Appl Psychophysiol Biofeedback (2011) 36:251–264
DOI 10.1007/s10484-011-9165-y

Neurofeedback for Insomnia: A Pilot Study of *Z*-Score SMR and Individualized Protocols

Barbara U. Hammer · Agatha P. Colbert · Kimberly A. Brown · Elena C. Ilioi

Published online: 26 July 2011

Abstract Insomnia is an epidemic in the US. Neurofeedback (NFB) is a little used, psychophysiological treatment with demonstrated usefulness for treating insomnia. Our objective was to assess whether two distinct *Z*-Score NFB protocols, a modified sensorimotor (SMR) protocol and a sequential, quantitative EEG (sQEEG)-guided, individually designed (IND) protocol, would alleviate sleep and associated daytime dysfunctions of participants with insomnia. Both protocols used instantaneous *Z* scores to determine reward condition administered when awake. Twelve adults with insomnia, free of other mental and uncontrolled physical illnesses, were randomly assigned to the SMR or IND group. Eight completed this randomized, parallel group, single-blind study. Both groups received fifteen 20-min sessions of *Z*-Score NFB. Pre-post assessments included sQEEG, mental health, quality of life, and insomnia status. ANOVA yielded significant post-treatment improvement for the combined group on all primary insomnia scores: Insomnia Severity Index (ISI $p < .005$), Pittsburgh Sleep Quality Inventory (PSQI $p < .0001$), PSQI Sleep Efficiency ($p < .007$), and Quality of Life Inventory ($p < .02$). Binomial tests of baseline EEGs indicated a significant proportion of excessively high levels of Delta and Beta power ($p < .001$) which were lowered post-treatment (paired *z*-tests $p < .001$). Baseline EEGs showed excessive sleepiness and hyperarousal, which improved post-treatment. Both *Z*-Score NFB groups improved in sleep and daytime functioning. Post-treatment, all participants were normal sleepers. Because there were no significant differences in the findings between the two groups, our future large scale studies will utilize the less burdensome to administer *Z*-Score SMR protocol.

Keywords Neurofeedback · *Z*-Score · EEG · Sleep · Insomnia · QEEG · SMR · Biofeedback

Introduction

About 20–30% of adults suffer from insomnia, which is associated with increased illness, accidents, healthcare utilization, and industrial expenses and is estimated to cost $14–80 billion annually (Daley et al. 2009; National Institutes of Health (NIH) 2005). In 2005, insomnia was declared an epidemic at the NIH State-of-the-Science Conference on Manifestations and Management of Chronic Insomnia in Adults (NIH 2005) and has since increased at an alarming rate (National Sleep Foundation 2009).

The most widely used treatments for insomnia are mild sedative and hypnotic pharmaceutical agents, which are effective, but only as short-term therapies (3–6 months duration) due to their sedating effects. When these therapies are discontinued, insomnia returns (NIH 2005). Though insomnia has the highest co-morbidity with other

A summary of this research was presented at the 2010 Association for Applied Psychophysiology and Biofeedback Annual Meeting 3/24/2010–3/27/2010.

B. U. Hammer (✉) · A. P. Colbert
Department of Psychophysiology, Helfgott Research Institute, National College of Natural Medicine, 049 SW Porter Street, Portland, OR 97201-4848, USA
e-mail: barbhammer37@yahoo.com

K. A. Brown
Helfgott Research Institute, National College of Natural Medicine, 049 SW Porter Street, Portland, OR 97201-4848, USA

E. C. Ilioi
Department of Psychology, McGill University, Montreal, QC, Canada

psychological disorders, only one psychological treatment, Cognitive Behavioral Therapy (CBT), has been shown efficacious for its treatment (McCrae et al. 2010). Since there appear to be multiple causes for insomnia (NIH 2005), various treatments or combinations are likely to be most beneficial for individual patients. Here, we present an alternative psychological treatment, neurofeedback (NFB), which may serve as a stand-alone intervention or an adjunctive therapy for treating insomnia.

Neurofeedback refers to the operant conditioning of EEG brainwaves. In his 1960s studies on cats, Sterman demonstrated that not only did NFB over the sensorimotor cortex (SMR) help cats sleep better, it also offered them sustained protection against developing future epilepsy (Sterman and Clemente 1962; Sterman et al. 1970, 2010, originally written in 1969 as a NASA technical report). The latter benefit, as a potential treatment for epilepsy, became the focus of most subsequent research on SMR NFB during the next decade. In the late 1970s researchers began to focus on the impact of SMR NFB on sleep behaviors in humans (Sterman and Egner 2006).

Hauri (1981) and Hauri et al. (1982) extended Sterman's work to the study of humans with insomnia and found significant improvement resulting from NFB. He found that those who had insomnia but were relaxed at baseline benefited from SMR neurofeedback. Since that time, only one study (Cortoos et al. 2010) attempted to replicate this finding, possibly due to the burdensome nature of the training. As stated by Hauri (2008) himself: "… in the early 1980s, SMR biofeedback was cumbersome (needing up to 40 sessions in the lab to reach criterion) and the equipment too expensive for home training. We therefore gave up using SMR neurofeedback as a clinical treatment for insomnia" (p. 246).

Recent technological advances allow less expensive, portable EEG recording, rapid computer analysis of EEG patterns, instantaneous statistical determination of *Z* scores, and instantaneous feedback (reward and inhibition) of many different categories of neurophysiological activity. Recently, Cortoos et al. (2010) demonstrated that SMR NFB was superior to relaxation biofeedback in improving sleep behavior.

An additional area of interest explored in this study was the importance of individualization of NFB training protocols to target the unique underlying brain dysfunctions. This increasing emphasis on QEEG guided protocols recently stimulated the publication of an entire issue of the Journal of *Applied Psychophysiology and Biofeedback* devoted solely to this topic (Andrasik 2010). A challenge remains in that no correlation has been found between insomnia symptoms and the location of brain dysfunction, except for the few studies of SMR cited above.

Our objectives in this pilot study were to explore a new methodological approach (*Z*-Score neurofeedback) and obtain preliminary information on two distinct NFB protocols (SMR and IND) for insomnia to help us decide which approach to use in a future large-scale trial. We wondered if the IND protocol might be superior to the SMR protocol in improving insomnia, since it has been suggested by a number of clinicians who informally report that when NFB patients are treated for other conditions using QEEG guided protocols, they also report improved sleep after training. Our primary measure of improved sleep was Sleep Efficiency (SE) as measured by the Pittsburgh Sleep Quality Index (PSQI).

Methods

We used a randomized, parallel-group, single-blind experimental design in this pilot study. All treatments were conducted by the same investigator (BUH), a clinical psychologist with more than 25 years in clinical practice and 10 years in the study and practice of NFB. Random group assignment was determined with a random number generator. Participants were blinded to the type of NFB treatment protocol used for their training. The Institutional Review Board at the National College of Natural Medicine approved the study before enrollment began.

Participants and Setting

Participants were recruited between October 2008 and April 2009 through posted announcements in the general community in Portland, Oregon. Those who responded were screened in a stepwise manner: a 15-min telephone interview followed by an extended in-person screening interview with Dr. Hammer, which was conducted at the Helfgott Research Institute's Psychophysiology laboratory. The extended interview consisted of completion of a Medical History Form, five questionnaires, a structured psychological assessment, and a 45-min in-depth medical and psychological interview.

Screening Questionnaires

Medical History Form (*MHF*): A basic intake form, with "yes" or "no" prompts about medical conditions, previous operations, current medications, alcohol and tobacco use, exercise, allergies and alternative medicine was used in order to determine any concurrent medical conditions. This was followed up during the interview to confirm or deny.

