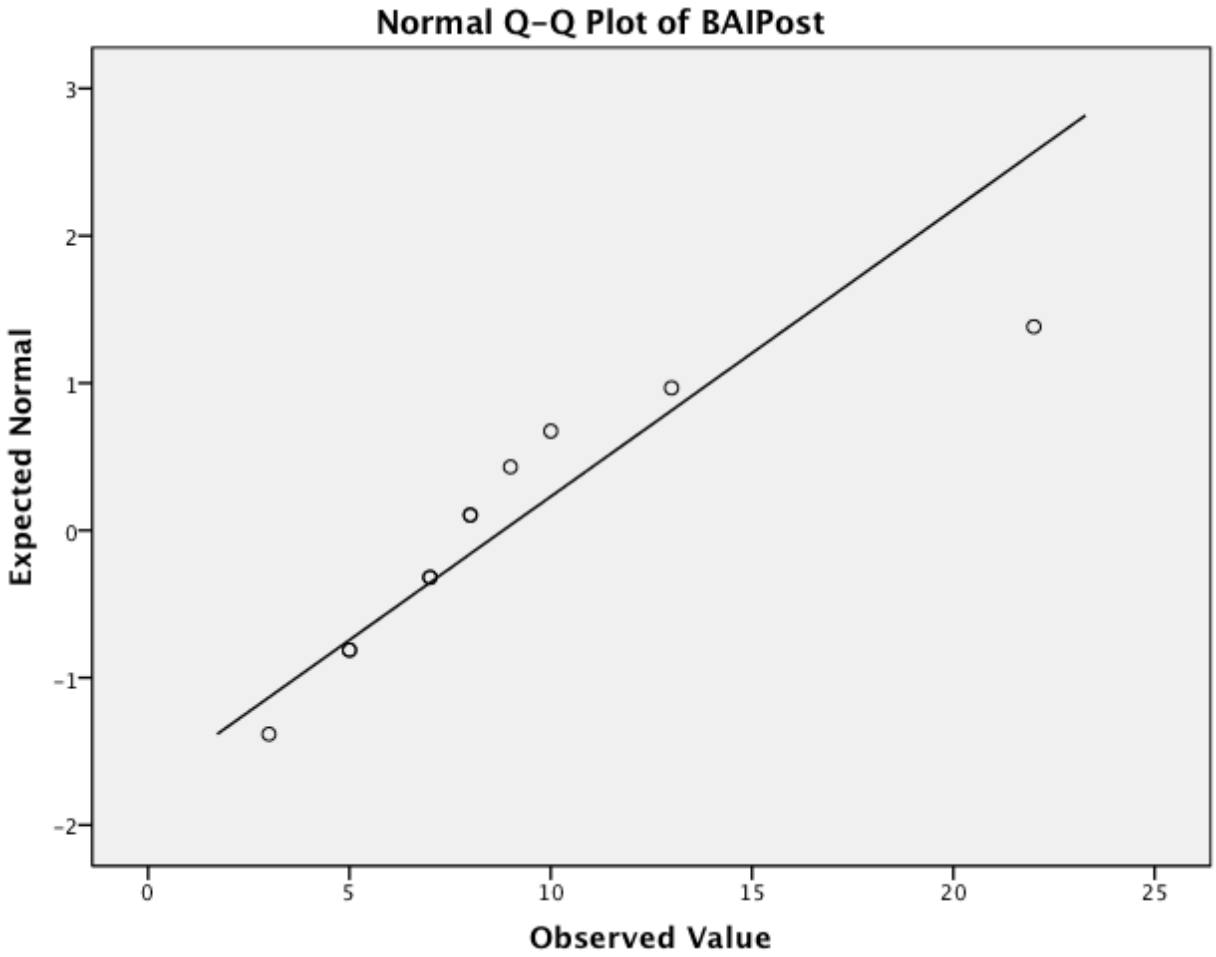

Figure 1. Visual examination of the distribution of Beck Anxiety Inventory post-assessment scores. Additional statistical analysis revealed significant deviation from a normal distribution. (Figure adapted from SPSS 17.)

