Discrepancy Between Subjective Symptomatology and Objective Neuropsychological Performance in Insomnia

Henry J. Orff, MS[1,2]; Sean P. A. Drummond, PhD[2,3]; Sara Nowakowski, MS[1,2]; Michael L. Perlis, PhD[4]

[1]Joint Doctoral Program in Clinical Psychology, San Diego State University and University of California, San Diego, CA; [2]Research Service, VA San Diego Healthcare System, San Diego, CA; [3]Department of Psychiatry, University of California San Diego, CA; [4]Department of Psychiatry, University of Rochester, Rochester, NY

Study Objectives: While daytime impairment is a defining feature of primary insomnia (PI), prior research using objective measures has not yielded clear and reliable evidence of global or specific deficits. In this investigation subjective and neuropsychological measures of daytime impairment were concurrently evaluated in subjects with primary insomnia (PIs) and in healthy good sleeper subjects (GSs). The goals for the study were to assess (1) whether PIs differ from GSs on subjective and/or objective measures and (2) the extent to which subjective and objective measures provide discordant information.

Design: Subjects were evaluated on multiple self-report measures of sleep and daytime performance and were administered a comprehensive set of neuropsychological tests.

Setting: The University of Rochester Sleep and Neurophysiology Research Laboratory (Rochester, NY).

Patients or Participants: Forty-nine subjects (32 PIs and 17 GSs). Seventy-one percent of the sample was female; average age 39 ±11yrs.

Results: Overall, PIs reported worse sleep, diminished activity levels, and a greater number and severity of daytime complaints. However, PIs did not show deficits on neuropsychological tests. Additionally, neuropsychological measures were not associated with severity of daytime complaints. Objectively measured sleep was found to be associated with performance (motor speed), while prospective and objective sleep measures were associated with level of daytime complaint.

Conclusions: The discrepancy between subjective daytime complaints and objective performance in individuals with insomnia is common, but poorly understood. This discordance may suggest that daytime impairment corresponds less to "output" and more to attentional bias or to the realistic appraisal that "effort" is required to maintain normal performance.

Keywords: Insomnia, neuropsychological performance, daytime impairment

Citation: Orff HJ; Drummond SP; Nowakowski S; Perlis ML. Discrepancy between subjective symptomatology and objective neuropsychological performance in insomnia. *SLEEP* 2007;30(9):1205-1211.

INTRODUCTION

ACCORDING TO THE INTERNATIONAL CLASSIFICATION OF SLEEP DISORDERS (ICSD-2), INSOMNIA MAY BE DEFINED AS BOTH A SYMPTOM AND A DISORDER.[1] AS a symptom, insomnia is defined as one or more of the following: difficulty initiating or maintaining sleep, waking from sleep too early, and/or the complaint of nonrestorative sleep. As a disorder, these sleep complaints must occur in association with *adequate opportunity for sleep* and the complaint of *impaired daytime function* (e.g., difficulties with attention, and memory and/or diminished vocational functioning). These 2 additional considerations serve to ensure that 1) the complaint of nonrestorative sleep is not the result of self-imposed sleep restriction and 2) the occurrence of sleep initiation or maintenance problems is not the result of a mismatch between sleep opportunity and basal sleep need. When formally assessed, patients with insomnia report problems with fatigue, weariness, and malaise. These complaints are accompanied by reports of impaired concentration, poor memory, and decreased ability to accomplish daily tasks.[2,3] Telephone surveys[4] suggest the majority of subjects with untreated insomnia report being too tired to do things (78%), having trouble remembering things (59%), and confused thinking and/or judgment (43%). These findings have been corroborated using various methodologies, and these self-reported deficits are positively correlated with insomnia severity.[5,6]

Given the ubiquitous nature of daytime complaints in this population, one would expect a substantial literature providing objective evidence of cognitive impairment in primary insomnia (PI). This is not the case. To date, approximately a dozen studies have examined neuropsychological measures in PIs, focusing on 3 main areas of performance: attention, memory, and working memory. The one cognitive domain in which PIs most consistently show deficits is attention. PIs exhibit worse performance than controls on several measures of attention, including choice reaction time tests,[7] simple reaction time tests, and a visual tracking test.[8,9] PIs also have been found to recall fewer numbers on the digit span tests[7,10] and have reduced vigilance during a 24-hour constant routine procedure.[11] Not all studies, however, have found evidence of attention related deficits.[9,12] In contrast to attention, the vast majority of studies examining memory have reported normal performance in PIs relative to controls on tests of both immediate[7,10,13] and delayed recall.[10] A few minor deficits in memory have, however, been reported. Mendelson et al reported that PIs displayed deficits in accessing semantic memory,[14] and Szelenberger reported that PIs required more repetitions to learn

Disclosure Statement

This was not an industry supported study. Dr. Drummond has received research support from GlaxoSmithKline and Sanofi-Aventis. Dr. Perlis has received research support from Cephalon, and Sanofi-Aventis; has been a consultant to Respironics, Gerson Lehrman Group, and Elan-King Pharmaceuticals; and is a shareholder and managing member of Internet Didactics Services. Mr. Orff and Ms. Nowakowski have indicated no financial conflicts of interest.