Insomnia Severity Index (*ISI*) (Bastien et al. 2001): A seven question self-report instrument with demonstrated reliability and validity was used to help quantify perceived current insomnia severity for pre versus post treatment assessment. Published in 2001, the ISI is a short, screening measure suitable for use in repeated measures, which targets the past week's symptoms and consequences of insomnia consistent using the criteria in DSM-IV. This five-point Likert scale obtains ratings of 0–4 on degree of severity of the primary sleep difficulties and daytime dysfunctions, as well as on degree of associated satisfaction and distress. It is intended to be a more direct measure of insomnia than the other primary measures generally in use. A score $\geq$10 is generally considered indicative of insomnia. It is used here in conjunction with other sleep measures and the diagnostic interview.

Pittsburgh Sleep Quality Inventory (*PSQI*) (Backhaus et al. 2002): A short self-report assessment of overall sleep quality during the previous month was administered to assist in determining the insomnia diagnosis. Nineteen individual items generate seven "component" scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The sum of scores for these seven components yields one global score. The PSQI is perhaps the most widely used, well researched, psychometrically sound instrument used in sleep research (Edinger et al. 2004) and is recommended as a standard research assessment of insomnia (Buysse et al. 2006). It has a well validated cutoff score of >5 which yields a diagnostic sensitivity of 89.6% and specificity of 86.5% in differentiating good and poor sleepers, The seven component scores have a high degree of internal consistency (Cronbach's α of 0.83), with the strongest correlations in Sleep Efficiency and Subjective Sleep Quality. The test–retest reliability of global scores is 0.85. Validity is supported by its ability to distinguish insomnia patients from controls, and to a lesser extent, by concurrent polysomnography (Buysse et al. 1989).

Minnesota Multiphasic Personality Inventory-2-Revised Form (*MMPI-2-RF*) (Gervais et al. 2007): The most frequently used and well researched measure of psychopathology, the newest, restructured form of the MMPI-2 was used to help verify the absence of co-morbid mental disorders and to confirm clinical impressions of mental status.

Psychiatric Diagnostic Screening Questionnaire (*PDSQ*) (Zimmerman and Chelminski 2006): This is a validated 20-min self-report instrument, that screens for DSM-IV Axis I disorders which are commonly encountered among individuals 18 years of age and older who are routinely seen in medical and outpatient mental health settings. It was used to assist in ruling out co-morbid mental disorders.

Quality of Life Inventory (*QOLI*) (Frisch et al. 2005): This 15-min self-report instrument assesses 16 different aspects of life, including: health, goal setting, money, self-esteem, learning, recreational interest, community, and the overall quality of these entities. It is a validated scale for the condition of insomnia. The QOLI extends the description of mental health beyond the absence of psychiatric symptoms to areas of happiness and satisfaction. We believed it was important to measure an extended range of possible mental health improvements, given that we were selecting participants who would not have significant psychopathology. This is especially important when studying insomnia, because by DSM-IV, definition the symptoms must include some evidence of dysfunction resulting from troubled sleep, which is ameliorated by successful treatment. Test–retest reliability of 0.73, internal consistency of 0.79, and significant predictive validity for a variety of clinical treatments have been reported for the QOLI (Frisch et al. 2005).

Those who passed the extended screening interview and consented to participate in the study were then administered a baseline sequential, quantitative EEG (sQEEG), described below, and randomly assigned to either the IND or SMR groups.

Inclusion/Exclusion Criteria

Participants qualified for the study if they met the diagnostic criteria for insomnia disorder (307.42) according to the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, 4th edition (DSM-IV) (2000) and:

- Were between the ages of 18 and 65 years
- Scored >5 on the PSQI
- Were free of other mental disorders as determined by the MMPI-2-RF, the PDSQ and the in-depth psychological screening interview
- Were free of other medical conditions which could interfere with sleep as determined by self-report on the Medical History Form, and the in-depth sleep and psychological screening interview
- Were not taking prescription medication for insomnia disorder
- Were free of over-the-counter sleep medications, herbal substances, or recreational drugs for 2 weeks prior to and during the study
- Were not pregnant or caring for an infant, not a shift worker, and did not usually drink more than 5 cups of coffee or caffeinated drinks per day
- Expected to be available for the 3 months the study might take
- Were willing to come to the psychophysiological laboratory for an hour, twice a week for 8–9 weeks

The determination "free of other mental disorders" was made from a combination of the MMPI-2-RF profile, the

PDSQ *T*-score, and the in-depth clinical interview which included structured questions based on the PDSQ. After determining the profile to be valid using the validity scales on the MMPI-2-RF, *T* scores (using the non-gendered database) between 38 and 65 on the Higher-Order and Restructured Clinical scales were considered to be normal scores. When all of these *T* scores were within that range, and the PDSQ *T* score was within the normal range, and there was nothing contradictory in the interview, participants were judged to be "free of other mental disorder." If more than two subscale *T* scores were above 65 or below 38, the participant was excluded. When one or two subscale scores were outside the normal limits but the PDSQ *T* score and interview determined the participant was within the normal range, he/she was classified as "borderline normal" and judged "free of other mental disorders."

Sleep logs were used to assess progress and to monitor possible adverse reactions during the study. Participants were given a Sleep Log (SL) with instructions to record their sleep behavior on a daily basis: they entered the time they turned the lights out, the approximate number of times they awoke during the night, if any, and the time they got out of bed the next day. SL data are very cumbersome to analyze statistically, and since our other measures yielded rather clear-cut results, we did not perform statistical analyses this set of data.

sQEEG Recording and Analysis

An sQEEG was obtained from each participant within a week after passing the extended screening interview. Participants were fitted with a 19-channel cap to record EEG behavior (designed for this purpose by Electro-cap International, Eaton, OH). The EEG signals were obtained in sequential monitoring at four sites simultaneously for 90 seconds; this enabled all 19 sites to be observed within 7.5 min in eyes closed and eyes open conditions. The Atlantis amplifier and MiniQ devices of BrainMaster Technologies, Inc. (Bedford, OH) were used to collect the EEG signals.

The EEG signals were then imported into the NeuroGuide (v. 2.5.9) software (Thatcher 2009) and artifact was removed prior to processing by the software. This database (Thatcher et al. 2003) has a 510K clearance by the FDA. It compares each participant's scalp electrophysiological activity to a normative database of over 625 participants aged 2–82 years. This analysis yields *Z* scores for age and sex distributions for amplitude and three connectivity measures (asymmetry, coherence, and phase lag) at 19 of the 20 standard sites, for frequencies of .5–30 Hz.

Actigraphy

After the baseline EEG assessment, participants were fitted with an Actiwatch (2008) and given instructions on its operation (Philips Respironics, Bend, OR). Actigraphy, which measures wrist movement, allows for in-home use and can serve as a surrogate for polysomnography in sleep studies. Participants were instructed to wear an Actiwatch for at least 72 successive hours at baseline and after the last treatment session. However, due to numerous technical difficulties, we were unable to process the actigraphy data with the degree of precision necessary to conduct meaningful, scientifically sound, comparisons. Therefore, we have omitted these data from the analyses.

Z-Score Neurofeedback

The goal of all NFB training is to regulate any existing dysregulated brainwave patterns in order to achieve their normalization. In this study, multivariate *Z* score statistical computations are used for real-time statistical processing of the EEG and determination of deviation from normal (*Z* score) as the EEG is being produced. It employs joint time frequency analysis (JTFA) (Collura et al. 2009, 2010). Since the *Z*-Score methodology uses the same database that is employed in post-processing, it produces live assessment simultaneously with operant training without the added time burden of post processing. Post processing usually takes an additional hour of patient and technician recording time using some means for collecting the EEG data (e.g. Electro-Cap) and for analysis via a standard database. In this study both 2-channel and 4-channel training protocols were used, providing 76 (2 channel) or 248 (4 channel) *Z* scores instantaneously derived for amplitude as well as for asymmetry, coherence, and phase lag connectivity measures. *Z* scores are computed well within the time period of each computation epoch, which is 33 ms, the maximal delay in the overall system (Collura et al. 2009).

