Sleep in the Laboratory and Sleep at Home II: Comparisons of Middle-Aged Insomnia Sufferers and Normal Sleepers

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Study Objectives: The study compared adaptation responses and sleep pattern differences shown by normal sleepers and insomnia sufferers during lab (LPSG) and home (HPSG) polysomnography.

Design: A counter-balanced, matched-group design was used. Participants underwent 3 consecutive nocturnal LPSG’s and 3 consecutive nocturnal PSG’s in their home (HPSG’s).

Setting: The sleep disorders laboratories at affiliated VA and university medical centers.

Participants: Thirty-five (18 women) middle-aged (40 to 59 years) non-complaining normal sleepers and an age-matched sample of 33 (17 women) individuals who met structured interview criteria for persistent primary insomnia were the study participants.

Measurements and Results: A series of multivariate and univariate analyses were conducted with 9 common sleep parameters to address study objectives. Bed partner influences were controlled by conducting separate sets of analyses for those with and without routine home bed partners. The interaction of participant type (normal vs. insomnia), sleep setting, and PSG sequence (HPSG 1st vs. LPSG 1st) affected first night values of sleep efficiency and stage 2 sleep among those without routine bed partners, and REM latency and sleep efficiency among those with routine bed partners. Analyses which controlled for first night and sequencing effects showed a significant participant type x sleep setting interaction among those with bed partners. These latter analyses suggested that LPSG’s may underestimate the home sleep time of insomnia sufferers and overestimate the sleep continuity of normal sleepers, at least among those who routinely sleep with a bed partner.

Conclusions: The nocturnal recording site may influence adaptation effects and sleep pattern differences noted between insomnia sufferers and normal sleepers.

Key words: Home- and laboratory-based polysomnography; insomnia; first night effects

INTRODUCTION

INSOMNIA IS A HIGHLY PREVALENT FORM OF SLEEP DISTURBANCE THAT COMPROMISES THE HEALTH STATUS, occupational pursuits, and social functioning of countless individuals worldwide.1-6 Whereas insomnia commonly results from medical, psychiatric, and substance-abuse disorders, a substantial subset of insomnia sufferers experience sleep problems which develop and persist independent of such causes. Many who suffer such persistent primary insomnia (PPI) enjoy reasonably satisfactory mental and physical health, yet they incur significant morbidity in association with their sleep complaints. Even when insomnia persists in the absence of co-morbid psychiatric, substance abuse, or medical disorders, it may significantly enhance subsequent risks for various psychiatric illnesses.7-10 Furthermore, PPI may contribute to reduced productivity, work-related accidents, chronic hypnotic dependence, substance abuse, and increased health care costs/utilization.11-15 Unfortunately, many studies conducted to improve our understanding of this form of sleep difficulty have produced rather perplexing results.

Over the past several decades, numerous studies have assessed the nature and extent of sleep pathology associated with PPI. These studies compared results of laboratory polysomnographic (LPSG) recordings derived from individuals with PPI complaints and matched non-complaining normal sleepers.16-24 Most such investigations excluded depressed, medically ill, and apneic patients, and derived sleep data from multiple LPSG nights. Although such studies typically found sleep differences between PPI and control samples, the magnitude of the differences observed consistently appeared rather modest. For example, the mean difference in total sleep time between these groups was less than 38 minutes across these studies whereas the average difference in total nocturnal wake time was less than 42 minutes. These differences seem particularly unimpressive in view of previous research which suggests that normal sleepers can endure extended periods wherein they reduce their sleep by one to two hours below their customary amounts without evidence of any daytime alertness/performance decrements.25 Indeed, we are left to wonder whether we are missing something in our laboratory study of those with PPI complaints.

In this regard, it seems important to question the potentially powerful influences the sleep laboratory setting may have on comparisons of insomnia sufferers and normal sleepers. Since normal sleepers and PPI sufferers differ markedly in their reported satisfaction with their usual home sleep patterns, the novelty and routines of the sleep laboratory may have contrasting effects on these two groups. Among normal sleepers, usual bedtime rituals and the familiar home sleeping environment provide a sense of stability and serve as powerful conditioned cues which facilitate a restorative sleep process. The absence of these cues in the laboratory setting may adversely affect the sleep of these individuals when, as has been the case in most research protocols, they complete only a few recording nights in the novel laboratory setting. Conversely, among those with PPI, aberrant sleep...
h habitats occurring at home and conditioned stimuli present in the home bedroom may obviate a regular sleep/wake routine and elicit bedtime arousal which perpetuate sleep disturbances.\textsuperscript{26,27} In addition, many insomnia sufferers may view the sleep laboratory as a setting where poor sleep is expected and understood by those professionals conducting the sleep recordings. This view, in turn, may reduce the conditioned bedtime arousal which typically confounds their sleep attempts at home. Given this rationale, it seems reasonable to speculate that insomnia sufferers may appreciate some sleep improvements when they are monitored in the sleep laboratory. Hence, LPSG might underestimate the degree of sleep difficulty such individuals typically experience in their home sleep settings.