Submitted for publication August, 2006
Accepted for publication May, 2007

Address correspondence to: Sean P. A. Drummond, PhD, Department of Psychiatry, 9151B, UCSD / VASDHS, 3350 La Jolla Village Dr., San Diego, CA 92161; Tel: (858) 642-1274; Fax: (858) 458-4201; E-mail: drummond@ucsd.edu

items relative to normal controls (although this may be related to attention).[13] Tests of working memory also show intact, rather than impaired, performance in PIs.[12]

Overall, despite consistent findings of self-reported problems, the evidence for overt neuropsychological impairment in PI remains equivocal, at best. In the present study, the relationship between daytime complaint and performance measures was evaluated in PIs and healthy controls to determine 1) if the groups differ on measures of daytime complaints and/or on neuropsychololgical performance, and 2) the extent to which these domains are correlated and/or associated with subjective or objective sleep measures. Rationales for the discrepancy between daytime complaints and performance are also presented.

METHODS

Recruitment/Screening

Data were obtained from a larger investigation comparing PIs and GSs on measures of high frequency EEG activity during NREM sleep (RO1 MH59392). All subjects in this study signed a consent form approved by the University of Rochester Human Subjects Committee (Rochester, NY). During screening, volunteers completed 2 weeks of sleep diaries and were evaluated for psychiatric illness with a PC-based structured clinical interview for DSM-IV to rule out Axis I diagnoses. Participants also were excluded if they scored ≥10 on the Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), or Hamilton Depression Rating Scale (HAMD). The cutoffs on the HAMD, BDI, and BAI were imposed to help assure that only individuals with primary insomnia and not comorbid insomnia would be enrolled in the study. Subjects with comorbid insomnia were excluded because the comorbid conditions may, in and of themselves, influence cognitive performance (e.g., depression). Subjects were given a brief medical history and physical and had blood and urine chemistries done at intake. Any individuals with significant medical comorbidities and/or unstable or untreated medical conditions, a history of seizures or seizure disorder, who were pregnant or breast feeding or had plans to become pregnant, or who had sleep apnea or periodic limb movements detected on a screening PSG were excluded. Female participants had to be either premenopausal or 2+ years postmenopausal.

Prior to intake and during the study, subjects were allowed to consume moderate amounts of caffeine (1-2 cups/day, not after 12:00) and alcohol (1-2 servings/day not less than 4 hours before bedtime). Subjects were not enrolled if they smoked or used nicotine-based products. Also, subjects were excluded if they were currently taking any medication known to affect sleep. Insomnia subjects were required to remain free of sleeping medications (prescription or OTC) for 2 weeks prior to the study and during the course of the study. Instruments administered at the initial evaluation to obtain a more complete subject profile included Pittsburgh Sleep Quality Inventory (PSQI)[15] Multidimensional Fatigue Inventory (MFI),[16] and Epworth Sleepiness Scale (ESS).[17]

Subjects

Forty-nine adults aged 26-63 years (32 PIs and 17 "good sleepers": GSs) participated. Enrollment was contingent upon subjects meeting eligibility requirements as determined by 2 weeks of prospective sleep diaries. PIs were 22 females and 10 males, aged 26-63 years (mean 40.6 ± 6.3) with 12-20 years (mean 15 ± 2.1) of education. GSs were 13 females and 4 males, aged 26-50 years (mean 36.1 ± 7.1) with 12-20 years (16 ± 1.7) of education. Initially, 17 control subjects were matched to 17 of the insomnia subjects for age, sex, body mass index (BMI), education, race, and handedness. Another 13 insomnia subjects from the same demographic group were later added to the sample to provide additional power to detect potentially small group differences in performance measures. This increased power was especially important given that the null hypothesis was predicted for performance measures.