Experimental Groups

Group 1 (IND)-Z-Score Individualized Protocol

The individualized treatment protocol was based on the selection of the four highest abnormal site(s) (HAS4) from the sQEEG obtained pre- treatment and analyzed by the NeuroGuide normative database software. The IND training protocol was designed to accomplish two training goals: (1) normalization of the abnormal amplitudes of delta, theta, alpha, and beta brainwaves at the four sites (HAS4) with the highest *Z* scores greater than ±1.96, as identified by the pre-treatment sQEEG, and (2) normalization of *Z* scores of all remaining variables at those training sites, including the three most commonly used connectivity measures (asymmetry, coherence, and phase lag). Reward was contingent upon the percentage of normal variables obtained. Thus, the training protocols were designed to train the EEG brainwaves at the locations of greatest amplitude deviations from the norm. The training rewarded the correct enhancement or inhibition of those elements of the sQEEG that were beyond ±1.96 Z scores, as well as the production of an increasingly larger percentage of normal ($<\pm1.96$) Z scores. This treatment employs operant conditioning shaping techniques that modify both the percent of correct responses required (set initially at 50%) and the variance of those responses (set initially at ±1.96) (Collura et al. 2010). When the percent of variables reached > 80, the Z score limit was reduced as much as possible, thereby decreasing the variability of the correct response. Training proceeded through the sets of four (or two if that was all that remained) abnormal Z score sites, using Linked Ears as references, as allowed by the design, until normalization (Z scores $\pm.5$) occurred for at least 80% of the variables, during $\geq$80% of the training time, or the 15 sessions concluded, or the participant felt her/his insomnia had been successfully treated and chose not to continue.

Group 2 (SMR)-Z-Score SMR Protocol

The SMR treatment protocol required placement of only two electrodes: an active electrode at site Cz, and another at C4, both referenced to Linked Ears, regardless of the presence of abnormal amplitudes at any sites. Reward was contingent on:

1. the production of SMR (12–15 Hz), and the inhibition of excessive theta (4–8 Hz) and high beta (25–30 Hz) and
2. the requirement that all amplitude and connectivity measures at those sites stayed within the same normal Z score range in the same manner as described above for the IND group (Collura et al. 2010).

Reward was received only when the trainee produced a normal amount of SMR and theta and high beta, and the amplitudes of all other variables (delta, theta, alpha, and beta brainwaves), as well as the variances of the connectivity measures between the sites, were within normal limits a given percentage of the time. The initial percent of all variables required to be within the normal range was set at 50% and raised as high as possible during the available training sessions, while the percent of time that occurred was maintained at >80% and the Z scores remained within normal limits (±1.96 SD). When the percent of variables reached >80, the Z score limit was reduced as far as possible. This procedure also acts as a limiting factor to prevent artifact from being trained, since artifact will tend to be outside the normal range. Training continued until normalization (Z scores $\pm.5$) occurred for at least 80% of the training variables, during $\geq$80% of the training time for all 76 variables, or the 15 sessions concluded, or the participant felt his/her insomnia was successfully treated and chose not to continue.

Outcome Measures

The sQEEG and all screening questionnaires, except the Medical History Questionnaire and the PDSQ, also served as outcome measures. Our primary outcome measure, Sleep Efficiency (SE) as reported on the PSQI, was chosen because of its frequent use in clinical trials of insomnia and its inclusion in some insomnia diagnostic guidelines. Other exploratory outcome measures included daily sleep logs, Total Sleep Time (TST), Wake After Sleep Onset (WASO), total scores on the ISI, the PSQI, and the QOLI, the MMPI-2-RF clinical subscales, and sQEEG.

Statistical Analysis

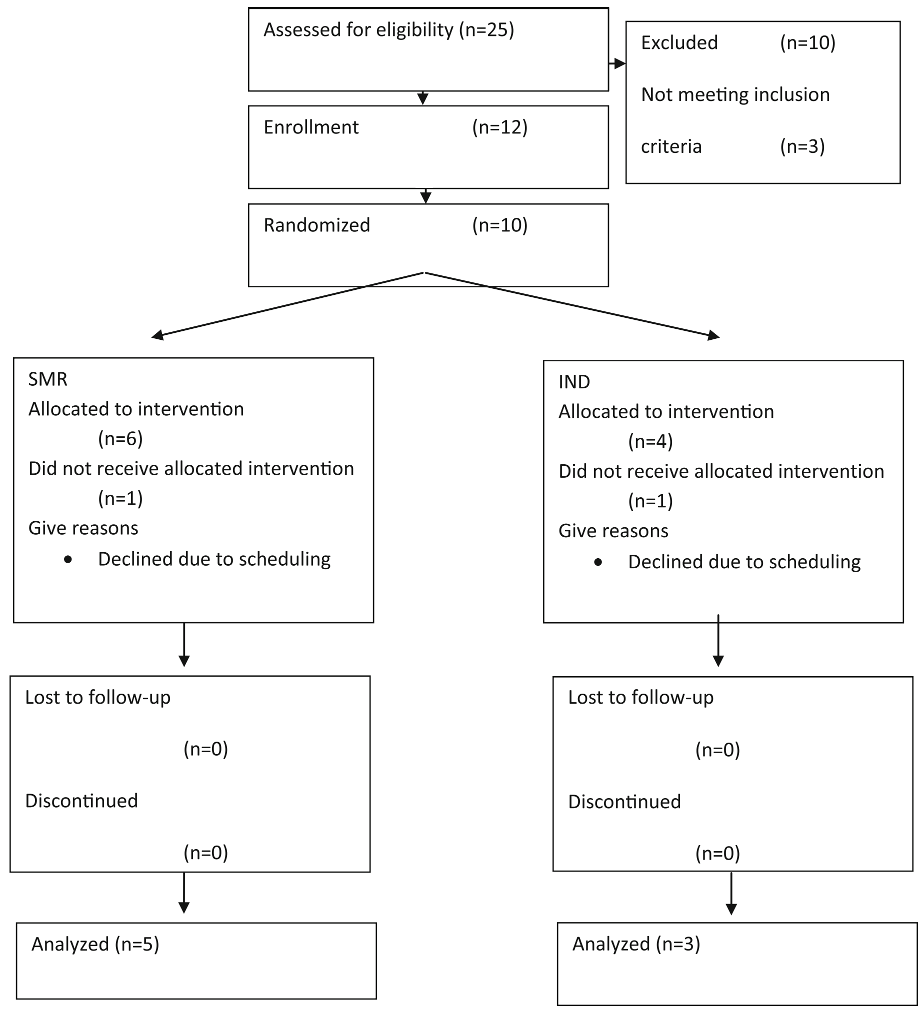
The SE data for both groups were analyzed using a 2 (between participants factor: treatment type) by 2 (within participants factor: baseline vs. post-treatment) mixed Analysis of Variance (ANOVA). The test of the between-by-within interaction determined if there were significant differences between treatments in any changes in the primary outcome measure, SE. All secondary outcome measures, except the sQEEG and WASO changes, were analyzed using these same two statistical methods. As this was a pilot study, no attempt was made to control for familial alpha error by setting more stringent alpha levels to determine significance.

A binomial test of significance was used to determine the significance of pre-post changes in sQEEG. This test is used to examine the distribution of a single dichotomous variable in small samples; it tests the difference between a sample proportion and a given proportion, assuming a normal distribution.

Results

Fifteen of the 25 individuals who responded to the study advertisements passed the initial telephone screening. Twelve of the 15 passed the extended screening interview; two of those who passed declined to enroll. Of the remaining 10 participants (Fig. 1) two dropped out of the study midway through treatment due to unanticipated personal problems that conflicted with the time demands of treatment (a residential move and family health problems).