Support for these contentions come from previous studies of sleep laboratory adaptation effects as well as investigations which have scrutinized insomnia sufferers and normal sleepers in their home sleeping environments. Various studies of the so-called “first night effect” (FNE) have shown that normal sleepers show more disrupted sleep (e.g., longer sleep onset latencies, longer REM latencies, greater sleep fragmentation, etc.) on the first laboratory recording night than they do on subsequent nights whereas a reversed FNE, characterized by improved sleep on the first laboratory night (relative to subsequent nights), has been noted among insomnia sufferers.\textsuperscript{28-32} In contrast, a few studies have suggested that neither normal sleepers nor insomnia sufferers show these pronounced adaptation responses when monitored in their homes.\textsuperscript{33-35} Moreover, in our previous work we found that home-based PSG (HPSG) recordings showed older (ages>60 yrs.) insomnia sufferers and normal sleepers had similar mean values of sleep measures across lab and home PSGs although the insomnia group showed significantly greater night-to-night sleep variability than did the normal sleepers only during HPSGs.\textsuperscript{35}

The current investigation was conducted as extension of our previous HPSG studies of insomnia. We conducted this study with the global premise that HPSG is relatively unencumbered by adaptation effects inherent in the sleep laboratory setting and, hence, should be more likely than LPSG to show sleep differences between insomnia sufferers and normal sleepers. In the current project we chose to enroll a middle-aged cohort inasmuch as we suspected that age-related sleep changes (i.e., sleep fragmentation, reduced slow-wave sleep, etc.) among the older controls enrolled our previous study\textsuperscript{35} may have served to minimize the degree to which their sleep differed from matched insomnia sufferers. Given these considerations, our specific study hypotheses for the current project were: 1) both normal sleepers and insomnia sufferers would show less pronounced FNEs (i.e., significant differences between sleep measures derived on the first recording night vs. similar measures from subsequent nights) during HPSGs than during LPSGs; and 2) mean values of sleep measures averaged across HPSG nights would suggest greater sleep differences between insomnia sufferers and normal sleepers than would averaged sleep measures derived from LPSG.

**METHOD**

**Participants**

Research participants were recruited via posted advertisements at the Durham (NC) VA Medical Center, via letters mailed to persons in the Duke University Center for the Study of Aging and Human Development Subject Pool, and via face-to-face solicitations of patients presenting to the Duke University Sleep Disorders Center. All participants completed an informed consent prior to entering the study and were compensated financially for their participation. Participants first underwent multiple screening procedures including a Structured Interview for Sleep Disorders\textsuperscript{36} and, once enrolled, they completed self-report questionnaires and multiple nights of sleep monitoring. The insomnia sufferers recruited for this study were middle-aged adults (ages 40 to 59 years) who reported sleep complaints consistent with current criteria\textsuperscript{37} for Primary Insomnia. These prospective participants were considered for inclusion if they: 1) reported chronic (i.e., of >6 months duration) difficulty initiating or maintaining sleep or noted chronic poor sleep quality (i.e., nonrestorative sleep) which, on average, minimally occurred at least three times per week; and 2) reported associated daytime deficits related to their nocturnal sleep difficulties. We also recruited a comparable sample of middle-aged, non-complaining adults. These normal sleepers had no identified major medical or psychiatric condition that might have contributed to an unreported, occult sleep disorder.

Using thorough screening procedures we excluded prospective participants if they: 1) had a terminal illness; 2) had a medical condition (e.g., rheumatoid arthritis, thyroid disease) that compromises sleep; 3) had abnormal TSH levels on a screening thyroid panel; 4) had a history of psychiatric illness; 5) met criteria\textsuperscript{37} for a current major psychiatric (Axis I) condition on the basis of a Structured Clinical Interview for Psychiatric Disorders;\textsuperscript{38} 6) were substance abusers; 7) showed sedative hypnotic dependence and were unwilling/unable to abstain from these medications while in the study; 8) were taking anxiolytics, antidepressants, or any other psychotropic medication; or 9) had objective evidence of clinically significant sleep apnea (i.e., and apnea/hypopnea index - AHI >15) on nights 1 or 2 of the objective sleep recordings described later herein. In addition, we excluded prospective insomnia sufferers if they met SIS-D criteria\textsuperscript{36} for another sleep disorder in addition to PPI whereas we excluded normal sleepers who met SIS-D criteria\textsuperscript{36} for any sleep disorder.

