

UTILIZING NEUROFEEDBACK FOR THE TREATMENT OF INSOMNIA: A
FEASIBILITY STUDY

A DISSERTATION PRESENTED TO THE FACULTY
OF THE CALIFORNIA SCHOOL OF PROFESSIONAL PSYCHOLOGY AT
ALLIANT INTERNATIONAL UNIVERSITY, SAN DIEGO

In partial fulfillment of
the requirements for the degree of
Doctor of Philosophy

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2019

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PREVIEW

Abstract

Insomnia is a significant public health problem impacting individual's daily life (Straten, Zwerde, Kleiboer, Cuijpers, Morin & Lancee, 2018). In fact, approximately 33%-50% of the adult US population has suffered from symptoms of insomnia (Ancoli-Israel, 1999). Despite insomnia's devastating effects, current treatments such as medications and cognitive behavioral therapy have major limitations. These include side effects, marginal efficacy rates in the case of medications, or the continuation of some impairment following CBT-I treatment (Longo & Johnson, 2000; Wilson & Nutt, 2008; Morin, Culbert & Scharz, 1994). Neurofeedback is a form of biofeedback which brain activity via electroencephalography (EEG) is measured and fed back to a participant in real time as a visual or auditory representation in order to self-regulate brain activity (Sitaram et al., 2018). In a handful of pilot studies, the instrumental conditioning of the sensorimotor rhythm (SMR; 12-15 Hz) utilizing neurofeedback over central brain regions has been preliminarily shown to have positive behavioral impacts on sleep.

The aims of this study were to further investigate the therapeutic potential and feasibility of neurofeedback for the symptoms of insomnia. Participants (n=13) received 20 sessions of neurofeedback aimed to up-regulate SMR over the right central parietal region at scalp site C4 (10-20 international classification system). Analysis of variance was used to compare participants on self-report and sleep-tracking data on measures of sleep latency, overall sleep quality, fatigue, hyperarousal, and physiological measures of heart rate variability. Subjects were screened and selected based on strict inclusion and exclusion parameters, which included meeting criteria for an insomnia disorder. Participants were individuals suffering from severe insomnia that had made previous attempts at improving sleep with little success.

Results indicate significant improvements on some variables of sleep for both self-report questionnaires and sleep tracking data. On the PSQI, group means indicate overall statistically significant findings for global sleep score ($p < .001$). Sleep tracking data indicated statistically significant findings for total sleep time only ($p < .05$). Self report measures of fatigue and hyperarousal also demonstrated significant findings ($p < .001$). Heart rate variability data did not demonstrate significant findings.

The results of this study corroborate previous pilot studies indicating that SMR neurofeedback may help improve aspects of sleep quality. Research investigating insomnia is especially important since it has been categorized as an epidemic in the US (Hammer, Colbert, Brown, & Illo, 2011). Forthcoming research studies of SMR neurofeedback should incorporate follow-up measurements to track whether improvements sustain over time, include larger sample sizes, include a control group, and carefully measure increases in SMR over the course of training to ensure that the treatment is successful.

Keywords: Sensory Motor Rhythm (SMR), neurofeedback, insomnia, physiology, Heart-Rate Variability (HRV), sleep, electroencephalography (EEG), operant conditioning

TABLE OF CONTENTS

Abstract	iv
List of Tables	ix
List of Figures	x
Chapter 1: INTRODUCTION	1
Deficits in Insomnia	3
Neuropsychological Consequences	5
Effects on Working Memory	6
Effects on Executive Functioning and Processing Speed	7
Heart Rate Variability and Insomnia	8
Available Treatments for Insomnia	9
Operant Conditioning in Humans	10
The Process of Feedback	12
Sensorimotor Rhythm	12
Neurofeedback and The Brain	13
Brain Imaging	15
Neurofeedback Research for Sleep	17
Present Study	21
Hypotheses	22
Chapter II: METHODS	23
Participants	23
Measures	24
Pittsburgh Sleep Quality Index (PSQI)	25
The Sleep Condition Indicator (SCI)	27
The Hyperarousal Scale (HAS)	28
Multidimensional Fatigue Inventory (MFI-20)	28
S+ Sleep Monitoring System (S+)	29

Procedures	30
Recruitment	30
Inclusion Criteria	30
Exclusion Criteria	30
Phone Screening	31
Informed Consent	31
Pre-treatment Procedures	31
EEG Measurement	32
Treatment Procedures	32
Neurofeedback Training	32
Post-treatment Procedures	33
Heart Rate Variability Measurement	33
Threats to Internal and External Validity	34
Protection of Human Participants	35
Statistical Analysis	36
Chapter III: RESULTS	36
Distributional Analysis	36
Description of Baseline Sleep Characteristics	36
Comparison of Baseline Sleep to Normative Outcomes	37
Treatment Outcomes for Sleep Variables	37
Improvement Across Sleep Variables	38
Treatment Outcomes for Variables of Daytime Impairment	39
Predictors of Improvement	39
Bivariate Correlations	40
SMR Main Effect on HRV	40
Comparison of Baseline HRV to Normative Values	40
CHAPTER IV: DISCUSSION	42
Limitations	44
Conclusions	45

