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Dissociable changes in sleep architecture with mindfulness and sleep hygiene intervention in older adults: secondary and exploratory analysis of polysomnography data from the Mindfulness Sleep Therapy (MIST) trial

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ABSTRACT

Objectives: We conducted a secondary analysis of the Mindfulness Sleep Therapy study, a randomized controlled trial testing Mindfulness-Based Therapy for Insomnia (MBTI) against a sleep hygiene education and exercise program (SHEEP). We investigated whether the interventions led to changes in sleep macroarchitecture (N2, N3 and REM), and microarchitecture (sleep fragmentation, slow wave activity, spectral band power) measured by ambulatory polysomnography (PSG).
Methods: 48 MBTI and 46 SHEEP participants provided usable PSG and subjective sleep quality data both pre- and post intervention. The interventions consisted of 8 weekly 2-hour group sessions, and daily practice. PSG data were staged according to the American Academy of Sleep Medicine criteria by 2 technicians blind to time point and condition. Repeated-measures ANOVA and permutation analysis were used to test for differences over time and between the interventions.
Results: Self-reported sleep quality improved in both study groups. We observed significant increases in N2 in MBTI but not SHEEP (p = .045), and significant increases in N3 in SHEEP but not MBTI (p = .012). No significant differences over time or between group were observed in N1, REM, or sleep fragmentation. Higher frequency non-REM EEG power decreased in SHEEP but not MBTI. Slow wave activity and slow wave activity dissipation did not differ over time or between groups. Among all variables, significant time by group interactions were observed in only N3 and non-REM alpha power.
Conclusions: MBTI and sleep hygiene education had different effects on sleep macro and microarchitecture, suggesting that the underlying mechanisms of mindfulness training in improving sleep quality may differ from traditional interventions.

Introduction

Poor sleep has wide-ranging consequences for health and well-being.[1] The American Academy of Sleep Medicine[2] recommends behavioral interventions as the initial approach to management of poor sleep quality caused by insomnia disorder. While cognitive behavioral therapy for insomnia (CBT-I) is the current frontline option, mindfulness-based approaches are emerging as an effective alternative; randomized controlled studies[3] and meta-analyses[4,5] have shown that mindfulness-based interventions (MBIs) have a large and sustained effect in improving scores on self-report measures such as the Pittsburgh Sleep Quality Index (PSQI).

Mindfulness is commonly defined as a psychological state characterized by attention to present-moment experience with accompanying attitudes of acceptance and nonjudgment.[6] An influential theoretical framework suggests that mindfulness relieves insomnia symptoms and improves sleep quality via a metacognitive model,[7] which encourages knowledge and awareness of one's own cognitive processes.[8] In the context of sleep, mindfulness may help individuals

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to disengage from dysfunctional thoughts during the presleep period, decreasing levels of cognitive hyperarousal.[7] Supporting this model, studies have shown that presleep cognitive arousal decreases following a relatively brief period of mindfulness training,[9] and reductions in cognitive arousal are a mediator of the benefits of mindfulness on subjective sleep quality.[10]

To date, only a handful of studies have reported on the effects of short-term mindfulness training on polysomnography (PSG), the current gold-standard objective measure of sleep.[11-14] These studies have generally shown that MBIs do not significantly increase deep sleep (ie, N3 duration[11-14]), a surprising finding given their robust effects in improving subjective sleep quality. However, these studies were limited by their small sample size (n = 24-50), nonspecificity of the mindfulness intervention to sleep, and/or lack of an active control group.[11,12]

Paradoxically, several papers have suggested that MBIs may even lead to *increases* in high-frequency EEG power during non-rapid eye movement (nREM) sleep,[14] a marker typically associated with nocturnal arousal. Using high-density PSG, Ferrarelli et al[15] studied a group of highly experienced Buddhist meditators and found higher parieto-occipito nREM gamma power in this group compared with meditation-naïve controls. This increase was correlated with the time spent practicing meditation daily. In secondary analysis of a randomized trial, Goldstein et al[14] reported that Mindfulness-Based Therapy for Insomnia (MBTI) participants showed increases in nREM gamma following the intervention, while nREM beta increased in a combined analysis of 2 different mindfulness groups (MBTI and mindfulness-based stress reduction[6]). These counterintuitive findings also bear replication and explication in larger samples.

The Mindfulness as Sleep Therapy (MIST) study is a preregistered randomized controlled trial of 127 older adults with sleep disturbances conducted in Singapore. It is the first well-powered trial to test manualized MBTI, and includes objective sleep measurements among its primary outcomes. The primary analysis[16] demonstrated that MBTI was superior to a sleep hygiene education and exercise program (SHEEP) in reducing symptoms of insomnia severity (Cohen's effect size d = -1.27), equivalent to SHEEP in improving subjective sleep quality (Pittsburgh Sleep Quality Index (PSQI); d = -1.19), had significant effects in reducing actigraphically measured sleep onset latency (SOL; d = -0.25 and wake after sleep onset (WASO; d = -0.30), and significantly reduced PSG-measured WASO (d = -0.26) but not SOL.

The current paper reports on secondary analysis of the completer-case PSG data, focusing on changes in sleep stages, sleep fragmentation, and PSG spectral analysis. We were particularly interested in replicating the results of the experiments discussed above, hypothesizing that slow-wave sleep would be unchanged in MBTI (with decreases in SHEEP) and that high-frequency EEG activity would increase in MBTI compared with the control condition.