Fig. 1 Consort flow chart



There were no adverse reactions reported. The remaining eight (mean age 49.63 years) completed the study (Table 1).

Duration of insomnia ranged from 1 year to childhood onset. Complaints, demographics and the Total scores for pre-treatment on the PSQI and ISI are listed in Table 1.

Neurofeedback Retention and Compliance

All but two of the study completers received the full 15 sessions. One felt his insomnia had abated satisfactorily after 13 sessions and chose to stop. The other participant asked originally to do the sessions once instead of twice per week, due to schedule constraints, though she was very eager to participate. We agreed to this alteration, since it was an exploratory pilot study and we anticipated that time would permit her to complete the 15 sessions within 3 months, the expected maximum duration for all participants. However, due to a variety of interruptions in schedule from illness and vacations, the 15 session limit was not completed within the time frame allotted, although she was repeatedly offered the option to make up the missed sessions. Her treatment was terminated by the investigators after 3 months during which she received nine treatment sessions. All data from these two participants who completed less than the maximum amount of training are included in all data analyses.

Success of Training

All participants reached the training goal of 80% correct within the normal range ($Z = \pm 1.96$) for 80% of the training time. The Mean percent of variables that were within normal limits at the end of training was 88, the range was from 80 to 95. The participant with the least amount of training (nine sessions) obtained the highest percentage of correct responses at termination of treatment. Though none of the participants reached the training goal of complete normalization ($Z = \pm .5$) of all training variables within the allotted, or chosen, treatment duration, by

Table 1 Complaints, demographics, global sleep scores

Subject number	Age	Sex	Duration	Symptoms	Group	Pre ISI	Pre PSQI
1	61	F	Since childhood	WASO, WE	SMR	18	14
2	50	F	1–5 years	WASO	SMR	15	11
3	34	M	5 years	SOL, WASO	SMR	19	17
4	40	M	1 year	SOL, WASO	SMR	17	16
5	54	F	20+ years	SOL, WASO, WE	IND	14	9
6	50	F	22+ years	SOL, WASO, WE	IND	12	11
7	58	F	Since Childhood	SOL, WASO, WE	SMR	28	17
8	50	M	10+ years	NS, WASO, WE	IND	14	12
Mean	49.63					17.13	13.38

WASO wake after sleep onset, *WE* wake too early, *SOL* sleep onset latency, *NS* non-restorative sleep, *SMR* *Z*-Score SMR Neurofeedback, *IND* sQEEG guided individualized *Z*-Score protocol. These pre-treatment ISI and PSQI scores are comparable to those of insomnia patients in other insomnia studies with larger patient samples (*Sleep* 2005). ISI score range = 0–28, PSQI score range = 0–21, cutoff >5

Table 2 Primary sleep and quality of life measures

Measure	Pre mean (95% CI)	Post mean–(95% CI)	*F*	*p*
ISI	17.13 (15.794,18.466)	6.56 (5.901, 7.220)	18.2	<.005
PSQI-T	13.38 (12.506, 14.254)	4.50 (4.194, 4.806)	55.6	<.0001
PSQI-SE	77.64 (74.85, 80.43)	93.18 (91.87,94.49)	15.8	<.007
QOLI	46.13 (42.908, 49.352)	52.63 (49.827, 55.433)	9.6	<.02

ISI insomnia severity index, *PSQI-T* Pittsburgh sleep quality index-total, *PSQI-SE* Pittsburgh sleep quality index total-sleep efficiency, *QOLI* quality of life index. All measures are based on the eight completers. Lower ISI and PSQI total scores are better. Higher QOLI and PSQI-SE scores are better. Significant post-treatment improvement on all measures

the end of training, half of the participants were training closer to normal, within $Z = \pm 1.5$ on all variables.

All but one in the SMR group improved their SMR *Z* scores (moved closer to $Z = 0$) at the Cz and C4 training sites. Those in the IND remained essentially the same on this variable at those two sites. All participants, except the one who stopped after 13 treatment sessions, expressed a desire for more treatment sessions.

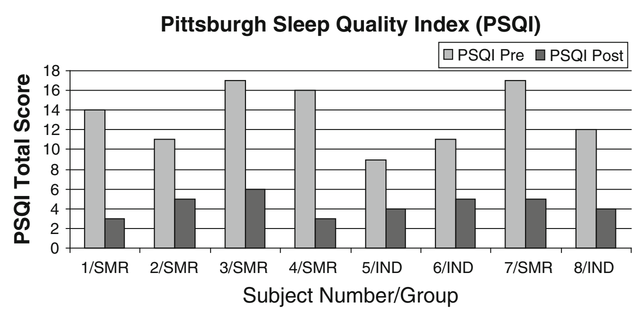


Fig. 2 PSQI pre-post change in global scores

Sleep Measures

The analyses of variance revealed that neither age, sex, PDSQ *T*-scores, nor Experimental Group were significant as covariates on the primary sleep measures (ISI and PSQI Global scores and PSQI SE), so they were dropped for subsequent statistical analyses. The combined groups score changes were significant on all of these primary measures (Table 2): ISI Global score significance was ($F_{(1,6)} = 18.2$, $p < .005$), that on the PSQI ($F_{(1,6)} = 55.6$, $p < .0001$), and that of SE ($F_{(1,6)} = 15.8$, $p < .007$). On all of these primary measures, there was no overlap of confidence intervals at 95% CI (Table 2, Figs. 2, 3).

The small number of participants precluded analysis of all types of insomnia sleep problems. Therefore, we considered only the pre- to post-treatment change of the most commonly studied sleep architecture measures: Total Sleep Time (TST), and Wake-After-Sleep-Onset (WASO) on the PSQI (Table 3, Fig. 4). The TST pre-post change for the combined group was significant ($p < .001$). Half of the participants reported an average decrease in WASO scores from "three or more times a week" down to "once or twice a week" or less. The other half reported no decrease. The four who changed included all of the men (three) and one woman (see "Discussion" below).

The individual participants change data is shown in Table 3. Sleep Efficiency increased for all participants; the

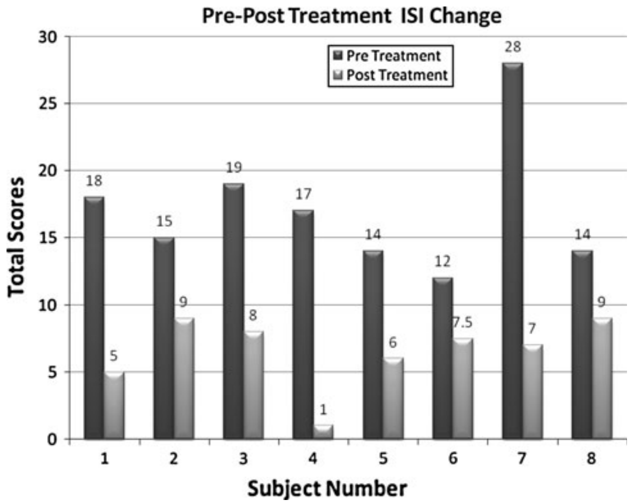


Fig. 3 Pre-post change in ISI

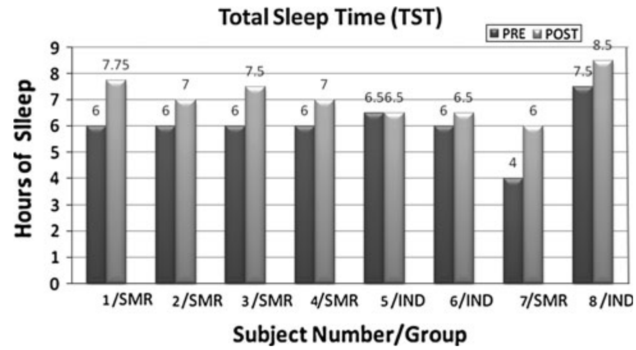


Fig. 4 Pre-post change in TST

mean increase was 15.88%, ranging from 7 to 28.6%. The average increase in TST was over an hour (61.88 min), ranging from zero (for the subject who was later diagnosed with a severe medical condition that interfered with sleep) up to two hours (120 min). As noted above, WASO improved in half the participants.