A total of 76 (35 women, 41 men) prospective participants underwent the study’s screening procedures. Of these, three of the male self-described normal sleepers and five men with insomnia complaints were dropped because they had AHI’s >15 during their initial PSG studies. The remaining group comprising our final sample consisted of 33 (17 women) insomnia sufferers and 35 (18 women) non-complaining, normal sleepers.

The normal sleepers retained had an average age of 46.5 years (SD=5.0 yrs.) and had completed an average of 16.2 years (SD=2.5 yrs.) of formal education. Twenty-seven (16 women) of these individuals were Caucasians, seven (one woman, six men) were African Americans, and one (woman) was an Asian American. Independent sleep interviews and medical exams suggested that none of these individuals had medical conditions that contributed in any way to sleep difficulties. However, a number of these volunteers reported histories of common medical conditions and symptoms including mild arthritis (n=6), coronary artery disease (n=1), hypertension (n=3), sinusitis (n=7), occasional heartburn (n=6), and intermittent headaches (n=3). Medical and sleep history evaluations conducted during screening suggested that these conditions/symptoms either were con-
trolled with ongoing treatment or were not present at the time of study entry.

The average age of the insomnia sufferers was 49.9 years (SD=5.8 yrs.) and their average years of formal education was 14.9 years (SD=2.8 yrs.). Of these individuals, 21 (11 women) were Caucasians and the remaining seven (six women) were African Americans. Most of the insomnia sufferers were research volunteers who were recruited via solicitation announcements/letters. However, three of the insomnia sufferers were sleep clinic patients who agreed to become study participants. Like the normal volunteers, these insomnia sufferers endorsed histories of medical symptoms and conditions including mild arthritis (n=11), coronary artery disease (n=1), diabetes (n=2), hypertension (n=5), sinusitis (n=7), occasional heartburn (n=8), and intermittent headaches (n=8). Nonetheless, as was the case among the normal volunteers, our screening evaluations suggested that these conditions/symptoms either were controlled with ongoing treatment or were not present at the time of study entry. Moreover, both the self reports of these individuals and our screening evaluations suggested that these medical conditions were not significant contributors to their reported sleep difficulties.

Although most of the insomnia sufferers enrolled were not clinical patients, they collectively appeared to suffer from long-standing sleep difficulties inasmuch as they reported having suffered from insomnia for an average of 9.5 years (SD = 7.6 yrs.). Three (9.1%) of the insomnia sufferers reported exclusively sleep onset difficulties, 14 (42.4%) reported only sleep-maintenance difficulties, 14 (42.4%) reported mixed onset/maintenance problems and two (6.1%) reported concerns in regard to chronic poor sleep quality. At the time of enrollment, 24 (72.7%) of the insomnia sufferers reported no current use of prescription or non-prescription sleep aids, four (12.1%) reported use of such sleep aids less than one time per week, and the remaining five (15.1%) reported use of sleep aids two or more times per week. Twenty-eight (84.8%) reported no use of alcohol as a sleep aid whereas four (12.1%) used low dose (one to two alcoholic beverages) alcohol as a hypnotic less than one time per week. Only one (3.0%) of the insomnia sufferers reported nightly reliance on one to two alcoholic beverages to aid sleep. However, all of these individuals agreed to totally abstain from the use of sleep aids during the time periods prescribed by the study (see details below).

**Polysomnography**

Each participant underwent six nights of polysomnographic (PSG) sleep monitoring. Three consecutive PSG recordings were conducted in the Duke Medical Center’s Sleep Laboratory and the other three consecutive PSG studies were conducted in participants’ homes.

All participants underwent both home and lab PSGs in order to address this study’s specific research objectives. The order of studies (lab vs. home) was randomly determined so that roughly one-half of the men and women in each group underwent lab recording first, whereas the other half completed home monitoring first. So that PSG measures would be reasonably contemporaneous, we scheduled each participant’s home and lab PSGs a minimum of four and a maximum 30 days apart.

All PSGs were conducted using Oxford Medilog® 9000 series ambulatory cassette recording devices. The recorders have the capability of recording eight channels of electro-physiological data as well as digital time in one-second intervals. They also include an event marker which participants used to electronically mark both the time they retired to bed at night and their subsequent final rising time on the following morning. Various studies conducted at our center and elsewhere have attested to the technical acceptability of the Oxford system and have shown that sleep measures derived from this form of monitoring are comparable to those obtained from standard laboratory polygraphs. The specific PSG-monitoring montage used in the current study included two electroencephalogram (EEG) channels (C3-A2, Oz-Cz), bilateral electrooculogram (EOG), submental electromyogram (EMG), two channels of anterior tibialis EMG (right and left leg,) and a nasal/oral respiration thermistor.