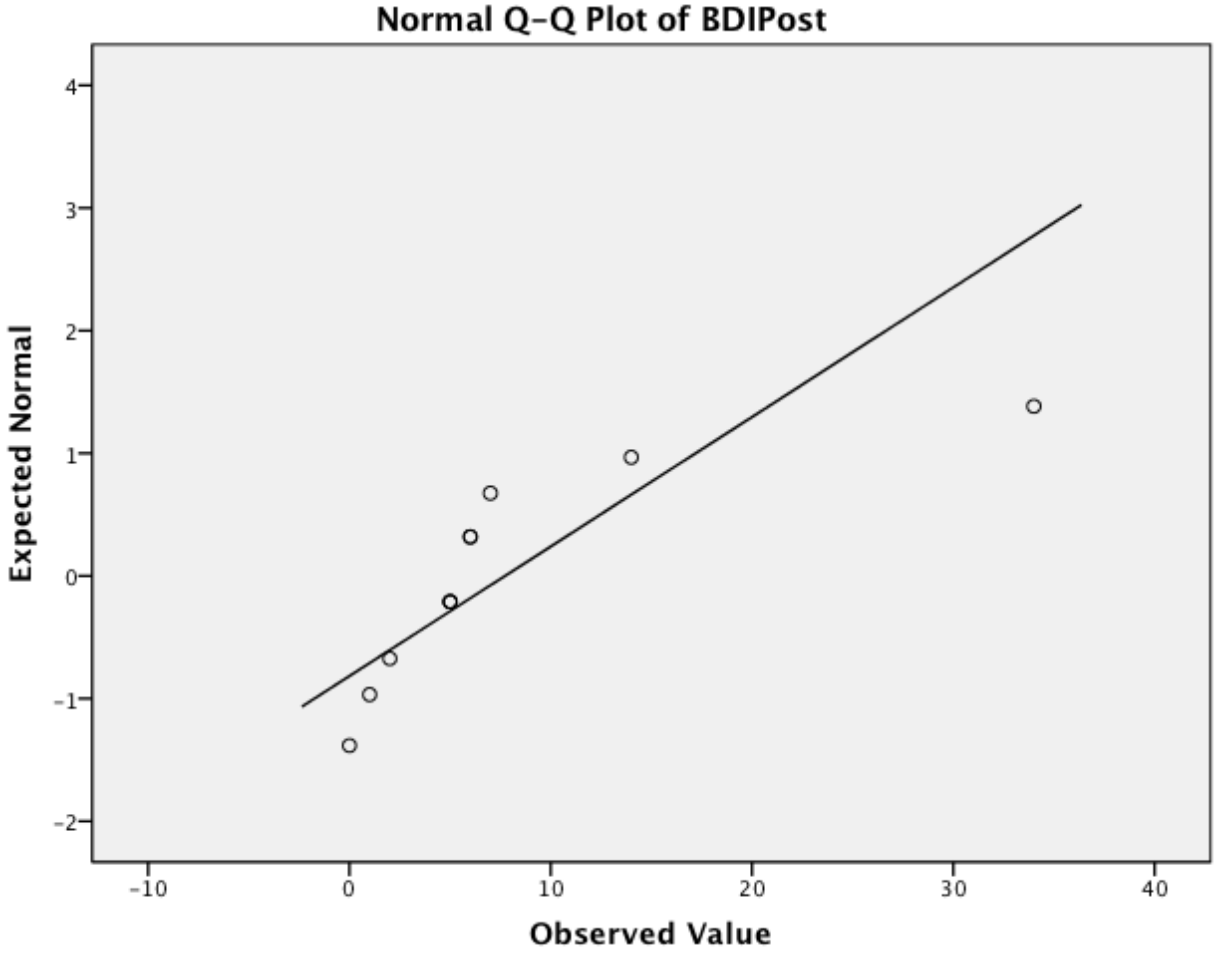

Figure 2. Visual examination of the distribution of Beck Depression Inventory-II post-assessment scores. Additional statistical analysis revealed significant deviation from a normal distribution. (Figure adapted from SPSS 17.)