Participants and methods

PSG data used in this study were collected during the MIST trial, the detailed description of which is reported elsewhere.[16] A total of 150 adults above 50 years of age were recruited of which 127 were randomized to either MBTI[13,17] or the active control SHEEP. We analyzed data from participants who contributed usable PSG data at both pre- and postintervention time points. This study was approved by both the SingHealth Centralised Institutional Review Board and National University of Singapore Institutional Review board and was conducted in accordance to the 1965 Helsinki declaration and its later amendments. Informed consent was obtained from all participants prior to enrolment.

Interventions

Both MBTI and SHEEP consisted of 8 weekly, 2-hour-long classes conducted in groups of 6-15 participants. The amount of home practice and contact time with instructions were approximately equivalent between the 2 interventions. Instructors for both intervention arms were given a syllabus to follow to ensure fidelity to the treatments.

Manualized MBTI[17] was administered with slight changes to suit the local context. Classes typically started off with a formal mindfulness practice, (eg, sitting meditation, body scan, mindful eating or mindful movement). The details of these practices are described in Kabat-Zinn.[6] This was followed by group discussions of participants' experiences during the previous week. Most classes included a didactic component regarding sleep difficulties (eg, models of the etiology of insomnia). Participants were introduced to the concept of sleep hygiene[18] during week 2, and sleep restriction therapy[19] and stimulus control[20] from week 3 onwards. For home practices, participants were given guided audio tracks and readings. At the start of the course, participants were encouraged to practice for around 20-30 minutes a day for at least 6 days per week. Daily practice time was increased incrementally to 45 minutes per session over the course of the intervention.

The active control intervention for the study (SHEEP) was developed by clinical psychologists from the Singapore General Hospital. The course aimed at improving sleep habits and changing the sleep environment to facilitate better sleep. Classes consisted of a didactic component, group discussions and exercises (eg, progressive muscle relaxation, diaphragmatic breathing and stretching movements) which promote sleep. Participants were also taught physical (isometric) and stretching exercises, and encouraged to do these at home. SHEEP participants were encouraged to practice daily for the same amount of time as the MBTI group and were given guided audio tracks and instructional booklets to follow.

PSG data

Two nights of polysomnography data (1 night each pre- and post-intervention) were collected at participants' homes using the portable SOMNOtouch RESP (SOMNOmedics GmbH, Randersacker, Germany). Home visits were performed by the same staff when possible and on the same day of the week for each participant.

A 10-channel montage (A1, A2, C3, C4, Cz, Fpz, EOG-left, EOG-right, 2 chin electrodes) following the international 10-20 system with online reference at Cz, ground at Fpz, sampled at 250 Hz. The PSG device was set up by 2 staff members approximately 60 minutes before the participants' preferred bedtime, which could vary between study sessions. Impedance of all electrodes was ascertained to be below 5kΩ before the staff left.

PSG data were extracted using DOMINO light software (version 14.0; SOMNOmedics, GMBJm Randersacker, Germany) and exported to be scored using FASST (http://www.montefiore.ulg.ac.be/~phillips/FASST.html). PSG data were assessed by visual inspection and a repeat sleep recording was requested if the data quality was deemed unacceptable. Bilateral electrooculography (EOG), bipolar submental electromyography (EMG) and central cross hemispheric re-referenced central electrodes (C3-A2, C4-A1) were used to assess sleep stages according to the AASM standard.

Time in bed (TIB) was calculated according to the bedtime and wake time specified by the participants, SOL was determined by the first nonwake and non-N1 sleep epoch. WASO is the total duration of wake between sleep onset and wake time. Total sleep time (TST) was calculated by the summation of all epochs scored between sleep onset and wake time excluding WASO. Sleep efficiency was calculated as

$$\frac{\text{Total sleep time}}{\text{Tme in bed}} \times 100.$$

Sleep recordings were scored by 2 trained staff members. Two thirds of all available nights were randomly selected to be scored by either member, allowing for 1/3 of all data to be double scored and inter-rater reliability calculated. All data labels were removed prior to scoring so that scorers were blind to condition and time point. The average of the sleep variables which were double-scored are reported along with the individually scored nights.

We computed sleep fragmentation using the weighted transition sleep fragmentation index (WTSFI[21]) for ease of calculation and its similarity to other fragmentation indices such as the arousal index.[21] Each stage of sleep is weighted (wake = 0, N1 = 3, N2 = 4, N3 = 6, and REM = 3) and a change in sleep stage is calculated by subtracting its weight from the previous stage. The sum of transitions into lighter sleep stages is divided by the total duration of sleep, then divided by the overall mean weight of the night. As the WTSFI was based off the R&K sleep scoring criteria, the average weight used for the R&K stage 3 and 4 was used for the ASSM stage 3 in this analysis.

Power spectra estimates for NREM (excluding N1) in the delta (1-4 Hz), theta (4-8 Hz), alpha (8-14 Hz), sigma (12-16 Hz), beta (16-25 Hz) and gamma (25-35 Hz) power for each PSG recording[14] were calculated using the pwelch function in EEGlab (6-second sliding window with no overlaps). Epochs that exceeded 3 SD in the delta range and 2 SD in the gamma range in a 21-epoch window were marked as artifacts and excluded from analysis.[22] Four additional metrics - root mean square and the 3 Hjorth parameters[23] (activity, mobility, and complexity) were applied iteratively 3 times to the remaining N2 and N3 epochs, and epochs that exceeded +- 2 SD of each metric in each iteration were removed.[24]

As participants were allowed to keep to their habitual sleep schedule and TIB differed across participants, sleep staging in minutes and sleep stages as a percentage of TST were both calculated and tested. Non-REM SWA power (delta band) over the night was broken down into quarters starting from SOL to the last sleep epoch for each participant and normalized over the total delta power each night to test for differences in dissipation between groups.