Prior to scheduling PSG recordings, participants were thoroughly interviewed to determine their customary bedtimes and rising times. Once these times were discerned for each participant, she/he was instructed to adhere to these customary bedtimes and rising times on all six nights (lab and home) that PSG recordings were scheduled. On dates home PSG studies were scheduled, participants reported to the sleep laboratory between 14:00 and 17:30 for electrode attachment before returning home where they were encouraged to follow their usual evening routines and pre-sleep rituals. Each individual was also instructed to sleep in her/his usual bedroom with her/his usual bed partner if such an individual was typically present. In the morning, they returned to the sleep laboratory for removal of electrodes.

On the nights of laboratory studies, participants reported to the sleep laboratory without bed partners approximately 60 to 90 minutes before their reported usual bedtimes for electrode placement. As a consequence of this scheduling, electrode attachment was routinely completed just prior to each participant’s target bedtime so, shortly after completing of this process, the individual was placed in a sleeping room, the lights were turned off, and the recording was started. For both home and lab studies, participants kept written records of their bed and rising times. These records were used to assist our research staff in finding the electronically marked bed and rising times when scoring the Medilog recordings.

All Medilog recordings were scored directly on the screen of the Medilog scanner by experienced scorers using standard criteria. This screen scoring procedure was used since it allows for both rapid scanning of sleep data and screen by screen editing for those recordings which prove difficult to score. Moreover, we have found screen scoring produces estimates of standard sleep parameters that are comparable to those obtained from conventional epoch by epoch scoring of records on paper. To minimize biases in scoring, scorers were kept blind to the date of each recording and the type of participant from whom each PSG study was obtained. Since H PSG electrode attachments occurred between 14:00 and 17:30, these recordings began well before the individuals’ bedtimes in the home settings. As a result, it was not possible to totally blind scorers to the setting wherein recordings were obtained.

Results of sleep-stage scoring were used to derive measures of total sleep period (TSP—time between “lights out” and final rising time) and several sleep parameters which previously have proven useful either for detecting FNE’s or for discriminating insomnia sufferers from normal sleepers. The specific sleep...
parameters we chose to use included total sleep time (TST), sleep efficiency (SE%=[total sleep time/sleep period] x 100%), latency to the onset of sustained sleep (SOL—time between “lights out” and the first 10 minutes of sleep containing no more than two minutes of wake time, stage 1 sleep or movement time), wake time after sleep onset (WASO—all wake time after SOL and before the final AM awakening), stage 1 time (STG1), stage 2 time (STG2), slow wave sleep time (SWS), REM time, and REM latency (RLMA—time between SOL and the first three consecutive minutes of REM minus intervening wake time) for each study night. For the purpose of this investigation, time and latency measures were expressed in minutes.

**Procedure**

All consenting persons who met inclusion criteria underwent the six PSG studies previously described. Following their first series of three consecutive PSGs (home or lab), participants underwent daytime sleepiness and performance testing which are to be described in a future report.46 All HPSG studies were scheduled for nights when subjects planned to have no overnight house guests. Participants who reported recent use of sleep medications were required to abstain from these medications for at least two weeks prior to their first series of studies and to not resume these medications until they completed both series of PSGs. Finally, they were instructed to abstain from alcoholic beverages and to not consume caffeinated substances after 18:00 on study nights.

**RESULTS**

**Sample Comparability and Effectiveness of Randomization**

Prior to conducting tests of our study hypotheses, we first conducted several analyses to compare our samples in regard to their demographic characteristics. Fisher Exact tests showed the samples of insomnia sufferers and normal controls were well balanced in terms of their females/males (p=1.00) and Caucasians/Non-Caucasians (p=1.00) ratios. Results of a 2 (insomnia sufferer vs. normal sleeper) x 2 (genders) ANOVA showed that the insomnia sufferers (Mage=49.9 years), on average, were slightly, albeit significantly (F1,63=7.33, p=.009), older than were the normal sleepers (Mage=46.5 years). A similar analysis showed that the normal sleepers (Meducation=16.2 years) had slightly, albeit significantly (F1,64=4.23, p=.04), more years of education than did the insomnia sufferers (Meducation=14.9 years). These statistical differences were consistent across genders inasmuch as the gender and type x gender effects were nonsignificant (p’s>.05).

In addition to these initial comparisons, we conducted preliminary analyses to test the effectiveness of our randomization procedures. As noted previously, we enrolled a total of 76 participants in the study and retained 68 participants. Since all subjects were randomized at the time of study enrollment, we reviewed participant assignments to determine if roughly equal numbers of men and women within each sample of our final cohort were assigned to each sleep monitoring sequence (i.e., HPSG 1st vs. LPSG 1st). This review showed that 18 (10 women) insomnia sufferers and 16 (8 women) normal sleepers underwent HPSG first whereas the remaining 15 (7 women) insomnia sufferers and 19 (10 women) normal sleepers underwent LPSG first. Chi-square analysis confirmed that the four subgroups were proportionally assigned to the contrasting sequences of PSG studies (x2=0.81, p=0.85).