References	46
APPENDIX A. Hyperarousal Scale	61
APPENDIX B. Study Flyer	62
APPENDIX C. Demographic and Health Questionnaire	63
APPENDIX D. Debriefing Form	66
APPENDIX E. PSQI Results Comparison	67
APPENDIX F. Disruption Results Comparison	68
APPENDIX G. Sleep Onset Latency Results Comparison	69
APPENDIX H. Total Sleep Time Results Comparison	70

PREVIEW

List of Tables

Table 1. Sample Demographics	25
Table 2. Baseline Sleep Averages and Standard Deviations	36
Table 3. Normative Values Compared to Baseline Sleep Outcomes	37
Table 4. Pre to post Treatment Variables, Means, Standard Deviations, and Repeated Measures ANOVA Results	38
Table 5. Percent Change for All Sleep Variables	38
Table 6. Pre to post Daytime Impairment Variables, Means, Standard Deviations, and Repeated Measures ANOVA results	39
Table 7. Frequencies of SMR Score	39
Table 8. Bivariate Correlations for SMR Scores Across All Variables	40
Table 9. Pre to post HRV Variables, Means, Standard Deviations, and Repeated Measures ANOVA Results	40
Table 10. One Sample T-test Comparing Baseline HRV Outcomes to Normative Values	41

List of Figures

Figure 1. Participant flow chart following Consolidated Standards of Reporting	24
Figure 2. Study Protocol	34

PREVIEW

Chapter 1:

Introduction

Insomnia is characterized by a range of symptoms that include a disruption in the duration, quality, and/or maintenance of sleep. According to the Diagnostic Manual for Mental Disorders Fifth Edition (DSM-5), the minimum criteria required for an insomnia disorder requires sleep disruption to occur for at least three nights a week and present for a minimum of three months (American Psychiatric Association, 2013). It also includes daytime symptoms such as fatigue or sleepiness, cognitive difficulties, and mood disturbances (Edinger et al., 2004). It is the most prevalent among the sleep disorders within the general population (Schutte-Rodin, Broch, Buysse, Dorsey & Sateia, 2008), reaching the status of an epidemic in the US (Hammer, Colbert, Brown & Illoi, 2011). In fact, approximately 33% to 50% of the adult US population has suffered from symptoms of insomnia, with 5% to 10% meeting criteria for a specific insomnia disorder (Ancoli-Israel, 1999). Within individuals who have symptoms of insomnia, between 10 and 18 percent of these individuals rate their difficulties to be chronic and severe (Ohayon, 2002).

The symptoms of insomnia have the potential to dramatically affect a person's wellbeing, health, and social and occupational functioning (Ishak et al., 2012). Left untreated, insomnia may lead to the development of psychological disorders such as major depression (Ford & Kamerow, 1989). In fact, longitudinal studies have demonstrated that the longer insomnia symptoms are left untreated, the greater the likelihood of developing a comorbid mental disorder (Breslau et al., 1996). In addition, individuals with chronic insomnia have been shown to abuse alcohol and drugs at higher rates and are more likely to commit suicide (Weissman, Greenwald, Nino-Murcia & Dement, 1997; Breslau, Roth, Rosenthal & Andreski, 1996). Other common co-morbid disorders that have been found are generalized anxiety, attention deficit/hyperactivity disorder in

children, and dementia (Belanger, Morin, Langlois, & Ladouceur, 2004; Breslau, Roth, Rosenthal & Andreski, 1996). Lastly, insomnia has also been shown to negatively impact a person's overall quality of life (Leger, Scheuermaier, Philip, Paillard & Guilleminault, 1998), even closely resembling that of those with congestive heart failure and depression (Katz & Mchorney, 2002).

It has been hypothesized that because insomnia leads to fatigue, it could directly participate in the development of some diseases or it may be partly responsible for decreasing the threshold of other diseases, having an additive exacerbating effect (Metlaine, Leger & Choudat, 2004). In fact, Correlational studies with large representative samples have found correlations between insomnia and a variety of medical disorders such as cancer, heart disease, diabetes, respiratory symptoms, and stroke (Foley, Monjan, Simonsick, Wallace & Blazer, 1999). Although insomnia may not directly cause these medical conditions, it may worsen them and act as a catalyst for their development.