Clinical Importance of Sleep Measures

In addition to the statistical tests showing significant improvements in SE, TST, and Global sleep behavior on all measures, it is useful to look at individual changes for clinical significance. All eight participants obtained a post-treatment SE (from 85.7 to 100%) above the most commonly used cut-off of 85%.

A PSQI score of >5 (out of 21) is a well-validated cutoff for normal sleep quality; this cutoff has been shown to have high sensitivity and specificity for discriminating normal sleepers from insomnia disorder sufferers (Backhaus et al. 2002). Seven of the eight participants obtained post-treatment PSQI scores below the cutoff >5. The eighth participant obtained a score at the cutoff point (6), indicating essentially that all participants were normal sleepers post-treatment on the PSQI.

On the ISI all eight completers were below (<10) the cut-off for insomnia. There was significant clinical improvement even for the three participants whose scores were close to the cut-off (8 or 9), as observed in their large change scores: from 19 down to 8, 15–9, and 14–9. Thus, all participants post-treatment scored within the normal category on both global sleep measures.

Psychological Health and Quality of Life Changes

The improvements in sleep were accompanied by improvements in daytime mood, psychological functioning and overall quality of life. Here too, the Group interaction was not significant. There was significant improvement in quality of life for all participants as judged in the clinical interview and measured by the QOLI questionnaire, $F_{(1,6)} = 9.6$, $p < .02$, without overlap of 95% CI (Table 2).

All participants had been judged to be “free of other mental disorders” pre-treatment. Two of the original ten participants were borderline normal on the MMPI-2-RF (with one and two *T* scores at 65–70), but were determined to be functioning within normal range (as required for inclusion) based on their normal PDSQ *T*-scores and the clinical interview. We performed a second MMPI-2-RF

Table 3 Post-treatment change summary

#/Group	#Rx	Δ WASO	Final %ZOK	Increase SE%	Increase TST min.
1-SMR	15	−1	80	7.2	75
2-SMR	9	0	95	14.3	60
3-SMR	13	−2	86	27.1	90
4-SMR	15	−3	90	23	60
5-IND	15	0	85	8.7	0
6-IND	15	0	93	7	30
7-SMR	15	0	87	28.6	120
8-IND	15	−2	88	11.1	60
Mean			88%	+15.88%	+61.88 min.

#Rx number of treatment sessions, *WASO* wake after sleep onset, *%ZOK* percentage of variables required for reward, *SE* sleep efficiency, *TST* total sleep time

Table 4 Binomial tests of sQEEG pre-post changes

Abnormal waves	Pre	Post	Significance
Delta	107	42	$p < .001$
Beta	54	33	$p < .01$
Hi beta	21	17	$p < .11$
Total possible abnormal Zs = 304			

Delta: Pre to post changes 107/304 versus 42/304 yields $Z = 6.0$, $p < .001$

Beta: Pre to post 54/304 versus 33/304 yields $Z = 2.6$, $p < .01$

Hi beta: Pre to post not significant, $p < .11$, not significant

measure post-treatment primarily to determine if NFB treatments might have caused any negative (side-effect) changes in psychopathology, particularly in those participants who were initially borderline normal. The intent was not to measure improvement on the MMPI-2-RF, since these participants were already determined to be "free of other mental disorder."

The six participants who were initially judged to have unequivocally normal profiles pre-treatment, remained unequivocally normal post-treatment, using the same criteria as was used pre-treatment (see above). Two of the eight who were judged to have borderline normal profiles pre-treatment, had more clearly normal profiles at the end of treatment, as determined by the same criteria specified above for the MMPI-2-RF. Thus, none of the participants showed increased psychopathology post-treatment, which could have been a side effect of the treatment. With only two repeated measures we were unable to run statistical tests of pattern changes.

sQEEG Changes

The binomial test of significance indicated that the total number of abnormal sQEEG Z scores at baseline for all study completers exceeded the number that would be expected due to chance, $p < .001$ (Table 4). This was also true of the filtered delta (1–4 Hz) and beta (12–25 Hz) waves at baseline, but not high beta (25–30 Hz). A paired z-test revealed that for the completers, the proportion of abnormal sQEEG Z scores at all 19 sites measured was significantly lower after treatment, $p < .001$. This reduction from pre- to post- treatment in total numbers of abnormal Z scores was also significant in particular for both delta ($Z = 6.0$, $p < .001$) and beta ($Z = 2.6$, $p < .01$) bands.

Overall, there were no significant Z score changes pre- to post-treatment at any one site, in part because the specific sites of abnormality at baseline were quite varied. However, this included the sites specifically trained in the SMR Group, Cz and C4. All but two of the participants had normal Z scores ($<\pm1.96$) pre- and post-treatment at these two sites. Of the two who had SMR abnormalities at baseline, only the one in the SMR group showed normalization post-treatment. In addition, three in the SMR group showed large movements toward $Z = 0$ after treatment (Z scores from 2.44 down to .71, from 1.15 down to .82, and from -1.17 to $-.07$). These movements toward $Z = 0$ suggest that it may be the Z-Score aspect of the combined Z-Score/SMR protocol that was more powerfully related to improvements in sleep quality than was the SMR element. The three in the IND group did not show this movement toward $Z = 0$. Their post-treatment Z scores at Cz and C_4 were essentially the same or higher than pre-treatment.

Follow-up

The eight completers were contacted 6–9 months after treatment for follow-up sleep information and asked to complete the 7-item ISI on the telephone or by email. Six of the eight completers gave follow-up data; two had become severely ill with physical conditions that interfered with sleep and therefore, were not included in the follow-up data set because they no longer met the qualifications for study participation. Five of those six responders remained free of insomnia disorder, and three of those five even continued to improve over the follow-up period. The sixth responder remained essentially the same as before treatment (on ISI Pre = 12, FU = 11); she is likely to have been peri-menopausal (a condition known to be associated with sleep difficulties) and was seeking treatment to ameliorate those symptoms at the time of follow-up assessment. Since her medical condition was not definitely known, we did not disqualify her from participation and her data are included in the statistical analyses. Nonetheless, the average ISI score of those 6 reporting at follow-up was 7, below the cut-off of 10 and categorized as "no clinically significant insomnia disorder" by the test developers (Bastien et al. 2001).

Discussion

In this study we found support for Hauri's (1981 and Hauri et al.'s (1982) early findings, as well as the more recent findings of Cortoos et al. (2010), that SMR neurofeedback benefits patients with insomnia disorder. We also found that the simpler-to-administer SMR protocol led to outcomes similar to those for the individually designed, sQEEG guided protocols. We did not evaluate Hauri's theta neurofeedback protocol. Compliance in this study was consistent with the proportions reported in national surveys (NIH 2005).

Our results suggest that using updated, advanced electronic hardware and innovative *Z*-Score Neurofeedback software, daytime NFB in the laboratory is effective for the treatment of insomnia disorder. Scores on all the primary sleep and Quality of Life measures pre- to post-treatment were significant. All eight participants achieved normal or near normal sleep. Furthermore, the treatment response was sustained for at least 6–9 months at follow-up in more than half of the study completers. Of note is the finding that three participants who had insomnia disorder for many years prior to treatment improved during the follow-up period. This sustained improvement is consistent with reports by practicing clinicians who have observed sustained EEG normalization following NFB treatment of other problems/complaints.

