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Neuroflexibility and Sleep Onset Insomnia Among College Students: Implications for Neurotherapy

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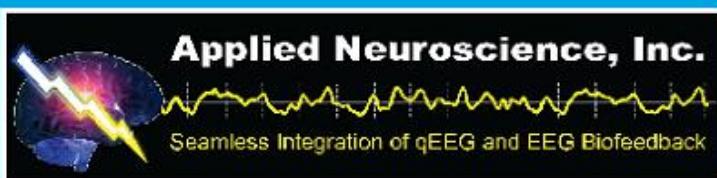
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SCIENTIFIC FEATURES

NEUROFLEXIBILITY AND SLEEP ONSET INSOMNIA AMONG COLLEGE STUDENTS: IMPLICATIONS FOR NEUROTHERAPY

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This study was designed to assess a neuroflexibility model of sleep onset insomnia among college students. Neuroflexibility refers to the ability to adjust cortical activation consistent with environmental demands. It was anticipated that good sleepers would demonstrate better feedback contingent alpha control, defined as the ability to both enhance alpha and suppress alpha, than poor sleepers. Ten good and 10 poor sleepers participated in two sessions of bidirectional alpha feedback. As predicted, good sleepers demonstrated better alpha control compared to poor sleepers, although this pattern was only partially replicated in a second session. This study provides a degree of empirical support for interventions designed to enhance neuroflexibility in the treatment of some people with sleep onset insomnia.

Poor sleep is a common complaint among young people in the United States, with as many as 86% of Generation Y (ages 19–29) participants reporting sleep problems at least a few nights a week. Relative to other age groups, Generation Y participants reported the most difficulties falling asleep (45%) and the latest bedtimes, and they were most likely to have driven while drowsy (67%; National Sleep Foundation, 2011). Utilizing the Pittsburgh Sleep Quality Index measure, Lund, Reider, Whiting, and Prichard (2010) found that 60% of college students were categorized as poor-quality sleepers. Poor sleep was characterized by restricted total sleep time and long sleep latencies, with 32% reporting an inability to fall asleep within 30 min at least once a week. Utilizing multiple regression analyses, these same investigators found that tension and stress were significant predictors of poor sleep quality among a college student

population, accounting for 24% of the variance. Poor sleep is associated with increased irritability, impaired cognitive performance, an increased number of accidents, and impaired immune system functioning.

The typical daytime, awake EEG pattern characterizing those reporting poor nighttime sleep is not entirely clear. One study, examining awake EEG's, found higher values of beta power, suggesting excessive arousal, and lower values of Theta power during daytime assessments among people with primary insomnia compared to healthy controls (Wolynczyk-Gmaj & Szelenberger, 2011). The beta wave activity was also positively correlated with indications of hyperarousal from a paper-and-pencil measure, The Hyperarousal Scale, whereas the inverse was true for theta wave activity, suggesting that 24-hr hyperarousal may occur in those with primary insomnia (Regestein, Dambrosia, Hallett,

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Murawski, & Paine, 1993). However, such daytime central nervous system hyperarousal has not been consistently found in other studies (Buckelew, DeGood, Roberts, Butkovic, & MacKewn, 2009; Hauri, Percy, Hellekson, Hartmann, & Russ, 1982). In fact, excessive theta wave EEG 4–8 Hz, suggestive of fatigue, is commonplace among poor quality sleepers (Thompson & Thompson, 2003).

Heightened responses to environmental stressors and increased tension accompanied by decreased slow EEG wave activity, although undoubtedly true for some, is not a sufficient explanation of all poor sleep quality. The International Classification of Sleep Disorders-Revised (American Academy of Sleep Medicine, 2005) distinguishes between psychophysiological insomnia and idiopathic insomnia. Psychophysiological insomnia, essentially a stress and tension model, is associated with increased muscle tension, anxiety, and conditioned or learned sleep disruptive cognitive associations. The etiology of idiopathic insomnia is more speculative but may be due to long-standing neurologic factors persisting across the lifespan and generally characterized by long sleep latencies. Variability in study results may occur as a function of a heterogeneous sample of people with insomnia, some with psychophysiological insomnia and some with idiopathic insomnia.