Subjective measures

Subjective sleep quality was assessed using the PSQI[25] and the Insomnia Severity Index (ISI[26]). We also collected self-reported ratings of sleep quality on the night that the PSG was collected using visual analogue scales, querying how well participants slept, how rested they felt and how their sleep compared with their usual sleep quality. Presleep arousal was measured using the presleep arousal scale (PSAS[27]), and anxiety was measured using the state-trait anxiety inventory (STAI[28]).

Statistical analysis

Statistical analysis was conducted with IBM SPSS Statistics 26 (Armonk, NY: IBM Corp). 2 × 2 repeated-measures ANOVAs were used to compare within- subject changes (time; ie, before and after intervention) between groups (MBTI vs. SHEEP), for the main sleep outcomes, sleep stages, and fragmentation index, absolute power (μV/Hz) in 6 frequency bands, and delta power over 4 quarters overnight. All models were also run with age as a covariate (Supplementary Table 1), with no material differences observed from the main findings from the ANOVAs. Permutation analysis[29] was performed using 5000 bootstrapped samples to obtain estimates of the effect sizes and confidence intervals associated with change in these variables for each intervention.

Results

Of the 127 participants who were randomized into the intervention groups (Fig. 1), 94 participants (mean age = 61.5 ± 6.44, 35 males, MBTI = 48, SHEEP = 46) provided data for all measures at both study time points. No significant differences were found in the demographic and clinical characteristics between the groups (Table 1). Descriptive values for all PSG-derived variables are reported in Table 2.

We found no baseline PSG macroarchitectural differences in TIB, SOL, WASO, TST, SE and duration spent in N1, N2, N3 and REM between completers and participants excluded from analysis (Supplementary Table 2).

Inter-rater reliability

Two-way mixed, absolute-agreement intraclass correlations were performed to assess reliability for sleep stages between the 2 raters

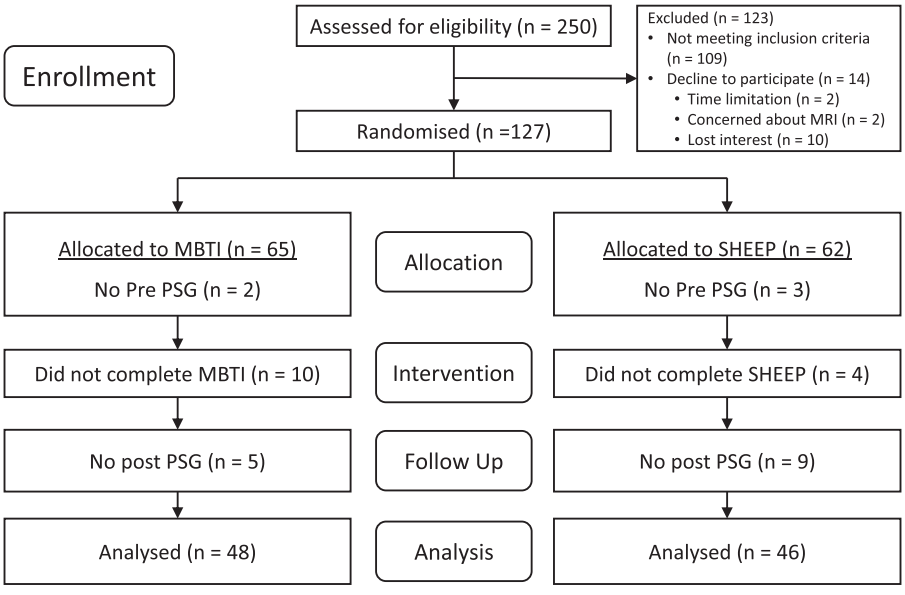

Figure 1. Consort chart depicting participant flow for PSG completers. MBTI, Mindfulness Based Therapy for Insomnia; SHEEP, sleep hygiene exercise and education program; PSG, polysomnography.

Table 1
Demographics of PSG completers by intervention group and total sample

	MBTI (n = 48)		SHEEP (n = 46)			
	Mean	SD	Mean	SD	*t*	*p*
Age	61.33	7.13	61.67	5.7	-0.26	.80
	n		n		X^2	*p*
Female	28		30		0.47	0.53
Occupation						
Employed	19		14		0.86	0.39
Unemployed	20		26		2.07	0.22
Unknown	9		6		0.57	0.58
Race/ethnicity						
Chinese	46		42		0.81	0.43
Indian	1		1		0.01	1.00
Others	1		3		1.14	.36
Education level						
Primary or less	0		1		1.06	0.49
Secondary	10		8		0.18	0.80
Post-secondary	9		15		2.37	1.58
University	20		14		1.28	0.29
Postgraduate	9		8		0.03	1.00
Religion						
No religion	18		14		0.52	0.52
Buddhist	3		9		3.74	0.07
Hindu	2		1		0.30	1.00
Christian	20		16		0.47	0.53
Muslim	0		1		1.06	0.49
Others	3		3		0.00	1.00
Not disclosed	2		2		2.00	1.00
Sleeping arrangement						
Sharing bed	21		22		0.16	0.84
Not sharing bed	24		23		0.00	1.00
Unknown	3		1		0.96	0.62
Marital status						
Single	12		10		0.14	0.81
Married	29		29		0.07	0.83
Separated	1		1		0.00	1.00
Widowed	4		3		0.11	1.00
Divorced	2		3		0.26	0.67

MBTI, mindfulness based therapy for insomnia; SHEEP, sleep hygiene education and exercise program; SD, standard deviation.
Demographics of study participants. No difference in demographics between groups were found.