One participant who, soon after treatment, was diagnosed with a medical condition that interfered with sleep, had no increase in TST but did improve in SE and Total sleep quality on the PSQI. She had reported that her sleep was more restorative and qualitatively better on the daily SLs, though she was not sleeping longer. It is likely that her undiagnosed medical condition was, at the time of the treatment, sufficiently advanced to affect her sleep, which would have also diminished the treatment response of the IND group as whole, because this participant was one of only three in that group.

Specific Sleep Parameters

All of the participants had primary sleep WASO problems, at baseline, which improved in four of the eight following treatment. Nonetheless, all reported improvement in SE and all but one improved in TST.

Three of the four participants whose WASO improved were males. Two of the four females who did not improve on this measure are likely to have had undiagnosed hormonal difficulties at the time of treatment. One was diagnosed post-treatment but before the 6 month follow-up with a severe medical condition that caused hormonal imbalances and interfered with sleep, and the other was diagnosed post-treatment as peri-menopausal, which also interferes with sleep. The other two women had lifestyle living arrangements that are unrelated to a physical condition but that directly interfered with their sleeping through the night and which did not change over the course of treatment. These two, however, reported that they were sleeping better when they slept and indeed both reported large increases in SE and TST (subjects 2 and 7 on Fig. 4; Table 3).

A biologically based sex difference in the etiology of some insomnia disorders or in its responsiveness to neurofeedback is possible. It is also possible (and likely) that the undiagnosed medical conditions at the time of treatment and psychological or lifestyle differences between the males and females interfered directly with sleep in this particular sample and had more of an effect upon the data given the small sample size. This is an important issue to investigate in future studies. Our review of the literature did not reveal any previously described sex differences on WASO, though there is a tendency for more women than men to report sleep difficulties (NIH 2005) and it is unclear if these are related to actual sleep differences or sleep reporting differences (AASM 2009).

sQEEG Changes

The significant pre-post sQEEG changes suggest that neurophysiological change has occurred, and that the observed changes are not solely in the self-reported changes on the questionnaires. At baseline, the EEG patterns in this insomnia group as a whole showed excessively high amplitudes of both beta and delta brainwaves. The significantly high levels of baseline delta are consistent with the overall daytime sleepiness of these participants prior to treatment (Demos 2006). The incidence of excessive delta was significantly diminished post-treatment. The significantly high levels of pre-treatment beta offer support for the hyperarousal theory of the etiology of insomnia disorder (Cortoos et al. 2006; Perlis et al. 2001).

Z-Score NFB resulted in general daytime electrophysiological normalization as measured by the NeuroGuide database, rather than solely specific frequency band changes at specific scalp sites trained. Fifteen 20 min sessions of NFB were well tolerated; these *Z*-Score protocols produced changes relatively quickly, often within $\leq$10 sessions, or less than 200 min, as indicated in the SL reports. However, we did not perform the post-treatment assessment until each participant completed the allotted or chosen number of sessions, nor did we perform it on either of the drop-outs. It would be interesting for future studies to focus on this issue in a dose–response experimental design.

Clinical Significance of Sleep Efficiency

The serious impact of insomnia disorder makes it imperative for the health and economic well-being of our society that we develop more long-term, effective treatments for insomnia disorder. Though a Sleep Efficiency cut-off of 85% is commonly used to identify individuals with insomnia disorder, this may not be the most clinically useful criterion. Cohen et al. (2009) recently found that Sleep Efficiency of less than 92% resulted in significantly less immunity than SE of 98% to even the common cold virus. The mean SE post-treatment in our study was 93.18

and the entire CI 95% range was $\geq$92, equal to the cut-off found to yield greater immunity by Cohen et al. (2009). The question of sensitivity and specificity of SE needs further clarification in future studies.

Theoretical Significance of *Z*-Score SMR NFB

The primary function of sensorimotor neurofeedback in the treatment of insomnia disorder might lie in its providing step-wise assistance to help the brain gradually slow down from beta to low beta as it moves toward its most restorative brainwave state: slow wave activity (SWA) or delta wave sleep. Tononi and Cirelli (2006) offer an interesting and important study of the possible underlying brain mechanism of sleep. Their hypothesis suggests that the primary role of “sleep is to downscale synaptic strength to a baseline level that is energetically sustainable, makes efficient use of gray matter space, and is beneficial for learning and memory” (Tononi and Cirelli 2006, p. 49).

Stage II sleep is characterized by SMR, sensorimotor LoBeta (12–15 Hz) brainwaves, and normal sleep requires this stage before satisfactory progression to deeper, restorative sleep stages. Because these participants with insomnia disorder had extremely high ($z > 1.96$) amplitudes of beta pre-treatment (similar to that reported by Perlis et al. 2001), perhaps a benefit of SMR NFB lies in its providing extra training to support and manage this slow-down process toward SWA. Following sleep, synaptic weight returns (downscales) to a baseline level, creating homeostasis and aiding learning and memory (Tononi and Cirelli 2006).

Practical Significance of *Z*-Score SMR NFB

Of particular interest is our finding that the amplitude of SMR did not increase in the *Z*-Score SMR group but became more normalized in all but one of the five participants. The amplitude of SMR in the IND group remained essentially unchanged. The normalization of SMR in the *Z*-Score SMR group suggests that the *Z*-Score component may have accounted for a greater percentage of the improvement in sleep quality than that resulting from rewarding of SMR itself. This is an important practical and theoretical question that could be addressed further in future studies.

Our finding utilizing *Z*-Score protocols, that the SMR treatment protocol was as effective as the individualized protocols, could have positive implications for *Z*-Score SMR NFB becoming an additional, readily available, easy-to-administer option for treating insomnia. It is significantly less burdensome for both patient and health care provider, given that it requires neither a full QEEG assessment in order to guide the protocol decision, nor expertise in designing a guided protocol. If this finding is replicated in future studies, it holds promise as a standard protocol that could potentially be used for many patients.

Absence of Adverse Events

Participants in this study reported no adverse events. This is in accordance with our informal review of forums of *Z*-Score providers who have reported no adverse events in their clinical practices since the treatment became available in 2005, with one exception that occurred when it was used by an untrained, unsupervised technician. In addition, Ros et al. (2009) has previously demonstrated that traditional SMR training improved microsurgical skills in medical students and Hoedlmoser et al. (2008) showed that it improved both sleep and declarative learning in normal college students. Neither study screened for excessive baseline SMR, nor did they report any adverse effects from rewarding SMR regardless of baseline amplitudes.

These findings suggest it is unlikely to be harmful to use *Z*-Score SMR NFB without first determining if there is SMR elevation at baseline, because the *Z*-Score part of our protocol functions to maintain the amplitude of SMR within normal limits. It is possible that *Z*-Score NFB, which reinforces movement toward $Z = 0$ for healthy individuals, may generally be safer than other forms of NFB in which the wrong choice of frequency, parameter, or training site can lead to problems (Hammond and Kirk 2008; Whitsett et al. 1982).

Quality of Life

Daytime dysfunction resulting from lack of sleep is one of the criteria for the DSM-IV diagnosis of insomnia, yet the present study is one of the few on insomnia in which daytime mood and functioning were systematically measured pre and post-treatment. If daytime dysfunction does not diminish post-treatment it could not be said that an individual obtained full recovery from insomnia even with normal scoring on all sleep measures. Therefore, we suggest that future studies of insomnia treatments utilize measures of quality of life and psychological health, such as the QOLI and MMPI-2-RF or similar, psychometrically sound, pre-post assessment tools.

Z-Score Neurofeedback

Like CBT, the only psychological treatment for insomnia disorder that has been classified as efficacious (McCrae et al. 2010), *Z*-Score NFB is non-invasive and non-pharmacological (Bootzin and Perlis 1992) and may not need to be limited to short term use. Both CBT and *Z*-Score NFB may have longer lasting effects than hypnotic medications, which generally need to be limited to short term use due to adverse reactions or side effects from prolonged drug usage. Long term insomnia sufferers, like most of the subjects in this study, need long term solutions. CBT and NFB are training methods, and as such, the behavioral changes take much longer to extinguish than the half-lives of benzodiazepine sleep medications.