In individual cases of poor sleepers, the psychophysiological versus idiopathic causes of poor sleep may not be mutually exclusive. We believe that another model that might better encompass both the psychophysiological stress and idiopathic models is a variation of a cortical dysregulation model (Othmer, Othmer, & Kaiser, 1999). We have previously proposed a brain dysregulation, or what we have called impaired neuroflexibility model (Buckelew et al., 2009). Impaired neuroflexibility refers to a lack of cortical flexibility such that, at any given moment, there may occur a mismatch between cortical activation and the constantly changing situational demands imposed on the central nervous system by the immediate environment. For example, people with impaired neuroflexibility may

demonstrate difficulty staying alert while reading, a task requiring focused attention and typically associated with increased beta activity. Conversely, people with impaired neuroflexibility may demonstrate delayed sleep onset because of difficulty shifting to a slow EEG wave and drowsy state to prepare for sleep. Furthermore, this model suggests that poor sleepers may also have difficulty adjusting brain activation to differing daytime situational demands. We have found some preliminary support for this model (Buckelew et al., 2009). When students were challenged with a “sensory attentiveness” task in which they listened to an orally presented story, good sleepers showed a theta suppression pattern more consistent with focused attention, whereas poor sleepers showed the opposite, a pattern of theta enhancement.

Evidence for an EEG dysregulation model for insomnia comes from other sources as well. Serman and colleagues noted improved sleep in cats (Serman, Howe, & Macdonald, 1970) following sensorimotor rhythm (SMR) EEG (e.g., 12–15 Hz) feedback. Similar sleep improvement was observed in humans who were trained to enhance low beta (12–15 Hz, SMR) for control of epilepsy (Serman & House, 1980). More recently, others have replicated and expanded this work, demonstrating that SMR training can impact EEG sleep architecture in cats and humans (Amzica, Neckelmann, & Steriade, 1997; Hoedlmoser et al., 2008). In a well-controlled study, Hoedlmoser and colleagues found that SMR training resulted in reduced sleep latency (and enhanced declarative learning) among healthy individuals who participated in SMR training compared to a control group.

More than three decades ago, recognizing that the EEG variability among poor sleepers has important treatment implications, Hauri (Hauri, 1981; Hauri et al., 1982) reported that people with psychophysiological insomnia responded positively to theta training, whereas people with idiopathic insomnia appeared to benefit from SMR training. Increasingly, SMR training has been found to be useful for both the treatment of epilepsy, ADHD,

and insomnia (Monastra, 2003; Schwartz & Andrasik, 2003). Effectiveness of this training may be EEG target specific, such that increased SMR amplitudes may directly result in improved sleep. Alternatively, SMR training may indirectly provide participants with an increased generalized ability to self-regulate EEG states, resulting in improvements in the ability to adapt to the sleep environment, thus enhancing neuroflexibility, consistent with the dysregulation model. However, a question remains as to whether SMR training is the only, or most efficient, form of EEG biofeedback training for helping individuals with sleep problems.

RESEARCH QUESTIONS

Our neuroflexibility model suggests that adjustment of brain activation to meet the demands of the current environment is essential to both good sleep and effective cognitive functioning while awake. The current study was designed, using a simple alpha feedback paradigm, to further assess a neuroflexibility model of sleep onset insomnia among college students. More specifically, it was proposed that good sleepers, as a test of neuroflexibility, would demonstrate better ability to self-regulate awake-alpha compared to poor sleepers. Alpha control here is defined as the ability to both enhance alpha (deactivate cortical arousal) and suppress alpha (activate cortical arousal) with contingent feedback. Furthermore, we were interested in exploring if alpha control would be stable across eyes-open and eyes-closed conditions and across two testing sessions. In other words, is alpha control, as defined here, a reliable diagnostic correlate of good versus poor sleepers in this population? Such a diagnostic demonstration is a prerequisite for suggesting that training in alpha regulation, or some other frequency, might produce improved sleep. Correlated theta activity was also monitored to further assess neuroflexibility. It was hypothesized that good sleepers would demonstrate greater variability of theta activity consistent with the demands of the situation.