PIs were required to report all of: a) a subjective perception of >30 minutes to fall asleep and/or >30 minutes awake after sleep onset (WASO), b) either 6 h TST and/or a sleep efficiency of less than 80%, c) a reported sleep disturbance of ≥4 nights per week for ≥6 months, and d) a complaint of daytime impairment of functioning and/or performance which the subject attributed to sleep disturbance. GSs must have reported <15 minutes to fall asleep, <15 minutes WASO, >7 h TST, and 90%+ sleep efficiency. All subjects had a habitual bedtime of 20:00-00:00 and waketime 06:00-08:00. Subjects were excluded from the study if they regularly reported sleeping outside this window to minimize the potential for enrolling subjects who might have a phase advance/delay disorder. Subjects were selected primarily for subjective estimates of sleep problems in their diaries and for self-reported presence of daytime impairment during the phone screen and at intake. No specific cut-off scores for daytime complaints in the diaries or other questionnaires were used to determine subject eligibility or to select patients for the study.

Procedures

SLEEP DIARIES

Subjects completed nightly sleep diaries for 2 weeks prior to screening PSG. These diaries included time to bed, sleep latency, number of awakenings, WASO, time out of bed, time of final awakening, and overall sleep quality; they also included assessments of daytime functioning, number of naps, and amount of exercise. Total sleep time and sleep efficiency were calculated by the investigators and added to the database. Performance questions (5-point Likert scales) provided subjective measures of ability to concentrate, level of fatigue, irritability, stress, overall physical well-being, and feelings of restedness.

POLYSOMNOGRAPHY

All prospective subjects underwent a one night screening PSG to rule out sleep apnea (RDI>5) and periodic limb movements (PLMI>5). Subjects' bedtimes and rise times were determined by their habitual sleep schedules. As a result of extensive phone screens and intake evaluations, no subjects were excluded from the study as a result of PLMs or OSA at this stage.

NEUROPSYCHOLOGICAL MEASURES

On the evening following the screening night, each participant completed a 1.5 h battery of neuropsychological tests consist-

ing of measures of motor speed, attention, verbal fluency, verbal learning, and memory. Test selection was not straightforward because most cognitive tasks used in the insomnia literature have shown equivocal results regarding impairment relative to good sleepers. This battery was therefore chosen to evaluate a wide range of cognitive domains, including both those in which performance would be expected to be maintained (verbal fluency, verbal learning, and memory) and those where there is evidence in the literature that insomnia patients may be impaired (motor speed, attention). Within these domains, standardized measures were selected where possible.

All testing sessions occurred between 19:00 and 21:00. This time was selected so as to test all subjects when the circadian system promotes greater alertness ("maintenance of wakefulness zone"),[18] and at a time that would be convenient for subjects participating in the study. Recent research[11] has shown that circadian phase has no differential effects on performance or daytime complaints in patients with insomnia as compared to controls. Therefore, it did not appear that an evening vs morning timing of testing would be critical to the outcome. During the 1.5-h testing session, 9 neuropsychological tests were administered.

ATTENTION TASKS

Stroop Color and Word Test (SCWT): This is a timed test assessing attentional persistence, processing speed, and efficiency of information sorting, and is designed to measure a subject's ability to inhibit habitual responding. It consists of 3 separate subtests.[19]

Digit Span Subtest of the WAIS-III: The test taps both attention and working memory. In this test, subjects are required to repeat a string of numbers presented verbally, exactly as given for "digits forward," and in reverse order for "digits backward."[20]

Brief Test of Attention (BTA): This test assesses auditory attention and requires participants to listen to an alphanumeric string that increases in length from 4 to 18 items and count either the number of letters or numbers presented. Unlike digit span, this test does not require subjects to recall which numbers or letters are presented.[21]

Letter-Number Sequencing Subtest of the WAIS-III: This test taps both attention and working memory.[20] In this test, subjects are required to listen to a string of letters and numbers and to repeat them back in alphabetical and chronological order.[20]

Sustained Attention: This test assesses attention and visuomotor reaction time. Subjects are required to respond as quickly as possible to a visual stimulus (large yellow circle) presented on computer screen at random intervals within a defined 10-minute period.

MOTOR SPEED TASKS

Digit Symbol Substitution Test (DSST): This test assesses visuomotor speed and attention. Participants are required to apply symbols to digits based on a symbol-digit key presented at the top of the testing sheet.[22]

Trail Making Test (TMT): This 2-part test assesses visual search and set-shifting abilities, psychomotor speed, and mental flexibility. On part A, the task is to connect lines to 25 circled numbers in sequence. On part B, each circle contains either a letter or a number, and the task is to draw lines alternating from number to letter, consecutively (e.g., 1-A-2-B…).[23]

VERBAL FLUENCY

Controlled Oral Word Association Test (COWAT): This test assesses verbal fluency and the ability to inhibit previous responses. The test requires that subjects spontaneously produce words beginning with a given letter within a limited amount of time.[24]