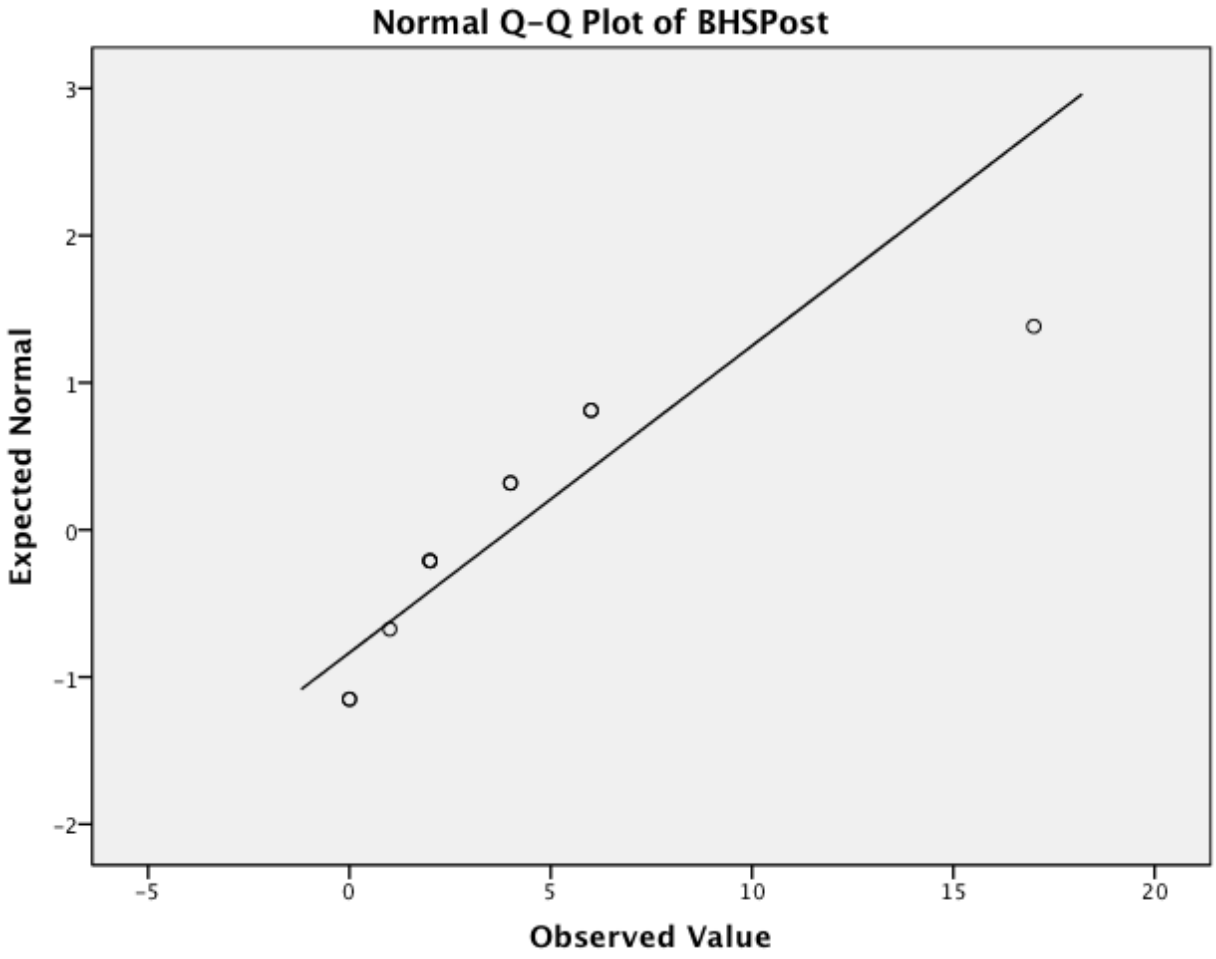

Figure 3. Visual examination of the distribution of Beck Hopelessness Scores post-assessment scores. Additional statistical analysis revealed significant deviation from a normal distribution. (Figure adapted from SPSS 17.)