Aside from insomnia's impact on the individual level, it has also been found to impact society on wider scale. For example, insomnia creates a major negative socioeconomic impact to the US economy (Chevalier et al., 1999). In addition, it has been found to impact society in terms of disability, increased health care costs, work absenteeism and poorer job performance (Morin & Jarrin, 2013; Chilcott & Shapiro, 1996). In the case of work productivity, insomniacs have been found to have up to 200 hours of less productivity per year compared to workers without the disorder (Bolge, Doan, Kannan & Baran, 2009). One of the biggest risks of insomnia is the increased occurrence of industrial and motor vehicle accidents (Roth, 2005). In a survey done by the Gallup Organization, it was found that insomniacs had nearly triple the risk when compared

to controls of having a driving accident (Gallup Organization, 1991). Estimates have found this disorder to cost the US between \$92 and 107 billion (Stoller, 1994; Rosekind & Gregory, 2010).

Deficits in Insomnia

Insomnia is best explained from a biopsychosocial framework because a holistic picture of the disorder best encapsulates it. Biological, psychological and societal factors have each been found to contribute to the disorder significantly (Lundh, Broman, & Hetta, 1995). For example, excessive worrying and proneness to introspection has been found to be a predisposing factor (Morin, Rodrigue, & Ivers, 2003). A genetic link has also been found in those who have a family history of the disorder (Beaulieu-Bonneau, Leblanc, Merette, Dauvilliers, & Morin, 2007). Other predispositions include being divorced, separated or widowed, and having a lower income and education (Gellis et al., 2005; Bastien, Vallieres, & Morin, 2004).

An emerging model of insomnia hypothesizes that individuals suffering from this disorder have an overall physiological profile of hyperarousal (Bonnet & Arand, 2010). Although insomniacs suffer from symptoms of fatigue and tiredness, they do not have the ability to be calm. This overall hyperarousal impairs their ability to fall asleep easily at night, contributing to their symptoms of insomnia. Further corroborating this hypothesis, patients with insomnia have been found to have an increase in indicators that point to sympathetic arousal, such as an increase in corticosteroids and adrenaline, increased EMG in the frontalis and mentalis regions, an increase in body temperature, and an increase in beta EEG activity (Johns, Gay, Mesterton & Bruce, 1971; Frankel, Buchbinder, Coursey, & Synder, 1973; Freedman & Sattler, 1982; Freedman, 1986). Cardiac measures such as increased heart rate during wake-time and sleeping, reduced heart rate variability, and an increased risk for hypertension have also been implicated in those suffering from insomnia (Bonnet & Arand, 1998; Vgontzas, Liao, Bixler,

Chrousos & Vela-Bueno, 2009). The overall presence of physiological hyperarousal is said to interfere with the overall quality of sleep, delaying sleep-onset and disrupting the maintenance of sleep (Perlis et al., 1997). In fact, insomniac's baseline level of central nervous system arousal as measured by cardiovascular activation has been found to be among the most meaningful measures in predicting the occurrence of the disorder in younger adults (Bonnet & Arand, 2010).

Individuals suffering from insomnia have also been found to have cognitive hyperarousal, which is defined by having aversive and unwanted thoughts during the process of initiating sleeping. Patients call this ruminative pre-sleep activity as a 'racing mind' (Robertson et al., 2007). These negative cognitions can even persist during periods of wakefulness throughout the night (Harvey & Tang, 2003). In fact, an inverse relationship has been found between the amount and severity of negative cognitions and poor sleep (Fichten et al., 1998). An interesting paradigm that lends support for the hypothesis that cognitions impact sleep is based on the "internalization of conflicts model" by Kales et al. (1976), which states that cognitive patterns such as those that lead a person to internalize psychological conflict increase emotional arousal. This in turn impacts physiological systems and promotes hyperarousal, making sleep increasingly difficult. The researchers lend support for this model based on their findings that the highest scores on the Minnesota Multiphasic Personality Inventory (MMPI) in a sample of insomniacs compared to controls were those that indicated more internalizing problems, such as the depression and psychasthenia scale.

A third finding that has been proposed which is of particular interest to this study is that insomnia is accompanied by an increase in overall cortical arousal (Perlis et al., 1997). Specifically, insomniacs have been shown to have an excess of high frequency activity during the entire process of sleep, including the pre-sleep awake stage (Merica & Gaillard, 1992;

Marzano, Ferrara, Sforza & De Gennaro, 2008). This high frequency activity has been found to be specifically in beta EEG, often defined in the 14-32 Hz range (Perlis, Merica, Smith & Giles, 2001). Focusing on cognitive arousal, Perlis et al. (1997) described a model known as the neurocognitive model that explains how insomnia is impacted by cortical arousal. His model is based on the findings that high frequency activity, namely beta and gamma EEG activity, are present during information processing. He also noticed that these same EEG patterns are elevated in insomniacs close to bedtime. Frequencies in this range have also been associated with attention, perception, and cognitive functioning in humans (Pantev 1995; Lutzenberger, Pulvermuller & Birbaumer, 1994). Overall, these findings have been found to directly relate to primary, and not secondary, insomnia (Lamarche & Ogilvie, 1997; Perlis, Smith, Orff, Andrews & Giles, 2001).