(Supplementary Table 3). At the outset, 75 datasets were double scored. To improve on reliability for N1 (original ICC = 0.183), discrepancies were discussed between the 2 raters and all nights were rescored. This resulted in an improvement in ICC across all PSG measures for the restaged data. By the criteria of Cicchetti[30], ICCs for N2, N3 and REM were excellent (all > 0.8), while agreement for N1 was poor.

Cohen's Kappa was calculated for the scorers across all stages, k = 0.768 (or 84.96%), showing substantial agreement.[31] As reported in a separate communication, ICCs for sleep onset latency, wake after sleep onset and total sleep time were also excellent (all > 0.9).

Analysis of sleep macroarchitecture and fragmentation

To test for changes over time and between the 2 interventions, we performed 2 × 2 repeated-measures ANOVA with time (pre- and postintervention) as a within-subjects factor and group (MBTI and SHEEP) as a between-subjects factor (Table 3). As the WTSFI is normalized to the median sleep stage, participants whose wake epochs made up at least 50% of TIB did not have a WTSFI score (MBTI = 4, SHEEP = 2).

Permutation analysis (Fig. 2D) showed a significant increase in N2 in the MBTI group (t_{47} = 2.061, p = .045, d = 0.331) but not in the SHEEP group (t_{45} = 0.013, p = .0989, d = 0.002). However, a significant time x group interaction ($F_{1,92}$ = 1.941, p = .17) was not observed. We observed a significant time x group interaction ($F_{1,92}$ = 4.748, p = .03, η_p^2 = 0.049) in N3 owing to a small decrease in the MBTI and an increase in SHEEP. Permutation analysis (Fig. 2E) showed significant increase in N3 in the SHEEP group (t_{45} = 2.633, p = .012, d = 0.355) but no change in the MBTI group (t_{47} = -0.588, p = 0.559, d = -0.082). No significant differences in either N1, REM or WTSFI (Table 3) were observed.

To ensure that our results were not influenced by inter-subject differences in the proportion of sleep stages, sleep stages as a percentage of TST were tested and showed the same results as time spent in each stage (Supplementary Table 4).

Analysis of sleep microarchitecture

Changes in nREM EEG power across the 6 different frequency bands are reported in Table 3 and Fig. 3. No significant main effects or interactions in the ANOVAs were found in any power bands with the exception of a significant interaction in alpha ($F_{1,92}$ = 5.041, p = .027). Permutation analysis showed reduced theta, alpha, beta and sigma power for SHEEP following the intervention but no change in the MBTI group. Normalized SWA dissipation showed similar reductions in both groups but changes in each quarter over night were not different across groups (Supplementary Fig. 1, Supplementary Table 5).

Subjective sleep quality of PSG completers

Within this subsample of PSG completers, both groups reported improvement in ISI ($F_{1,92}$ = 108.26, p < .01, η_p^2 = 0.541) and PSQI ($F_{1,92}$ = 79.03, p < .01, η_p^2 = 0.462) over time with a significant time x group interaction found in ISI ($F_{1,92}$ = 5.242, p = .024, η_p^2 =0.054) and no significant interaction in PSQI ($F_{1,92}$ = 0.611, p = .44). Estimation analysis showed that both groups had a reduction in ISI scores from pre to postintervention (MBTI: d = -1.36, 95%CI -1.73, -0.95); SHEEP: d = -0.86, 95%CI -1.18, -0.56). Similarly, estimation analysis confirmed an improvement in PSQI for both groups (MBTI: d = -1.16, 95%CI -1.55, -0.74); SHEEP: d = -0.96, 95%CI -1.28, -0.60). We note that these calculations differ from our original report as this paper reports on a subsample of participants and does not use intent-to-treat analysis, although the pattern of the results is the same.

Both subscales of the PSAS showed similar results: a significant reduction in self-rated presleep somatic symptoms ($F_{1,92}$ = 5.642, p = .02, η_p^2 =0.058) and cognitive symptoms (F1,92 = 19.174, p < .001, η_p^2 =0.172) in both groups. Time-by-group interactions were not significant in either somatic ($F_{1,92}$ = 0.49, p = .825) or cognitive ($F_{1,92}$ = 0.0, p = .99) subscales.

Self-reported sleep quality on the night of PSG data collection was analyzed. One participant's data were unavailable and excluded from the analysis. Both groups reported improved subjective sleep quality post intervention ($F_{1,91}$ = 9.557, p = .003, η_p^2 = .095) but no group interaction was seen ($F_{1,91}$ = 2.248, p = .137). Subjective ratings of restedness showed the same trend with both groups reported feeling more refreshed after the intervention ($F_{1,91}$ = 21.118, p < .001, η_p^2 = 0.188), with no time x group interaction ($F_{1,91}$ = 2.143, p = .147).

State anxiety index scores showed reduction across time for both groups, ($F_{1,92}$ = 10.695, p = .002, η_p^2 = 0.104) but no time x group interaction ($F_{1,92}$ = 0.409, p = .524). Trait anxiety index scores showed the same pattern with reduction across time ($F_{1,92}$ = 24.732, p < .001, η_p^2 = 212), but no time x group interaction ($F_{1,92}$ = 1.355, p = .247).