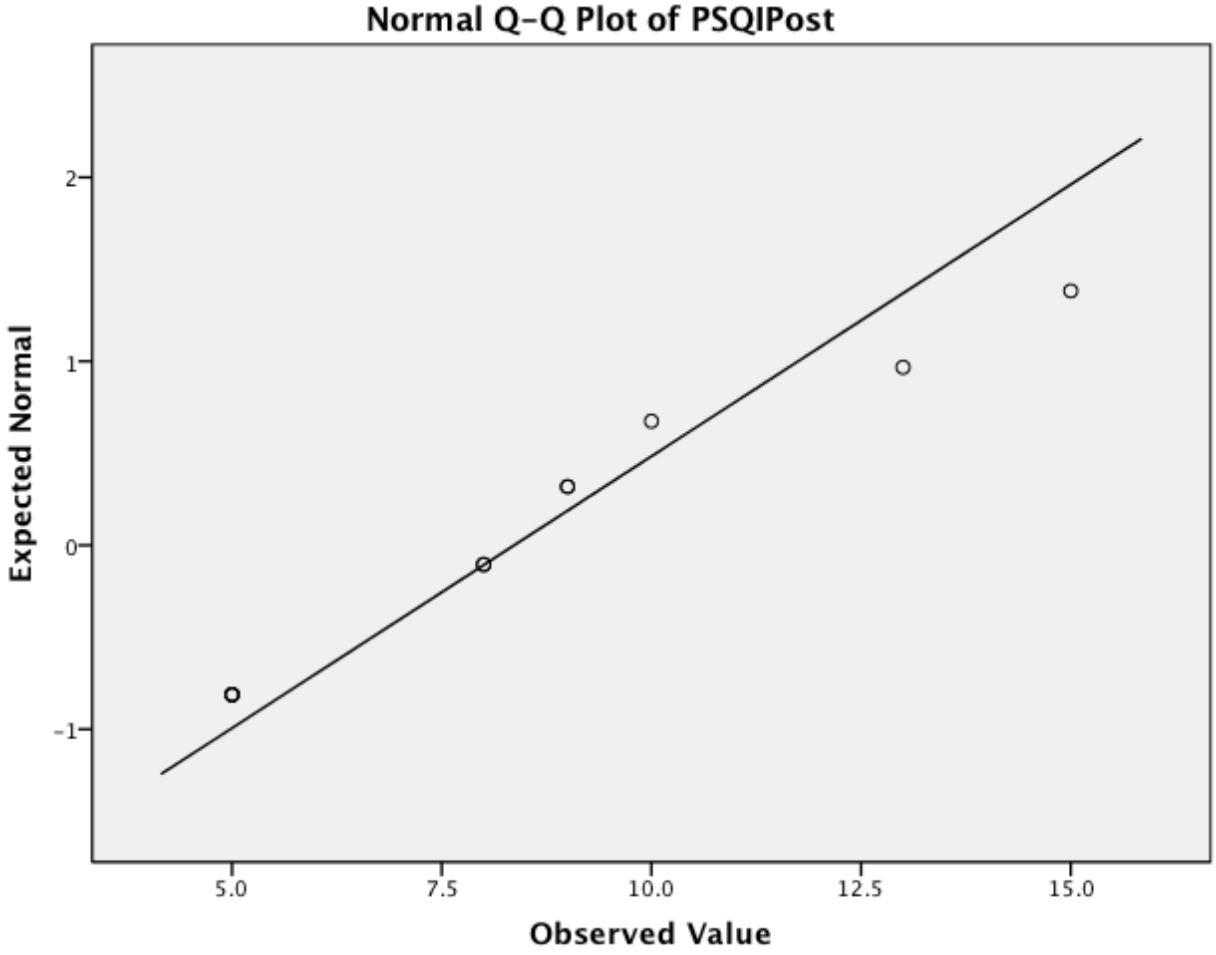

Figure 4. Visual examination of the distribution of Pittsburgh Sleep Quality Index post-assessment scores. Additional statistical analysis did not find significant deviation from a normal distribution. (Figure adapted from SPSS 17.)