METHODS

Participants

Twenty students (10 good and 10 poor sleepers) participated in a psychophysiological assessment protocol. This sample was identified from a larger pool of 223 introductory psychology students at a 4-year state university who completed an informed consent, demographic measure, the Pittsburgh Sleep Quality Index-Adjusted (PSQI-A), and the Spielberger Trait Anxiety Inventory. The poor sleep group included students with no health problems who reported chronic trouble falling asleep, defined as "taking 30 minutes or longer to fall asleep for at least 6 months" and obtained a score of 6 or greater on the PSQI-A. The good sleep group included students who reported no sleep or health problems and obtained a score of 5 or less on the PSQI-A. There were two male and eight female participants in the good sleep group and 10 female participants in the poor sleep group. There was a significant difference in the mean age of the two groups, $t(9.80) = 2.48$, $p = .033$. The mean age of those in the good sleep group was 19.00 years ($SD = 1.49$), and the mean age of those in the poor sleep group was 24.70 years ($SD = 7.10$).

Apparatus and Measures

EEG. Psychophysiological data were measured using a customized script for the Biograph Infiniti (EEG Suite) data acquisition system for the Procomp Infiniti (Thought Technology LTD). This eight-channel research grade device acquires 256 samples/second. EEG was recorded using the EEG-Z sensor, a preamplified electroencephalograph sensor with a built in impedance-checking device. Alpha band pass frequencies were 8–12 Hz.

Sleep quality. The PSQI-A is a 19-item self-report instrument that measures sleep quality over the preceding month (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Global sleep quality scores range from 0 to 21, with higher scores reflecting poorer sleep quality. Utilizing a cutoff score of 5 or below for good sleepers, diagnostic sensitivity was 89.6% and specificity was 86.5%. The PSQI

has been adjusted for use with college students (F.C. Brown, personal communication, September 7, 2006). The adjusted score reflecting sleep quality during the week was used in this study because of the well-documented discrepant patterns of college student sleep behavior between weekdays and weekends.

State-Trait Anxiety Inventory. The state and trait portions of the State-Trait Anxiety Inventory (Form Y) require that participants rate 20 items on a 1-to-4 scale, based on “how you feel now” (state anxiety) or “how you generally feel” (trait version; Spielberger, 1983). This measure, developed primarily for use with college students, has been used successfully in previous research with college students with sleep onset insomnia (Buckelew et al., 2009). Test-retest reliability correlations range from .73 to .86, concurrent validity correlations range from .73 to .85, and interrater reliability coefficients range from .83 to .94 for college students (Spielberger, 1983).

Procedure

Each participant was seated in a cushioned recliner in a sound attenuated room, facing a computer monitor. A standard gold-plated EEG electrode was secured using conductive paste (Ten20) on the vertex (Cz), with a right earlobe reference and left earlobe ground. Three electromyogram (EMG) electrodes were placed on the participant’s forehead to measure frontalis EMG. Skin conductance response (SCR) electrodes were secured on volar surface of the distal phalanxes on the second and fifth fingers of the right hand (EMG and SCR measures not reported here). Each participant signed an informed consent form and completed the state portion of the Spielberger State-Trait Anxiety Inventory. Sessions began after verifying the EEG impedance level was below 5 k Ω and the EMG level was below 5 microvolts. Both visual and auditory feedback was provided with continuous feedback proportional to amplitude and frequency once individually determined auditory thresholds were achieved. Thresholds were set at 80% of the baseline alpha mean for the eyes-open segments and increased by 2 microvolts for