**Technical Acceptability of Medilog Recordings**

Of the 408 PSGs conducted on our final cohort, 397 were technically acceptable and scorable. The remaining 11 (2.7%) were not scorable either because the clock module inside of the Medilog recorders malfunctioned or critical electrodes became dislodged during the study. All but one of these unscorable recordings occurred on nights 2 or 3 in the one or the other of the recording sites (home or lab). Single nights (two HPSG and three LPSG) were lost from five (four women and one man) normal sleepers and four (three men, one woman) insomnia sufferers (one HPSG and three LPSG’s). In addition, data were lost from a female normal sleeper from night three in the lab and night one in the home. The unscorable LPSG recordings were lost because we chose not to tether subjects to an online recording apparatus in the lab; this decision assured that the recording procedures used in both recording sites were identical. Because of data loss, results reported herein are based on the 397 scorable recordings.

**Pattern and Reliability of PSG Scoring**

A total of three scorers participated in the scoring of the PSGs obtained in this study, and except for those records used in the reliability checks, each PSG was scored by only one of these individuals. However, all (98%) but eight of the scorable records were scored by one of the two main scorers who participated in this project. For 52 of the 68 participants, the task of scoring the multiple nights of PSG recording was split between the two scorers with one scorer reviewing some of the six nights and the other scoring the remainder. Scorer 1 scored 44 HPSGs and 46 LPSGs from the normal sleepers and 51 HPSGs and 52 LPSGs from the insomnia sufferers. Scorer 2 scored 56 HPSGs and 54 LPSGs from normal sleepers, and 44 HPSGs and 42 LPSGs from the insomnia sufferers. An overall chi square test showed that the four specific types of records were equally distributed across these two main scorers (x2=3.64 p=.30).

To test for inter-scorer reliability, 10 randomly selected PSG’s were independently scored by each of the two main scorers. Epoch-by-epoch comparisons showed an overall agreement rate of 90% between scorers. This highly acceptable agreement rate suggested little variance due to scorer differences.

**Adherence to Prescribed Bed and Rising Times**

Despite instructions to the contrary, study participants varied their prescribed bed and wake times across the six PSG nights. For the normal sleepers, the mean bedtimes across the three LPSG and three HPSG studies respectively were 23:02, 23:07, 23:12, 23:11, 23:01, and 22:44. For the insomnia group these respective times were 22:54, 22:51, 22:52, 22:41, 22:44, and 22:35. The LPSG and HPSG mean rising times across nights for the normal sleepers respectively were 6:08, 6:13, 6:14, 6:16, 6:13 and 5:57; these respective mean times for the insomnia group were 6:18, 6:17, 6:17, 6:08, 6:27, and 6:26. Analyses via 2 (normal sleeper vs. insomnia sufferer) x two (LPSG vs. HPSG) x
three (night) repeated measures ANOVAs showed the participants’ rising times were statistically similar across nights and settings but the bedtimes of both groups were significantly (F(1, 328) = 4.02, p = 0.05) earlier in their homes (M_time = 10:50 PM) than they were in the lab (M_time = 11:00 PM). Normal sleepers tended to choose a later rising time on night 1 in their homes than they did on subsequent nights in that setting. In contrast, the insomnia sufferers arose earlier on HPSG night 1 than they did on subsequent HPSG nights. Finally, ANOVA results showed that average total sleep periods—TSPs (times in bed) of the insomnia sufferers (M_TSP = 452.4 min., SD = 58.0 min.) were significantly (F(1, 328) = 5.34, p = 0.02) longer than those of the normal sleepers (M_TSP = 427.3 min., SD = 57.8 min.) and both groups had significantly (F(1, 328) = 1.63, p = 0.12) longer TSPs in their homes (M_TSP = 444.5 min., SD = 67.4 min.) than they did in the sleep lab (M_TSP = 434.6 min., SD = 49.4 min.).

Tests of Normality and Data Transformations

Prior to conducting planned tests of FNEs, we inspected each sleep measure to determine if its distribution approximated normality. In doing so, we computed Shapiro-Wilk tests* of normality and constructed frequency histograms with each measure so that we could statistically as well as visually evaluate each distribution. Results of these procedures showed that the majority of these measures had distributions that were normal or approached normality (W values > 0.95). However, the distributions of the sleep onset latency, sleep efficiency, stage 1 time, WASO, and REM latency were somewhat skewed. As a result, we used arithmetic data transformations (e.g., Logarithm) to normalize these measures before conducting any tests of study hypotheses.