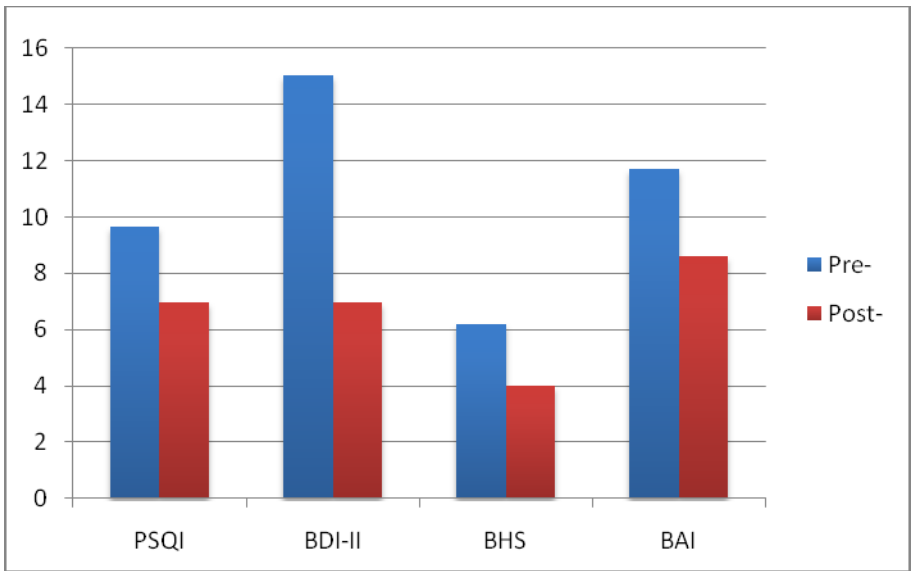

Figure 5. Changes from pre- to post-intervals on brief self-report measures in a population of outpatients who sought neurotherapy. Self-reported symptoms as measured by the PSQI, BDI-II, and BHS realized statistically significant changes in the parametric matched-pairs measures *t*-tests ($p < .05$). (Figure adapted from SPSS 17.)

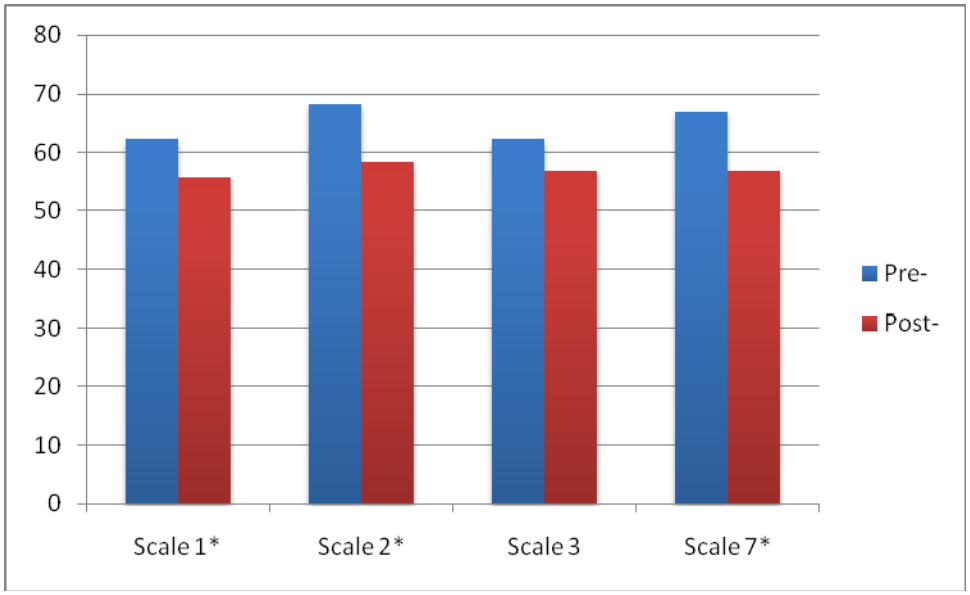

Figure 6. Changes from pre- to post-intervals on select MMPI-2 Scales in a population of outpatients who sought neurotherapy. Participant symptoms as measured by MMPI-2 Scales 1, 2, and 7 realized statistically significant changes in the parametric matched-pairs *t*-tests (*$p < .05$ level). (Figure adapted from SPSS 17.)

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