the eyes-closed segments. In addition to the neurofeedback, participants were provided general instructions on self-regulation strategies, including to maintain calm and relaxed thoughts during enhance alpha segments and to have focused thoughts like mental arithmetic during the suppress alpha segments. The script for the psychophysiological assessment protocol consisted of six segments: (a) Eyes-Open Pre-baseline, (b) Eyes-Open Alpha Enhancement, (c) Eyes-Open Alpha Suppression, (d) Eyes-Closed Alpha Enhancement, (e) Eyes-Closed Alpha Suppression, and (f) Eyes-Open Post-Baseline. The baseline segments were 4 min each, and the feedback segments were 5 min. At the completion of data collection, participants received a \$20 gift card to the campus bookstore.

EEG recordings were reviewed for movement and other sources of artifact. High amplitude artifact was rejected. A minimum of 25% of the low amplitude data from each task was required to include this data in data analysis. To increase power and simplify data analyses, pre- and postbaseline data were omitted from primary data analysis.

Statistical analyses. Statistical analyses were conducted for raw alpha values, alpha control scores, cutoff alpha scores, raw theta values, and anxiety scores. For raw alpha values and raw theta values, two mixed analyses of variance (ANOVAs) were conducted for two levels of group (good vs. poor sleepers) and four levels of task (eyes-open alpha enhance, eyes-open alpha suppress, eyes-closed alpha enhance, eyes-closed alpha suppress) for each of the two sessions. Simple effects (independent *t* test for between-group differences) were conducted to further examine significant interactions.

Alpha control scores were derived by subtracting raw alpha mean scores for the suppress condition from the raw alpha mean score for the enhance condition. The task effects included two levels of alpha control for the eyes-open and the eyes-closed conditions.

To assess the clinical application of the neuroflexibility model to specific individuals

rather than group data only, an alpha control score of $.33 \mu\text{V}$ was used to define good or poor alpha control. (This score reflects the highest median alpha control value across the sessions for all participants.) The proportion of good sleepers and poor sleepers who met the alpha control cut-off scores are reported.

Last, independent t tests were conducted comparing good versus poor sleepers on trait anxiety and state anxiety (reported during Session 1 and Session 2).

RESULTS

Raw Alpha Values

Data comparing the two sleep groups across the four experimental conditions were analyzed with separate ANOVAs for Session 1 and Session 2 data. Figures 1 and 2 reveal the mean alpha scores for Sessions 1 and 2. In both cases, the ANOVAs for the task effect were significant, $F(3, 57) = 31.25, p < .001$, and $F(3, 57) = 13.29, p < .001$. Consistent with the literature (Thompson & Thompson, 2003, p. 36), alpha mean scores were higher in the eyes-closed compared to the eyes-open condition for both groups. Also, the Task \times Group interactions were significant for both Session 1 and 2, $F(3, 54) = 3.14, p = .03$, and $F(3, 54) = 5.91, p = .001$. Post hoc t tests indicated significant differences between the poor and good sleepers during the alpha enhancement eyes-open task for Session 1 and 2, $t(18) = 3.15, p = .006$;

$t(18) = 2.7, p = .015$, respectively, and for alpha suppression eyes-open for Session 1 and 2, $t(18) = 1.82, p = .04$ (one-tailed) and $t(18) = 2.42, p = .027$, respectively.

Alpha Control Scores

Alpha control is defined as the ability to both enhance (deactivate) and suppress (activate) alpha and is derived by subtracting the mean alpha suppress score from the mean alpha enhance score for each of the eyes-open and eyes-closed conditions. For alpha control data, ANOVAs revealed no significant task or Task \times Group interaction effects for Session 1, $F(1, 18) = .59, p = .45$; $F(1, 18) = .03, p = .87$, or Session 2, $F(1, 18) = 1.12, p = .30$; $F(1, 54) = .66, p = .43$, respectively. However, in Session 1 there was a significant sleep group effect, $F(1, 18) = 6.4, p = .02$, with good sleepers demonstrating better alpha control across the session than poor sleepers (see Figure 3). Although the group effect was not significant in Session 2, $F(1, 18) = .19, p = .67$, the pattern of higher alpha control for good versus poor sleepers was observed in the eyes-open condition in Session 2 (see Figure 4).