First Night and Sequencing Effects

We predicted that both groups would show less pronounced differences between first and subsequent night values of each sleep parameter in their homes than they would in the lab. To test this prediction, we first computed the difference between values of each sleep parameter derived from the first recording night in each setting and mean values of these respective sleep measures for nights 2 and 3 in each setting. Combining data from nights 2 and 3 in these computations seemed justified since preliminary comparisons showed no significant differences between these two nights for any of the sleep measures in either of our samples.

To conduct our comparisons of these resultant difference scores, we first used an omnibus 2 (normal sleeper vs. insomnia sufferer) x 2 (recording site: lab vs home) x 2 (PSG sequence: Sleep in the Laboratory and Sleep at Home II—Edinger et al.

<table>
<thead>
<tr>
<th>Measure*</th>
<th>Normal Sleepers Lab PSG</th>
<th>Normal Sleepers Home PSG</th>
<th>Insomnia Sufferers Lab PSG</th>
<th>Insomnia Sufferers Home PSG</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nt 1-Nts 2/3</td>
<td>Nt 1-Nts 2/3</td>
<td>Nt 1-Nts 2/3</td>
<td>Nt 1-Nts 2/3</td>
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<tr>
<td>TST</td>
<td>-1.1 (9.2)</td>
<td>-13.4 (12.9)</td>
<td>-25.3 (8.9)</td>
<td>-53.8 (12.5)</td>
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<tr>
<td>S2 onset</td>
<td>5.3 (5.6)</td>
<td>5.8 (3.2)</td>
<td>13.9 (5.5)</td>
<td>3.8 (3.1)</td>
</tr>
<tr>
<td>WASO</td>
<td>2.5 (6.9)</td>
<td>1.2 (8.1)</td>
<td>8.2 (6.7)</td>
<td>26.4 (7.9)</td>
</tr>
<tr>
<td>SE %</td>
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<td>-0.8 (2.1)</td>
<td>-4.4 (1.5)</td>
<td>-7.7 (2.1)</td>
</tr>
<tr>
<td>Stage 1</td>
<td>-0.4 (3.5)</td>
<td>-1.3 (3.2)</td>
<td>0.7 (3.4)</td>
<td>1.7 (3.1)</td>
</tr>
<tr>
<td>Stage 2</td>
<td>-2.1 (8.0)</td>
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<td>3.2 (7.7)</td>
<td>-24.7 (8.9)</td>
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<td>SWS</td>
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<td>-17.3 (6.0)</td>
<td>-16.4 (5.3)</td>
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<td>REM</td>
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<td>-5.8 (5.9)</td>
<td>-11.9 (5.0)</td>
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<td>RLMA.</td>
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<td>1.3 (9.6)</td>
<td>5.9 (5.6)</td>
<td>10.9 (9.3)</td>
</tr>
</tbody>
</table>

*Note: Full definitions for the abbreviations used for sleep measures in this table can be found in the portion of Method section labeled “Polysomnography.” The differences shown represent minutes for all sleep measures except sleep efficiency which is expressed as differences in %. Data presented are means and (SE’s). Nt. = night. Data from one female normal sleeper without a bed partner was excluded due to PSG recorder failure on the first home PSG night. Thus, data for the group without bed partners are from 11 (8 women) normal sleepers and 9 (5 women) insomnia sufferers; data for the subgroup with bed partners are from 23 (9 women) normal sleeper and 24 (12 women) insomnia sufferers.
HPSG 1st vs. LPSG 1st) x 9 (sleep parameters) repeated measures multivariate ANCOVA model.

To control for demographic and gender differences, we included age, educational level, and gender (female = 0; male = 1) as covariates in this model. Also included as covariates were measures which corrected for the previously noted differences in bed times and times in bed (TSPs) across nights and settings. Furthermore, in anticipation of potential influences of routine bed partners, we performed separate analyses for the subgroups with and without routine home bed partners. Finally, these analyses were conducted both with raw and normalized values of our dependent measures so as to determine the effects of data transformations on the results obtained.

Since the analyses of raw and normalized dependent measures produced similar results, only the results for the raw data are presented herein to simplify data interpretation. Table 1 shows the adjusted mean differences between initial and subsequent PSG nights (i.e., First night value—Mean of Nights 2 & 3) and standard error terms for these differences scores. Our omnibus ANCOVA comparisons of FNEs showed significant participant type x recording site, x PSG sequence x sleep measure interactions among both those without routine home bed partners (F8,88 = 4.20, p=0.003) and those who had routine home bed partners (F8,304 = 2.20, p=0.03). Follow-up univariate tests showed significant participant type x recording site x PSG sequence interaction effects for the measures of sleep efficiency (F1,11 = 7.89, p=0.02) and stage 2 sleep (F1,11 = 5.59, p=0.04) among those without routine bed partners, and for the measures of sleep efficiency (F1,38 = 4.68, p=0.04) and REM latency (F1,38 = 5.68, p=0.02) among those with routine bed partners.