Discussion

We conducted secondary analysis of PSG data from a randomized controlled trial comparing MBTI against SHEEP. Adding to our previous report[16] of changes in objective total wake time, this report

Table 2
Descriptive statistics of polysomnographic sleep parameters by intervention group

	MBTI		SHEEP	
	Pre mean (SD)	Post mean (SD)	Pre mean (SD)	Post mean (SD)
TIB (min)	453.12 (67.41)	454.03 (65.85)	449.87 (71.94)	453.24 (75.44)
SOL (min)	22.46 (17.63)	20.40 (20.52)	21.52 (20.56)	19.86 (21.19)
WASO (min)	88.34 (58.59)	75.10 (59.29)	71.66 (46.87)	60.84 (42.02)
TST (min)	340.39 (70.92)	358.53 (75.20)	356.68 (73.37)	372.54 (56.70)
SE (%)	75.70 (12.13)	79.25 (12.95)	79.39 (11.45)	82.78 (7.88)
N1 (min)	16.45 (12.05)	15.87 (11.99)	16.54 (11.09)	15.78 (12.05)
N2 (min)	204.85 (55.25)	223.40 (55.90)	226.83 (56.35)	226.96 (49.57)
N3 (min)	50.98 (35.35)	48.10 (34.82)	41.15 (28.04)	52.59 (35.38)
REM (min)	70.28 (28.11)	73.29 (29.01)	74.19 (24.73)	78.90 (25.42)
WTSFI	4.91 (1.82)	4.98 (1.87)	4.83 (1.95)	4.52 (1.64)
Delta power (μV/Hz)	27.10 (11.78)	27.31 (11.25)	26.34 (8.43)	25.83 (9.76)
Theta power (μV/Hz)	4.56 (1.78)	4.55 (1.67)	4.75 (1.97)	4.44 (1.75)
Alpha power (μV/Hz)	2.62 (1.09)	2.68 (1.28)	2.94 (1.59)	2.69 (1.35)
Sigma power (μV/Hz)	1.61 (0.67)	1.67 (0.84)	1.84 (0.90)	1.70 (0.80)
Beta power (μV/Hz)	0.43 (0.18)	0.51 (0.52)	0.47 (0.29)	0.39 (0.16)
Gamma power (μV/Hz)	0.24 (0.17)	0.31 (0.54)	0.22 (0.21)	0.18 (0.08)
Normalized SWA Q1	1.25 (0.12)	1.25 (0.14)	1.23 (0.14)	1.26 (0.16)
Normalized SWA Q2	1.02 (0.11)	1.00 (0.11)	1.03 (0.13)	1.02 (0.12)
Normalized SWA Q3	0.90 (0.10)	0.90 (0.13)	0.94 (0.11)	0.91 (0.10)
Normalized SWA Q4	0.83 (0.12)	0.84 (0.15)	0.81 (0.10)	0.81 (0.11)

MBTI, mindfulness-based therapy for insomnia; SHEEP, sleep hygiene education and exercise program; SD, standard deviation; TIB, time in bed; SOL, sleep onset latency; WASO, wake after sleep onset; TST, total sleep time; SE, sleep efficiency; N1, stage N1 sleep; N2, stage N2 sleep; N3, slow wave sleep; REM, rapid eye movement sleep; WTSFI, weighted transition sleep fragmentation index; SWA, slow wave activity; Q1, 2, 3, 4, slow wave activity broken down into quarters.
Descriptive statistics for the PSG variables analyzed for both groups, before and after intervention.

found that both interventions led to changes in sleep macro- and microarchitecture. Specifically, we found that N2 increases from baseline to post-treatment exclusively in the MBTI group, and N3 increases exclusively in the SHEEP group. No significant changes were observed in either group in sleep fragmentation and decreased power in the NREM theta, alpha, sigma and beta bands were observed in the SHEEP group only. We note that of these changes, only N3 and alpha power yielded a significant time by group interaction in repeated-measures ANOVA. These results are discussed in turn.

Sleep macroarchitecture

Slow-wave sleep, or N3, is thought to be the most restorative sleep stage and is robustly associated with subjective sleep quality.[32] N3 increases are generally anticipated with

Table 3
Analysis of variance (ANOVA) and estimation statistics of sleep parameters