Aside from elevated beta EEG, sleep issues and fatigue due to lack of sleep have been found to manifest as excess frontal alpha and theta (Arns & Kenemans, 2014). Spindling excessive beta (SEB), which is considered to be excessive beta activity that is rhythmic in morphology (Johnstone, Gunkelman, & Lunt, 2005), has been found to be correlated with sleep maintenance problems and impulsivity (Arns, Swatzyna, Gunkelman & Olbrich, 2015). This lends evidence to the possibility that SEB may possibly be an EEG pattern of hypoarousal that indicates behavioral psychopathology (Arns, Swatzyna, Gunkelman & Olbrich, 2015). SEB has been found in patients with severe insomnia (Van Sweden et al., 1986).

Neuropsychological Consequences

Insomnia has been found to impact neuropsychological functioning (Fulda & Schulz, 2001). For example, attention span has been found to be reduced in insomniacs when compared to controls (Hauri, 1997). Vigilance, or the ability to maintain attention and/or alertness over

time, has also been found to be reduced in insomniacs when compared to controls (Schneider-Helmert, 1987; Sugerman, Stem & Walsh, 1985; Altema, Van Der Werf, Strijers & Van Someren, 1998). There is also partial evidence for executive functioning, memory and simple motor speed deficits among insomniacs, although further research is needed to validate these claims (Randazzo, Schweitzer, Stone, Compton & Walsh, 2000; Bonnet & Arand, 1995; Szelenberger & Niemcewicz, 2000; Mendelson, Garnett, Gillin, & Weingartner, 1984). A meta analysis conducted by Fortier-Brochu et al. (2011) analyzed studies of neuropsychological functioning in participants with insomnia compared to controls. They found medium effect sizes to support the conclusion that insomnia is associated with moderate deficits on specific cognitive domains, namely working memory, episodic memory, problem solving, reaction time, selective attention and information processing. These neuropsychological impairments possibly reflect some of the negative daytime impairments that are associated with the disorder.

Effects on Working Memory. The effects of insomnia on working memory have been found in a handful of studies. For example, Haimov, Hanuka and Horowitz (2008) recruited 35 adults with chronic insomnia and compared them to 64 adult controls without insomnia on a variety of cognitive tasks that included working memory. Exclusion criteria included any history of psychiatric or neurological disorder, current consumption of any medication that affects the central nervous system, and any major medical disease. The results of this study found that chronic insomnia in adults was associated with impairments in cognitive performance, specifically working memory. Corroborating these findings, Hauri (1997) also found similar results in a sample of 26 paired insomniacs and controls that were matched on age, sex, education and occupation. Similarly, insomniacs have been found to perform poorer on the Digit Span Test, a commonly used neuropsychological test of working memory (Vignola, Lamoureux,

Bastien & Morin, 2000). Despite these findings, other studies have found mixed results, arguing that working memory remains relatively intact in insomniacs (Bonnet, 1985).

Effects on Executive Functioning and Processing Speed. In a large study recruiting the biggest sample to date, Fernandez-Mendoza et al. (2010) utilized a cross-sectional population-based sample of 1,741 men and women in order to investigate the effects of chronic insomnia on neuropsychological performance. The participants were given a neuropsychological battery that included The Symbol Modalities Test (SDMT), The Trail Making Test (TMT), The Benton Visual Retention Test (BVRT), and The Thurston Work Fluency Test (TWFT) in hopes of measuring psychomotor processing speed and visual scanning, set-switching, short-term visual memory and visual perception/visual-constructive abilities, and word fluency. They found no significant difference between the insomniac group and the controls on basic demographic variables such as a race, gender, age, education, physical and mental health, and body mass index. The researchers found impairments in set-switching attention, short-term visual memory, and processing speed in insomniacs. This evidence has lead the researchers to postulate that these findings demonstrate deficits in the “executive control of attention,” implicating areas of the brain such as the prefrontal cortex which is intimately involved in executive processing. Lending further evidence to this hypothesis is the finding of a functioning imaging study done by Altena et al. (2008) that found hypoactivation of the prefrontal lobe in insomniacs when asked to generate as many words as possible from a category within a given time. This task, which is referred to as verbal fluency, is said to be heavily dependent on the prefrontal executive system (Lezak et al., 2004).

Edginger, Means, Carney, and Krystal (2008) examined psychomotor processing speed in 79 participants with insomnia as compared to 84 controls with no sleep difficulties. Participants