Figure 1 shows the trends in the night 1 vs. nights 2/3 difference scores which contributed to these interaction effects. Each
graph in this figure shows the differences between the first night and subsequent nights in each recording site. Negative values imply that first night values of the sleep parameter were lower than values obtained for subsequent nights, whereas positive values suggest first night values were higher than those shown on subsequent nights. Among the subgroup without bed partners, contrasting patterns of FNEs were found between the normal sleepers and insomnia sufferers who underwent LPSGs first, showed standard FNE’s in sleep efficiency and home FNEs among the insomnia sufferers.

Results of Group Comparisons

To determine if, as predicted, home PSG recordings showed greater differences between our two samples than did LPSGs, we conducted a series of analyses which controlled for the above-noted first night and sequencing effects, and allowed us to isolate setting-specific effects of the two recording sites on our two samples. To do so, we eliminated first night data and used participants’ averaged values of the nine sleep measures derived from nights two and three in each setting. Subsequently, we conducted an omnibus 2 (normal sleeper vs. insomnia sufferer) x 2 (recording site: lab vs home) x 9 (sleep parameters) repeated measures multivariate ANCOVA model to analyze these data. Included as covariates in this model were such variables as age, educational level, gender (female = 0; male = 1), as well as measures which corrected for the noted differences in bed times and times in bed (TSPs) across settings. Also, we included a dichotomous covariate (LPSG 1st = 0; HPSG 1st = 1) in our statistical model so as to partial out PSG-sequencing effects from our final results. As was the case in our tests of PSG-adaptation effects,
we conducted these analyses with both raw and normalized data and performed separate analyses for the subgroups with and without routine bed partners.

The analyses of raw and normalized dependent measures produced similar results, so, once again, only the results for the raw data are considered in order to simplify data interpretation. Table 2 provides descriptive statistics concerning the combined sleep data from nights 2 and 3 for the various participant subgroups. Results of our omnibus ANCOVAs showed no significant effects among the subgroup without routine home bed partners. However, among those with routine bed partners, a significant participant type x recording site x sleep measure (F_{3,312}=2.82, p=0.005) interaction effect was obtained. Follow-up univariate ANCOVA’s showed significant participant type x recording site interactions for measures of total sleep time (F_{1,30}=5.95, p=0.02), WASO (F_{1,30}=6.62, p=0.01), and sleep efficiency (F_{1,30}=6.61, p=0.01) among this subgroup. Post-hoc tests showed the normal sleepers and insomnia sufferers had statistically similar values of total sleep time within each sleep setting. However, the insomnia sufferers slept significantly longer in their homes than they did in the sleep lab. In contrast, the insomnia sufferers had significantly more WASO and significantly lower sleep efficiencies than did the normal sleepers within each recording site. Nonetheless, these group differences were less marked during HPSSGs than during LPSGs. Moreover, whereas the insomnia sufferers had statistically similar values of WASO and sleep efficiency across settings, the normal sleepers had statistically higher values of WASO and lower sleep efficiencies in their homes than they did in the sleep lab. Thus, contrary to predictions, these findings suggest that LPSGs may imply greater relative sleep disturbances among insomnia sufferers than do home-based recordings.

DISCUSSION

The current investigation was conducted, in part, to test our prediction that both insomnia sufferers and normal sleepers would show significantly less pronounced FNEs during HPSSG than they would during LPSG. Our findings both failed to support this hypothesis and suggested the few FNEs observed seem dependent upon the particular sequence in which PSGs are conducted, the type of individual undergoing monitoring, and the individual’s habit of sleeping with or without a routine home bed partner. Among those without bed partners, FNEs in sleep efficiency and stage 2 sleep were more pronounced among the insomnia sufferers than among the normal sleepers but the nature of these effects seemed influenced more by the order of recording sites than by the sleep settings themselves. These individuals showed standard FNEs during their first series of recordings and reverse FNEs during their second series of PSGs. Among those with routine home bed partners, differences in FNE’s were most pronounced in comparisons of the normal sleepers and insomnia sufferers who underwent LPSG prior to HPSSG. Within this group, the normal sleepers showed reduced FNE’s in sleep efficiency and reverse FNEs in REM latency on their subsequent HPSSGs whereas the insomnia sufferers undergoing the same PSG sequence showed much more marked standard FNE’s in these measures during HPSSGs than during LPSGs.