	ANOVA						Estimation							
	Time			Time* Group			MBTI				SHEEP			
Measure	df	*F*	*p*	df	*F*	*p*	d	CI	*t*	*p*	d	CI	*t*	*p*
TIB (min)	1,92	0.15	.65	1,92	0.14	.97	0.04	[-0.19 0.28]	0.36	.72	0.05	[-0.26 0.33]	0.31	.76
SOL (min)	1,92	1.00	.34	1,92	0.17	.92	-0.11	[-0.47 0.18]	-0.71	.48	-0.08	[-0.37 0.17]	-0.66	.51
WASO (min)	1,92	4.86	.03*	1,92	0.05	.83	-0.22	[-0.53 0.05]	-1.57	.12	-0.24	[-0.56 0.06]	-1.59	.12
TST (min)	1,92	3.97	.05	1,92	0.02	.89	0.25	[-0.07 0.58]	1.54	.13	0.24	[-0.11 0.59]	1.29	.21
SE (%)	1,92	7.91	.01*	1,92	0.00	.95	0.28	[-0.02 0.61]	1.87	.07	0.34	[0.05 0.64]	2.19	.03*
N1 (min)	1,92	0.24	.63	1,92	0.00	.95	-0.05	[-0.33 0.21]	-0.36	.73	-0.07	[-0.45 0.30]	-0.34	.73
N2 (min)	1,92	2.00	.16	1,92	1.94	.17	0.33	[0.02 0.68]	2.06	.05*	0.00	[-0.37 0.35]	0.01	.99
N3 (min)	1,92	1.69	.2	1,92	4.75	.03*	-0.08	[-0.36 0.19]	-0.59	.56	0.36	[0.10 0.61]	2.63	.01*
REM (min)	1,92	1.65	.2	1,92	0.08	.78	0.11	[-0.21 0.43]	0.65	.52	0.19	[-0.11 0.48]	1.25	.22
WTSFI	1,86	0.36	.55	1,86	0.95	.33	0.04	[-0.22 0.31]	0.30	.77	-0.17	[-0.46 0.16]	-1.03	.31
Delta power	1,92	0.04	.83	1,92	0.27	.61	0.02	[-0.15 0.19]	0.22	.83	-0.05	[-0.28 0.05]	-0.51	.61
Theta power	1,92	2.6	.11	1,92	2.18	.14	-0.01	[-0.15 0.20]	-0.09	.93	-0.17	[-0.29-0.00]	-2.28	.03*
Alpha power	1,92	2.06	.15	1,92	5.04	.03*	0.05	[-0.13 0.25]	0.56	.58	-0.17	[-0.27-0.01]	-2.69	.01*
Sigma power	1,92	0.57	.45	1,92	3.53	.06	0.08	[-0.13 0.36]	0.70	.49	-0.17	[-0.26 0.04]	-2.28	.03*
Beta power	1,92	0.00	1.00	1,92	3.4	.07	0.19	[-0.12 0.52]	1.05	.30	-0.31	[-0.43 0.22]	-2.05	.05*
Gamma power	1,92	0.16	6.94	1,92	2.47	.12	0.19	[-0.06 0.52]	1.08	.26	-0.29	[-0.39 0.34]	-1.54	.13
Normalized SWA Q1	1,91	0.79	.38	1,91	1.23	.27	-0.03	[-0.42 0.33]	-0.16	.87	0.24	[-0.10 0.57]	1.38	.18
Normalized SWA Q2	1,91	0.71	.40	1,91	0.06	.81	-0.15	[-0.50 0.16]	-0.91	.37	-0.07	[-0.45 0.31]	-0.37	.71
Normalized SWA Q3	1, 89	0.99	.32	1, 89	0.59	.45	-0.03	[-0.44 0.40]	-0.15	.88	-0.28	[-0.64 0.13]	-1.33	.19
Normalized SWA Q4	1,92	0.19	.66	1, 92	0.41	.53	0.13	[-0.26 0.49]	0.66	.51	-0.03	[-0.36 0.33]	-0.18	.86

TIB, time in bed; SOL, sleep onset latency; WASO, wake after sleep onset; TST, total sleep time; SE, sleep efficiency; N1, stage N1 sleep; N2, stage N2 sleep; N3, slow wave sleep; REM, rapid eye movement sleep; WTSFI, weighted transition sleep fragmentation index; SWA, slow wave activity. ANOVA and estimation statistics for PSG variables tested.
* $p < .05$.