Though the training time allotted for effective *Z*-Score NFB in this study was generally about the same as that offered in most CBT interventions, most participants in our study improved their sleep behaviors after less than ten sessions (i.e. <200 min) of *Z*-Score NFB. Furthermore, all patients in this study achieved post-treatment normal sleep, whereas most patients receiving CBT for insomnia disorder do not become good sleepers, and the effect sizes are smaller than those treated with CBT for other conditions (Harvey and Tang 2003). The post-treatment (extinction) effects of our interventions in this small group of participants lasted longer than those of the usual course of both CBT and pharmacological treatments for insomnia disorder.

In general, NFB treatment improvements may be easier to maintain post-treatment because NFB does not require post-treatment conscious, intentional adherence to specific rules or behaviors on the part of the patient, and the changes occur at the electrophysiological level (if they endure over extended time periods, as suggested here). NFB requires only enough patient motivation to attend the training sessions and allow one's brain to learn to regulate itself by responding to feedback. Its usefulness may well derive from the neurophysiological changes in dysregulated EEG patterns, which may indeed be the underlying cause of at least some types of insomnia disorder, rather than treating or changing lifestyle habits and behaviors.

As it appears that the *Z*-Score training component of our combined SMR/*Z*-Score protocol may have contributed more to improving sleep quality, it would be especially interesting in future studies to explore the comparison of *Z*-Score SMR training on the sensorimotor strip with traditional SMR NFB.

Limitations of This Study and Recommendations for Future Research

Inadequate Actigraphy Data

The Standards of Practice Committee of the American Academy of Sleep Medicine stated that Actigraphy was useful in assessing treatment response in patients with insomnia disorder (Morgenthaler et al. 2007). Earlier, Vallieres and Morin (2003) and Lichstein et al. (2006) found that the Actigraphy was useful for measuring insomnia disorder treatment response. However, after the Practice Parameters were published, reports on normal participants under various sleep conditions questioned that conclusion because of the very low ability of Actigraphy to detect wakefulness (Paquet et al. 2007). Because the Actiwatch is worn like a wrist watch and measures activity of wrist movements, it gives inaccurate information if the person is awake but lying still, thus overestimating sleep duration. This behavior, however, is common among people with insomnia during those periods when they are trying, but unable, to fall asleep.

Sample

This pilot study has a number of obvious limitations including the small sample size, the lack of a control group, and the lack of more objective sleep measures. Our statistical tests of significance are likely to be inflated for a variety of reasons. Evaluating only those who complete treatment ignores the data of those who did not, for whatever reason, and some of those reasons might have been related to treatment issues. In this study, that is particularly important given that all who dropped out, whether just prior to treatment onset or partway through, did so for reasons related to the high schedule demands of 2–4 h weekly for up to 9 weeks. Performing repeated measures can also lead to practice and habituation effects that might inflate apparent reductions in symptoms. In addition, one or two extreme responders or nonresponders can overly influence the outcome in a small group as a whole, as may have been the case in our IND group (see above "Discussion"). However, this exploratory study has provided enough preliminary evidence of the effectiveness of *Z*-Score NFB to warrant further, more controlled investigations.

Lack of Control Group

Animal studies that have demonstrated brainwave and behavioral changes in cats and primates as a function of EEG biofeedback offer evidence suggesting that the changes observed in the present pre-post *Z*-Score NFB study are not solely a function of human intention or expectation, i.e. placebo (Philippens and Vanwersch 2010). In addition, Ros et al. (2010) have recently provided direct evidence of brain changes following just 30 min of alpha inhibition NFB. Thus, the QEEG changes observed are likely to be objective measures of changed electrophysiology that occurred pre- to post-treatment. Nonetheless, a credible sham intervention will be used in our future studies.

Shortcomings of These Z-Score Individualized Protocols

Further exploration of various Z-Score protocols for insomnia disorder using complete sets of QEEG Z score connectivity measures (asymmetry, coherence, and phase) to guide the individually designed NFB protocols is recommended in order to fully examine this treatment variable. Within the scope of this study, only amplitude-based protocols could be explored. However, it is sometimes the case that a QEEG record will show abnormalities, or the highest abnormalities, solely in the connectivity Z scores. For this reason, it would be useful to obtain the Z score connectivity measures between all 19-scalp sites in order to explore this dimension in a training protocol. Nonetheless, the live Z-Score protocols used herein simultaneously train the primary connectivity measures between the chosen EEG sites, which are often, though not always, the same sites as those with extreme amplitude measures. It would also be valuable to study and compare the individual components of our combined SMR/Z-Score protocol to understand better the contributions to change of each of the two parts.

Future Directions

The limitations of the Actiwatch in measuring sleep in patients with insomnia disorder are discussed above. Portable polysomnography has recently been developed that may prove to be more accurate than Actigraphy for measuring sleep architecture, and is easily adaptable to in-home, normal life use similar to the advantage of actigraphy. Thus, we suggest that future studies of insomnia disorder use three nights of portable polysomnography, rather than the often used 3 days (72-h) of actigraphy, to gain an objective measure of sleep, unless the newer versions of the Actiwatch are able to demonstrate more clearly their accuracy in measuring wakefulness.

Conclusions

This exploratory pre to post comparison study of Z-Score NFB for insomnia disorder revealed reductions in insomnia which warrant further investigation. We found that it is feasible to recruit and retain compliant participants for such a study. QEEG, QOLI, and both measures of sleep (ISI and PSQI) used here, are likely to capture clinical change related to NFB treatment. Both the SMR protocol and the IND protocol reduced symptoms of insomnia. The SMR protocol is considerably easier to administer, so it will be used as the intervention in our future trial.

Acknowledgments The authors are grateful to the Helfgott Research Institute of the National College of Natural Medicine in Portland, Oregon for its generous support of this research. We especially appreciate the assistance of William L. Gregory and Heather Jaskirat Wild on the statistical analyses, Mark L. Smith and Nancy Wigton on the design of the neurofeedback protocols, and the generous support of our research assistants, Sean E. Griffith and Tineke Malus. In addition, we thank all participants in this study, including those who took the time to complete the telephone screening and the extended screening sessions but who were not offered the opportunity to participate and receive treatment.

Conflict of interest None of the authors has any financial conflicts of interest.

References

Actiwatch [Apparatus and Software]. (2008). Bend, OR: Philips Respironics.

American Academy of Sleep Medicine (2009, October 1). Elderly women sleep better than they think, men sleep worse. *ScienceDaily*. Retrieved December 18, 2009, from http://www.sciencedaily.com/releases/2009/10/091001081207.htm.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Andrasik, F. (2010). Editor's note: Focus on QEEG… and EEG. *Applied Psychophysiology and Biofeedback, 35*(1), 1.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test-retest reliability and validity of the Pittsburgh sleep quality index in primary insomnia disorder. *Journal of Psychosomatic Research, 53*(3), 737–740.

Bastien, C. H., Vallieres, A., & Morin, C. M. (2001). Validation of the insomnia disorder severity index as an outcome measure for insomnia disorder research. *Sleep Medicine, 2*(4), 297–307.

Bootzin, R. R., & Perlis, M. L. (1992). Nonpharmacologic treatments of insomnia disorder. *Journal of Clinical Psychiatry, 53*(Supplement), 37–41.

Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Lichstein, K. L., & Morin, C. M. (2006). Recommendations for a standard research assessment of insomnia disorder. *Sleep, 29*(9), 1155–1173.

Buysse, D. J., Reynolds, C. F., I. I. I., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213.

Cohen, S., Doyle, W. J., Alper, C. M., Janicki-Deverts, D., & Turner, R. B. (2009). Sleep habits and susceptibility to the common cold. *Archives of Internal Medicine, 169*(1), 62–67.