Although PSG adaptation effects varied as a function of setting, study sequence, and usual sleeping arrangement, a number of generalities are suggested by these data. First, for most of the sleep parameters examined, FNEs were not more pronounced in the lab than they were in the home. In addition, the FNEs observed appeared minimal among our normal sleepers. Furthermore, regardless of their usual sleeping arrangement, insomnia sufferers did not show a reduced first night adaptation response in their second series of studies relative to their first series. Instead these individuals seemed reactive to change in PSG monitoring venue particularly when switching from lab to home-based monitoring. Finally, the home-lab differences in FNEs observed varied between those who did and did not have a routine home bed partner. This observation seems noteworthy inasmuch as the influence of a companion in the home sleeping environment has generally been ignored in studies of lab and home FNEs. Indeed, our data would suggest that more systematic scrutiny of this factor’s influence on FNEs seems warranted.

As an additional study objective, we tested our prediction that HPSSG’s would show greater differences between insomnia sufferers and normal sleepers than would LPSG’s. When conducting analyses pertinent to this prediction, we eliminated first night data and employed a multivariate model that isolated sleep setting effects via statistically controlling for demographic differences, varying sleep scheduling across recordings sites, and PSG sequencing effects. Our results did suggest that LPSG and HPSSG provide somewhat distinctive views of the sleep differences between insomnia sufferers and normal sleepers. However, these results were limited to the subgroup of participants with home bed partners and were not in the direction anticipated. Within this cohort, LPSGs generally suggested greater relative sleep difficulties (lower sleep times and sleep efficiencies, more wake time) among the insomnia sufferers than did HPSSGs. Whereas both LPSG and HPSSG showed the insomnia sufferers had more WASO and lower sleep efficiencies than did the normal sleepers, the group differences were less dramatic during the HPSSGs. Furthermore, whereas the average total sleep times of the two groups were statistically similar in both sleep settings, the insomnia sufferers slept significantly longer in their homes than they did in the lab. Moreover, the normal sleepers had significantly less consolidated sleep in their homes than they did in the sleep lab. Hence, contrary to prediction, these findings suggest that LPSG may actually overestimate insomnia sufferers’ relative disruption, at least among those with routine bed partners. These data also suggest normal sleepers may have somewhat less consolidated sleep in their homes than we have been led to expect from laboratory studies.

Overall, these group comparisons might be considered disappointing in the sense that our HPSS studies failed to show much more marked differences between our study samples than did the LPSG comparisons. In fact, like many previous studies,16-24 both our lab and home PSGs suggested rather modest relative sleep deficits among our insomnia cohort. Given the level of daytime distress often reported by PPI patients as well as the apparent morbidity associated with insomnia, per se, it may well be that standard Rechtshaffen and Kales47 parameters provide a very limited view of disease severity among many who suffer from this form of sleep difficulty. What our findings do suggest in this regard is that much more research concerning the nature of insomnia is needed before we understand the relationship between insomnia’s objective sleep dysfunction and its eventual untoward consequences.
In view of our results, it seems useful to consider the relative merits of HPSG and LPSG. First, it appears that, for the types of individuals studied herein, LPSG does not appear to have more pronounced FNEs than HPSG, at least for the majority of the sleep measures we employed. As a result, expectations for reduced FNE’s does not appear to be an adequate justification for choosing HPSG over LPSG. However, since we used relatively unobtrusive ambulatory recorders for home and lab recordings, our findings may underestimate differences that would be found between ambulatory and more traditional LPSG in which individuals are tethered to a large, stationary recording apparatus. Secondly, since our night 2/3 data showed no recording site effects for those without routine bed partners, HPSG, which requires no in laboratory bedrooms and no over-night technologist, may, for practical reasons, be favored over LPSG for studies in this subgroup, particularly when strict experimental control is not essential. For those who typically sleep with bed partners, HPSG and LPSG clearly give distinctive views of differences between normal sleepers and insomnia sufferers on sleep measures that are thought to be very relevant to insomnia complaints. In studies of such individuals, HPSG may be favored in naturalistic studies focused on capturing “typical” sleep patterns. In contrast, LPSG may remain the gold standard for group comparisons when the goals of PSG monitoring require a standardized, controlled sleeping environment. For example, studies concerned with basic sleep physiology or the endogenous circadian system may require LPSG.

In reviewing our results, it is important to consider this study’s limitations. Our sample was moderate in size and consisted of only middle-aged normal sleepers and non-clinical insomnia sufferers who presented to us as research volunteers. Whether our findings apply to normal sleepers in general, younger age groups, and clinical samples of insomnia patients remains to be determined. Also, the small number of participants without bed partners may have prevented us from detecting group and setting differences that would be apparent in a larger cohort of this nature. In addition, we attempted to achieve comparable bedtimes and rising times for our subjects during LPSG and HPSG studies, but, in no way did we intrude into subjects’ homes to enforce these prescriptions. Although we attempted to statistically control for the sleep scheduling variability seen across participant types and sleep settings, replication of this investigation with more rigidly controlled sleep schedules may be beneficial. Despite such limitations, our findings suggest that HPSG may provide an alternative view of the sleep differences between insomnia sufferers and normal sleepers.

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