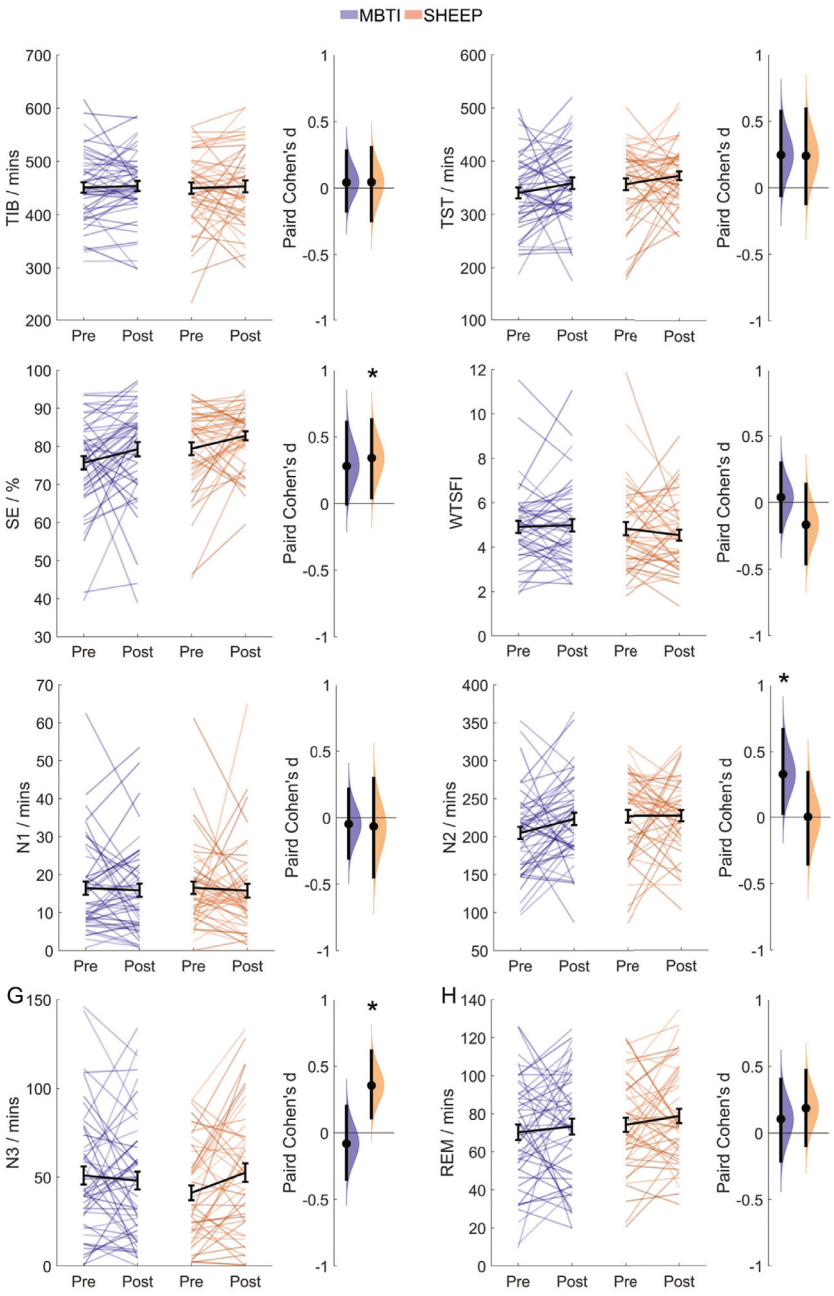

Figure 2. Change in time spent in N1, N2, slow wave and REM sleep, WTSFI, slow-wave activity (SWA) dissipation and SWA power from pre to post intervention. Panels depict the change in variables from pre to post intervention for mindfulness based therapy for insomnia (MBTI; blue) and (sleep hygiene exercise and education program SHEEP; red). The bold lines represent the change in mean and standard error from pre to post intervention and each colored line represents a participant. Estimation and confidence intervals of effect size of the 2 interventions are presented on the right as paired Cohen d's. A) Time in bed (TIB) B) total sleep time (TST) C) sleep efficiency (SE) D) weighted transition sleep fragmentation index (WTSFI) E) duration of N1 sleep. E) duration of N2 sleep. F) duration of N3 sleep. G) duration of REM sleep. * $p < .05$

successful behavioral sleep interventions; however, no studies of MBIs have reported this to date, and the only significant change in macroarchitecture reported has been increased time spent in N1.[11] The lack of significant change in N3 in the MBTI group is thus aligned with these older findings. In addition, we made the novel observation of a significant increase in time spent in N2 following MBTI, although this did not result in a significant time by group interaction, and thus may not be specific to the intervention.

While it is unusual to observe N3 increases after sleep education/hygiene alone, we note that the SHEEP program was fairly intensive, including physical and stretching exercises, and progressive muscle relaxation, and these may have contributed to the increase observed in this group.

Our study contrasts with Ong et al[13] in that MBTI participants in our trial had objectively shorter sleep (measured by PSG) at baseline (348.06 minutes vs. 381.87 minutes in Ong et al[13]), and significantly increased sleep (373.19 minutes) postintervention.[16] We thus draw the tentative conclusion that when mindfulness training leads to increases in TST (which may occur more readily in shorter sleepers), the added sleep time tends to load into N2. In contrast, SHEEP participants, who also had increased TST, spent this added time mostly in N3. In other words, the increase in TST seen in both groups from pre- to postintervention loaded into different sleep stages in the 2 interventions.

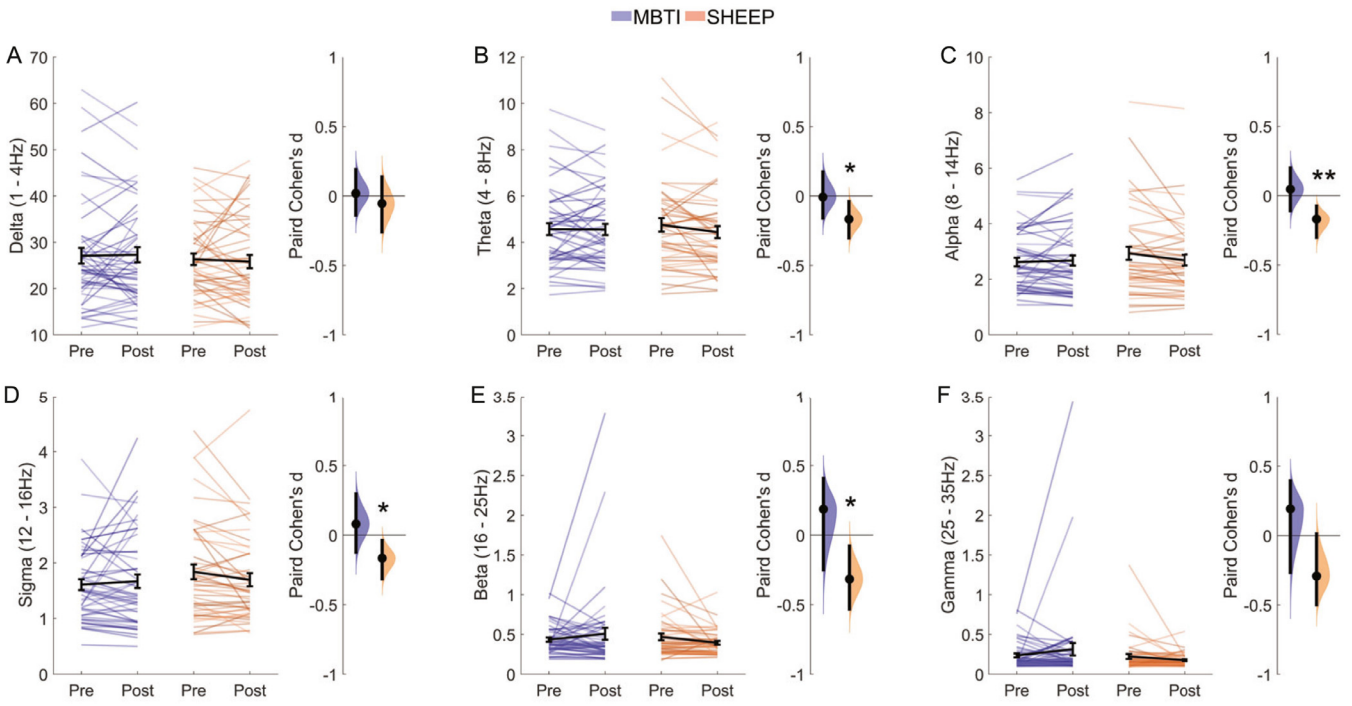

Figure 3. Change in spectral power in 6 bands of Interest**.** Estimation stats for absolute power (μV/Hz) changes from before to after intervention in the A) delta band, B) theta band, C) alpha band, D) sigma band, E) beta band, and F) gamma band. Cohen's d is shown on the right of each panel. We observed significant reduction of power in the theta, alpha, sigma and beta band for participants who underwent the sleep hygiene exercise and education program (SHEEP) intervention. MBTI, mindfulness based therapy for insomnia; * $p < .05$, ** $p < .01$

We note that there was a 10-minute discrepancy in baseline values between MBTI and SHEEP (51 vs. 41 minutes), indicating the possibility of a ceiling-effect in MBTI being a partial driver of our results. However, the randomization procedure makes this explanation unlikely.