Cutoff Alpha Scores

A $.33 \mu\text{V}$ cutoff score was used to define good or poor alpha control. (This score reflects the highest median value across the sessions for all participants.) In Session 1, eyes-open condition, nine of the 20

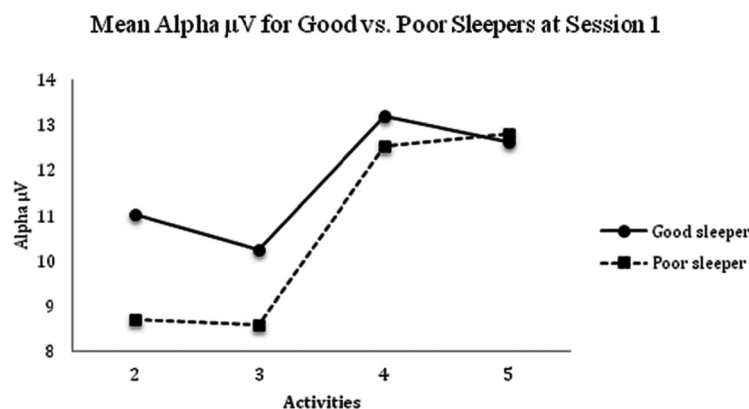


FIGURE 1. Session 1. Note. Mean alpha μV values for good and poor sleepers across activities 2 (eyes-open enhance alpha), 3 (eyes-open suppress alpha), 4 (eyes-closed enhance alpha), and 5 (eyes-closed suppress alpha) conditions.

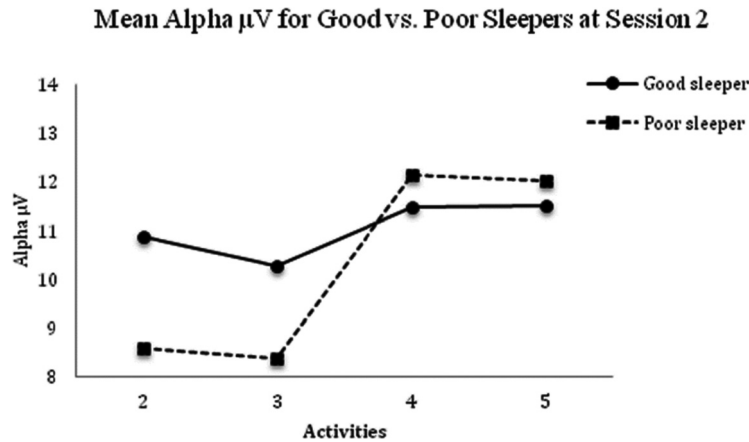


FIGURE 2. Session 2. Note. Mean alpha μV values for good and poor sleepers across activities 2 (eyes-open enhance alpha), 3 (eyes-open suppress alpha), 4 (eyes-closed enhance alpha), and 5 (eyes-closed suppress alpha) conditions.

participants demonstrated good alpha control. Of these, 67% were good sleepers. In Session 1, eyes-closed condition, 10 participants demonstrated good alpha control. Of these, 80% were good sleepers.

Using the $.33\ \mu\text{V}$ cutoff score to define good alpha control, across both eyes-open and eyes-closed conditions of Session 1, five people showed good alpha control consistently; all were good sleepers. Using below the $.33\ \mu\text{V}$ cutoff to define poor alpha control across the first session, five people showed consistently poor alpha control; four of the five were poor sleepers.

Last, using the $.33$ cutoff score to define good or poor alpha control, across the eyes-open condition in Sessions 1 and 2, there were five participants who showed good alpha control across both sessions. Three (60%) of these were good sleepers. Across the eyes-open condition in Sessions 1 and 2, there were eight participants who consistently demonstrated poor alpha control; five (75%) of these were poor sleepers.