Collura, T., Guan, J., Tarrent, J., Bailey, J., & Starr, R. (2010). EEG biofeedback case studies using live *z*-score training and a normative database. *Journal of Neurotherapy, 14*(1), 22–46.

Collura, T., Thatcher, R., Smith, M. L., Lambos, W., & Stark, C. (2009). *EEG biofeedback training using live z-scores and a normative database*. Philadelphia: Elsevier.

Cortoos, A., De Valck, E., Arns, M., Breteler, M. H., & Cluydts, R. (2010). An exploratory study on the effects of tele-neurofeedback and tele-biofeedback on objective and subjective sleep in patients with primary insomnia disorder. *Applied Psychophysiology and Biofeedback, 35*(2), 125–134.

Cortoos, A., Verstraeten, E., & Cluydts, R. (2006). Neurophysiological aspects of primary insomnia disorder: Implications for its treatment. *Sleep Medicine Review, 10*(4), 255–266.

Daley, M., Morin, C. M., LeBlanc, M., Gregoire, J. P., & Savard, J. (2009). The economic burden of insomnia disorder: Direct and

indirect costs for individuals with insomnia disorder syndrome, ainsomnia disorder symptoms, and good sleepers. *Sleep, 32*(1), 55–64.
Demos, J. N. (2006). DCN-128 (V. 1.0) [Computer Software] Brattleboro, VT: EEG Vermont. Retrieved from http://eegvermont.com/.
Edinger, J. D., Means, M. K., Stechuchak, K. M., & Olsen, M. K. (2004). A pilot study of inexpensive sleep-assessment devices. *Behavioral Sleep Medicine, 2*(1), 41–49.
Frisch, M. B., Clark, M. P., Rouse, S. V., Rudd, M. D., Paweleck, J. K., Greenstone, A., et al. (2005). Predictive and treatment validity of life satisfaction and the quality of life inventory. *Assessment, 12*(1), 66–78.
Gervais, R. O., Ben-Porath, Y. S., Wygant, D. B., & Green, P. (2007). Development and validation of a response bias scale (rbs) for the mmpi-2. *Assessment, 14*(2), 196–208.
Hammond, D. C., & Kirk, L. (2008). First, do no harm: Adverse effects and the need for practice standards in neurofeedback. *Journal of Neurotherapy, 12*(1), 79–88.
Harvey, A. G., & Tang, N. K. (2003). Cognitive behaviour therapy for primary insomnia disorder: Can we rest yet? *Sleep Medicine Review, 7*(3), 237–262.
Hauri, P. (1981). Treating psychophysiologic insomnia disorder with biofeedback. *Archives of General Psychiatry, 38*(7), 752–758.
Hauri, P. J. (2008). EEG Biofeedback in the treatment of insomnia: A historical perspective. *Applied Psychophysiology and Biofeedback, 33*(4), 246.
Hauri, P. J., Percy, L., Hellekson, C., Hartmann, E., & Russ, D. (1982). The treatment of psychophysiologic insomnia disorder with biofeedback: A replication study. *Biofeedback and Self Regulation, 7*(2), 223–235.
Hoedlmoser, K., Pecherstorfer, T., Gruber, G., Anderer, P., Doppelmayr, M., Klimesch, W., et al. (2008). Instrumental conditioning of human sensorimotor rhythm (12–15 Hz) and its impact on sleep as well as declarative learning. *Sleep, 31*(10), 1401–1408.
Lichstein, K. L., Stone, K. C., Donaldson, J., Nau, S. D., Soeffing, J. P., Murray, D., et al. (2006). Actigraphy validation with insomnia disorder. *Sleep, 29*(2), 232–239.
McCrae, C. S., Taylor, D. J., Smith, M. T., & Perlis, M. L. (2010). The future of behavioral sleep medicine: A report on the presentations given at the Ponte Vedra behavioral sleep medicine consensus conference, March 27–29, 2009. *Behavioral Sleep Medicine, 8*(2), 74–89.
Morgenthaler, T., Alessi, C., Friedman, L., Owens, J., Kapur, V., Boehlecke, B., et al. (2007). Practice parameters for the use of actigraphy in the assessment of sleep and sleep disorders: An update for 2007. *Sleep, 30*(4), 519–529.
National Institutes of Health. (2005). State-of-the science conference statement on manifestations and management of chronic insomnia disorder in adults, June 13–15, 2005. *Sleep, 28*(9), 1049–1057.
National Sleep Foundation. (2009). Sleep in America poll, summary of findings. Retrieved from www.sleepfoundation.org.
Paquet, J., Kawinska, A., & Carrier, J. (2007). Wake detection capacity of actigraphy during sleep. *Sleep, 30*(10), 1362–1369.
Perlis, M. L., Kehr, E. L., Smith, M. T., Andrews, P. J., Orff, H., & Giles, D. E. (2001). Temporal and stagewise distribution of high frequency EEG activity in patients with primary and secondary insomnia disorder and in good sleeper controls. *Journal of Sleep Research, 10*(2), 93–104.
Philippens, I. H., & Vanwersch, R. A. (2010). Neurofeedback training on sensorimotor rhythm in marmoset monkeys. *Neuroreport, 21*(5), 328–332.
Ros, T., Moseley, M., Bloom, P. A., Benjamin, L., Parkinson, L. A., & Gruzelier, J. H. (2009). Optimizing microsurgical skills with EEG neurofeedback. *BMC Neuroscience, 10*, 87. doi:10.1186/1471-2202-10-87.
Ros, T., Munneke, M. A., Ruge, D., Gruzelier, J. H., & Rothwell, J. C. (2010). Endogenous control of waking brain rhythms induces neuroplasticity in humans. *European Journal of Neuroscience, 31*(4), 770–778.
Sterman, M. B., & Clemente, C. D. (1962). Forebrain inhibitory mechanisms: Sleep patterns induced by basal forebrain stimulation in the behaving cat. *Experimental Neurology, 6*, 103–117.
Sterman, M. B., & Egner, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. *Applied Psychophysiology and Biofeedback, 31*(1), 21–35.
Sterman, M. B., Howe, R. C., & Macdonald, L. R. (1970). Facilitation of spindle-burst sleep by conditioning of electroencephalographic activity while awake. *Science, 167*(921), 1146–1148.
Sterman, M. B., LoPresti, R. W., & Fairchild, M. D. (2010). Electroencephalographic and behavioral studies of monomethyl hydrazine toxicity in the cat. *Journal of Neurotherapy, 14*(4), 293–300.
Thatcher, R. (2009). NeuroGuide (2.5.9) [Computer software]. St. Petersburg, FL: Applied Neuroscience, Inc. Retrieved from http://www.appliedneuroscience.com/.
Thatcher, R., Walker, R., Biber, C., North, D., Curtin, M., & Curtin, R. (2003). Sensitivity and specificity of an EEG normative database: Validation and clinical correlation. *Journal of Neurotherapy, 7*(3/4), 87–121.
Tononi, G., & Cirelli, C. (2006). Sleep function and synaptic homeostasis. *Sleep Medicine Review, 10*(1), 49–62.
Vallieres, A., & Morin, C. M. (2003). Actigraphy in the assessment of insomnia disorder. *Sleep, 26*(7), 902–906.
Whitsett, S. F., Lubar, J. F., Holder, G. S., Pamplin, W. E., & Shabsin, H. S. (1982). A double-blind investigation of the relationship between seizure activity and the sleep EEG following EEG biofeedback training. *Biofeedback and Self-Regulation, 7*, 193–209.
Zimmerman, M., & Chelminski, I. (2006). A scale to screen for DSM-IV Axis I disorders in psychiatric out-patients: Performance of the Psychiatric Diagnostic Screening Questionnaire. *Psychological Medicine, 36*(11), 1601–1611.