Findings from short-term intervention studies of meditation-naïve individuals are at odds with some reports indicating that expert meditators have profoundly different sleep architecture and more consolidated sleep than nonmeditators. For example, Pattanashetty et al[33] observed that experienced Vipassana meditators spend a greater percentage of time in slow-wave sleep compared with matched controls. Similarly, Sulekha et al[34] studied a group of Sudarshan Kriya yoga and Vipassana meditators with PSG recordings and found that they had more N3 (Stage 3 and Stage 4) than age-matched controls, and a similar proportion of N3 than younger (21-30-year-old) nonmeditators. Vipassana meditators also had a significantly greater percentage of REM sleep than age-matched controls. More longitudinal research is required to understand if and how these more striking changes develop with longer-term meditation practice.

Sleep microarchitecture

The second main aim of this analysis was to replicate the findings that MBTI was associated with increases in beta and gamma power.[14] It has been suggested that, unlike interventions such as CBT-I, mindfulness training has a paradoxical effect whereby improvements in self-reported sleep quality are accompanied by increases in markers of nocturnal arousal in sleep EEG. It has been suggested that high frequency EEG changes with meditation practice are a marker of increased alertness that can persist through sleep because of long-term plastic neural changes.[15] This alertness putatively arises because of a tonic heightened awareness of both internal (somatic and cognitive) and external stimuli.

In contrast with Goldstein et al,[14] we did not observe significant increases in high-frequency NREM EEG power in the current study's MBTI group, although there was a nonsignificant numerical increase in both the beta and gamma frequency bands. In the context of other findings, our data suggest that high-frequency power changes with short-term mindfulness training, if any, are modest, and possibly dependent on sample characteristics. However, we did observe decreases in, theta, alpha, sigma, and beta power in SHEEP but not MBTI, with a significant time by group interaction in the alpha band. Patients with insomnia without objective short sleep have been reported to show increased activity from the alpha through the gamma range and these can be reduced after cognitive-behavioral therapy for insomnia.[35] Our overall findings are thus consistent with the notion that mindfulness may *not* exert its beneficial effects on sleep by reducing markers of nocturnal hyperarousal as is sometimes observed in CBT-I. We note that this is despite self-reported pre-sleep arousal significantly decreased in both the MBTI and SHEEP group. Further work is needed to test the robustness of these findings and elucidate the reason for this dissociation.

As prior work has shown that SWA increases following CBT-I,[36] we expected to see similar changes following MBTI. This hypothesis was not supported. SWA is a reliable index of sleep homeostasis (higher SWA indicates higher sleep drive)[37] and may support cognitive functioning.[38] However, conflicting evidence that SWA levels does not differ between poor and good sleepers,[39] makes it challenging to interpret the relationship between SWA and subjective sleep quality. Additionally, as slow-wave activity decreases in middle-aged and older adults,[40] the ability of behavioral interventions to rescue this decrease may be limited. Notwithstanding these caveats, the observed dissociation in our data further underscores the notion that different biological mechanisms may be at play in the 2 interventions.

Sleep fragmentation

No differences were observed in our analysis of sleep fragmentation, in contrast with Britton et al,[11] who reported more night time

awakenings following mindfulness practice. This suggests that the variable may not be a sensitive marker of change following mindfulness-based interventions for poor sleep as previously hypothesized. Further work is needed to explore the clinical implications of this null finding as sleep fragmentation has been associated with negative outcomes such as cognitive decline.

Limitations

The current analysis has several limitations. First, only 1 night of PSG data were collected at each timepoint. Given the night-to-night sleep variability in insomnia patients, this could have impacted macro and micro architecture on the PSG night. This concern is partly mitigated by the fact that participants did not report that sleep on the PSG night was significantly different from their regular sleep. Second, PSG was only measured directly post-treatment, and changes in macro- and microarchitecture may take longer to emerge. Future studies should employ longer-term follow-up of objective measurement to address this question. Third, MBTI differed from SHEEP in several different ways other than the inclusion of mindfulness training (eg, sleep consolidation), and the current results should not be taken as strong evidence that mindfulness alone accounted for our observed dissociation.

Conclusion

We found that MBTI and an active sleep hygiene/exercise control program had different effects on 2 PSG-measured variables: N3, and alpha power. In within-group comparisons, we found that MBTI but not SHEEP participants increased in N2, whereas SHEEP but not MBTI participants showed decreases in high-frequency EEG power. This analysis complements intriguing findings from earlier studies suggesting that mindfulness training may affect sleep via a unique and yet poorly understood mechanism that differs from sleep medication and other forms of psychotherapy. If these effects bear out in future research, they may have important implications for our understanding of the pathophysiology of insomnia, and our concept of sleep quality more generally.

Declaration of conflict of interest

The authors have declared no conflicts of interest.

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Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.sleh.2022.02.003.

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