Theta Wave Values

Mean microvolt theta wave activity was analyzed across the four conditions for both

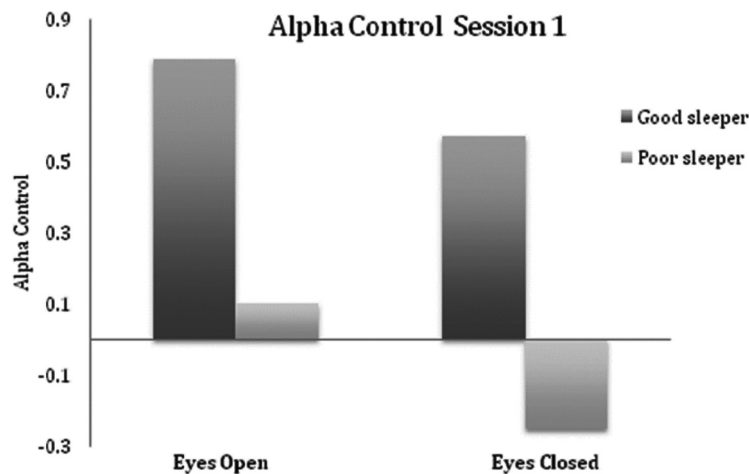


FIGURE 3. Session 1. Note. Mean alpha control scores, defined as the ability to both enhance and suppress alpha and derived by subtracting the mean alpha suppress score from the mean alpha enhance score, for good and poor sleepers across activities 2 (eyes-open enhance alpha), 3 (eyes-open suppress alpha), 4 (eyes-closed enhance alpha), and 5 (eyes-closed suppress alpha) conditions.

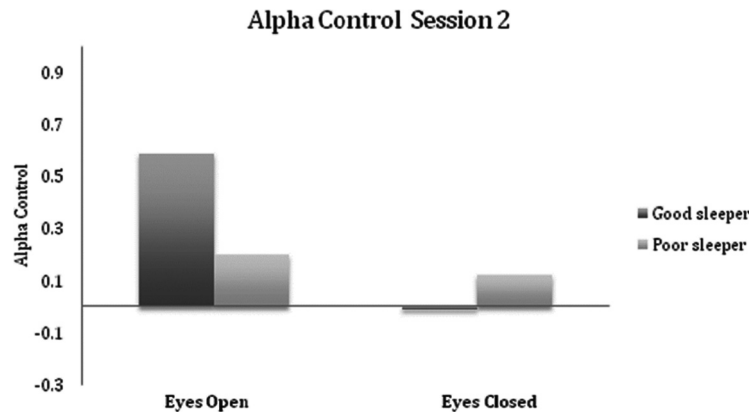


FIGURE 4. Session 2. Note. Mean alpha control scores, defined as the ability to both enhance and suppress alpha and derived by subtracting the mean alpha suppress score from the mean alpha enhance score, for good and poor sleepers across activities 2 (eyes-open enhance alpha), 3 (eyes-open suppress alpha), 4 (eyes-closed enhance alpha), and 5 (eyes-closed suppress alpha) conditions.

Sessions 1 and 2. Across the two sessions, the ANOVAs for the task effect were significant, $F(3, 54) = 12.35$, $p < .001$, and $F(3, 54) = 15.59$, $p < .001$, and Task \times Group interactions were significant for both Sessions 1 and 2, $F(3, 54) = 6.8$, $p = .001$, and $F(3, 54) = 12.03$, $p < .001$, respectively, with good sleepers showing significantly more theta with eyes closed. Follow-up post hoc t tests indicated consistent trends between the poor and good sleepers with good sleepers showing more theta than poor sleepers during the alpha enhancement eyes-closed task, $t(18) = 1.77$, $p = .09$, $t(18) = 1.54$, $p = .14$, and alpha suppression eyes-closed task, $t(18) = 1.52$, $p = .15$, $t(18) = 2.18$, $p = .04$, for Sessions 1 and 2, respectively. Because the mean theta

scores for Session 1 and 2 were essentially identical, only Session 1 data are reported in Figure 5.

Anxiety Values

There was also a significant difference in the Trait Anxiety measure among the good and poor sleepers, $t(17) = 2.98$, $p = .008$, with mean scores of good and poor sleepers being $M = 34.90$, $SD = 7.82$ and $M = 47.67$, $SD = 10.75$, respectively. Although no significant differences were found between the two groups on the State Anxiety measure for the first session, a trend was identified, $t(18) = 1.86$, $p = .079$. Good sleepers scored a mean of 31.00 ($SD = 8.60$), and poor sleepers scored a mean of 39.10 ($SD = 10.75$).

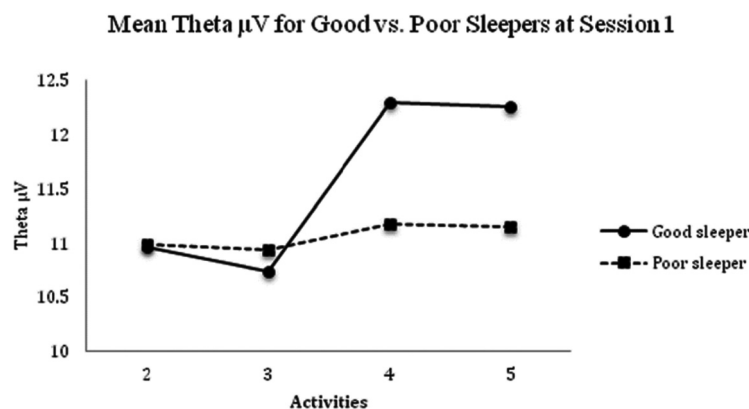


FIGURE 5. Session 1. Note. Mean theta μ V values for good and poor sleepers across activities 2 (eyes-open enhance alpha), 3 (eyes-open suppress alpha), 4 (eyes-closed enhance alpha), and 5 (eyes-closed suppress alpha) conditions.

Significant between group differences were found during the second session, $t(17) = 2.51$, $p = .022$. Good sleepers scored a mean of 29.80 ($SD = 6.56$), and poor sleepers scored a mean of 40.67 ($SD = 11.83$) in Session 2.

DISCUSSION

The present results appear to support a neuroflexibility deficit model as an alternative to both the psychophysiological stress and idiopathic models of insomnia. In general, the poor sleepers in this sample demonstrated greater difficulty in self-regulation of alpha, relative to good sleepers. In fact, the alpha control score was negative for poor sleepers in the first session eyes-closed condition, reflecting the fact that poor sleepers, as group, increased alpha activity rather than decreased alpha activity in the suppress alpha feedback condition. Second, there appears to be some modest support for the reliability of alpha control across an initial session condition (eyes-open and eyes-closed feedback conditions) and across sessions (in the eyes-open only condition).

Additional data consistent with the neuroflexibility model was found in the concomitant theta wave data. Mean theta wave activity was essentially the same for good versus poor sleepers in the eyes-open feedback condition across both sessions. However, the good sleepers demonstrated a marked increase in theta wave activity in the eyes-closed conditions, whereas no change was observed for the poor sleepers. Normally, one would expect such a slow wave increase, as demonstrated by the good sleepers, with the removal of visual stimulation. Poor sleepers, on the other hand, failed to demonstrate this theta wave visual condition effect and thus appeared to be lacking in what might be considered healthy and resilient neuroflexibility. This same group pattern also appeared reliably across the two sessions. This heightened theta wave activity demonstrated by good sleepers during the eyes-closed conditions may also be consistent with other reports of higher theta activity among good sleepers compared to poor sleepers (Wolynczyk-Gmaj & Szelenberger, 2011).

The failure to replicate the alpha control group differences in the second session eyes-closed condition raises some question about the reliability of the sleep group findings, especially as a differential diagnostic tool for good versus poor sleepers. Neither good nor poor sleepers were able to effectively self-regulate alpha wave activity during the eyes-closed condition of the second session. It is our speculation that as the task difficulty increased, the between sleep group differences may “wash out.” It is commonly found that alpha regulation is more difficult with eyes closed, especially alpha suppression, because normal alpha levels from a vertex location tend to be quite elevated with eyes closed. In this case, the difficulty of the task demands may have reduced our ability to consistently demonstrate alpha control.

Yet another consideration in the relative ease of eyes-open versus eyes-closed alpha control is the role of visual focusing with eyes open. One might argue that the good sleepers are simply better at recognizing that focusing their vision tends to reduce alpha production, whereas employing a blank stare tends to enhance alpha. However, our view is that no matter how they do it, the better sleepers are demonstrating greater ability to figure out how to adjust their brain activation to the immediate demands of the environment; in this case, adjusting to the contingent EEG feedback demands.

These findings are particularly noteworthy in light of some sampling limitations in this current study (higher trait anxiety among poor sleepers and higher age among the poor sleepers, not present in our earlier study (Buckelew et al., 2009)). This group difference in trait anxiety would tend to argue for a psychophysiological model of poor sleep. Certainly, the role of anxiety in accounting for these group differences cannot be completely ruled out. But it is our position that the neuroflexibility model is a more broadly encompassing model that still remains compatible with both psychophysiological and idiopathic models. Although the greater age among poor sleepers might contribute to a tendency toward less slow wave (alpha and theta) activity relative to the good sleepers, to

our knowledge, it seems unlikely that the age difference would account for the pattern of observed group differences in EEG. In addition, utilizing difference scores for the alpha control score helps to control for individual baseline differences.

This study is limited by the use of a mostly female college student population rather than a clinical sample. A group with more severe clinical sleep disorders and with more male participants might produce different results. These students were volunteers for a study, not patients seeking treatment for their sleep concerns. Many college students develop a habit of going to bed very late and then sleeping late into the morning resulting in a forward shift of their circadian rhythm. They may self-identify a sleep problem only when circumstances require them to go to bed earlier because of an early class or a summer internship. If such students with altered circadian rhythms were mixed into the poor sleep group, there is no particular reason to believe that, for them, a lack of neuroflexibility is a key issue.

One of the authors (DDG) has been attempting to treat individuals with clinical insomnia utilizing this EEG control based neuroflexibility model. Patients who have not responded to traditional cognitive-behavioral sleep counseling, sleep hygiene education, and general relaxation training have participated in EEG biofeedback based on the goal of enhancing neuroflexibility via bidirectional alpha feedback. In the course of a 30-min feedback session, divided into six 5-min feedback segments, the individual patient's feedback contingency is switched back and forth between 5-min segments of cortical alpha activation and deactivation in an attempt to improve the ability to adjust situational brain activation to the demands of the immediate environment. Several individuals, highly resistant to past sleep treatment efforts, have reported improvements in their sleep, sometimes after only three or four such sessions. Most critically, they are encouraged to simulate this same cognitive deactivation-activation practice on their own at least twice a day, but without the feedback. Of course, nonspecific

treatment factors including motivation and placebo cannot be ruled out in such individual cases.

Further evidence is needed before the neuroflexibility model of sleep proposed here can be considered to be validated as a sound diagnostic and treatment model, especially using only a simple single-channel monitoring and feedback site. Although the group results are encouraging, on an individual basis the diagnostic power with the current sample appears only moderate. Too many individuals within each sleep group do not neatly fall into the predicted alpha control category. It would be interesting to see how this alpha control parameter might appear in a sample group with more severe sleep concerns, such as is seen in those seeking treatment in a sleep clinic.

Despite the aforementioned noted limitations, we believe that the development of treatment routines based on evidence-based theoretical models is important for the continued development of all forms of biofeedback therapy. With that in mind, it is our hope that this current neuroflexibility model of sleep disorders will contribute to furthering the behavioral understanding and treatment of sleep disorders.

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