VERBAL LEARNING AND MEMORY

Hopkins Verbal Learning Test (HVLT): This test assesses free recall and recognition memory over 2 time intervals (immediate recall and delayed recall [15 minutes]). Participants are required to learn a series of 12 words over 3 trials. They are then asked to recall these words after the delay period and to recognize the words from a list including distracters.[25]

Analyses

Group characteristics, PSG variables, and neuropsychological profiles were analyzed with MANOVAs and follow-up univariate ANOVAs. MANOVAs were performed separately for objective (PSG), retrospective (questionnaire) and prospective (sleep diaries) sleep measures, daytime complaint (questionnaires and sleep diaries), and 4 domains of neuropsychological performance (attention, motor speed, verbal fluency, and verbal learning and memory). As age was presumed to differentially affect sleep and performance variables, it was included as a covariate in all analyses. As education was thought to be a potential confound for performance, this variable was included as a covariate in the analysis of neuropsychological measures.

The following variables were used as response variables in the various MANOVAs (see Tables 1-4). Objective sleep was assessed with sleep parameters derived from the adaptation night PSG between groups. Retrospective sleep between groups was assessed using the scores of the Epworth and PSQI. Prospective sleep profiles were evaluated by analyzing the average sleep continuity for the 2 groups, including self-reported measures of sleep latency, number of awakenings, wake after sleep onset time, and the derived measures of total sleep time and sleep efficiency. Daytime complaint between groups was assessed with scores on the MFI and 5-point Likert scales on the sleep diaries. Finally neuropsychological performance between groups was evaluated by comparing test results grouped across 4 domains of functioning: attention, motor speed, verbal fluency, and verbal learning and memory.

To examine associations that might exist for linear combination of variables (as opposed to univariate associations), we ran canonical correlations between objective/subjective sleep measures, daytime complaint reports, and performance measures.

RESULTS

Objective (PSG) Sleep Measures

Using Wilk's criterion (λ) as the omnibus MANOVA test statistic, and age as a covariate, the combined dependent variables (DVs) for objective sleep measures failed to reveal group differences: $F_{19, 28} = 0.625$, P= 0.855, partial $\eta^2 = 0.298$. Descriptive statistics are presented in Table 1.

Table 1—Objective Sleep Measures (MANOVA Wilks' λ P = Ns) Covariate = Age

Measure	PI Mean (SE)	GS Mean (SE)
Efficiency (%)	83.47 (1.76)	83.52 (2.43)
SL (MIN)	12.65 (1.92)	14.87 (2.65)
REML (MIN)	92.29 (6.37)	90.57 (8.79)
# Awakenings	11.24 (0.98)	9.55 (1.35)
WASO (MIN)	53.91 (6.96)	55.08 (9.61)
Wake%	12.25 (1.64)	12.45 (2.26)
TST (MIN)	361.75 (9.41)	365.94 (13.00)
STG1%	5.16 (0.65)	6.73 (0.89)
STG2%	63.10 (1.63)	61.70 (2.25)
STG3%	6.03 (0.78)	6.50 (1.07)
STG4%	3.15 (0.93)	3.65 (1.28)
REM%	22.57 (1.42)	21.41 (1.96)

Note: Values reported are of the adjusted means based on the covariates. SL=Sleep Latency, REML=REM Latency, WASO=Wake After Sleep Onset, TST=Total Sleep Time, STG=Stage. SE = standard error

Subjective Sleep Measures

The omnibus MANOVA evaluating retrospective sleep measures, using age as a covariate, resulted in a significant main effect for group $F_{4,43} = 44.429$, P <0.001, partial $\eta^2 = 0.805$. Follow-up univariate ANOVAs indicated that PIs had significantly higher global scores on the PSQI, but not the Epworth. Comparison of PSQI component values revealed that PIs reported significantly worse SL and TST relative to GS. See Table 2.

The omnibus MANOVA evaluating prospective sleep measures, using age as a covariate, showed a significant main effect for group: $F_{6, 41} = 17.953$, P <0.001, partial $\eta^2 = 0.724$. Follow-up univariate ANOVAs indicated that on the sleep diaries, PIs reported greater impairment than GSs on all major sleep continuity measures. On Likert scale diary measures of self-reported sleep quality, PIs also quantified their sleep as worse than that of GSs. See Table 2.

Daytime Complaints

The omnibus MANOVA evaluating daytime complaints, using age as a covariate, resulted in a significant main effect for group: $F_{12, 35} = 6.223$, P <0.001, partial $\eta^2 = 0.681$. Follow-up univariate ANOVAs indicated that PIs reported higher levels of impairment relative to GSs on both the MFI and diary scales. Overall scores on the MFI differed significantly between the groups and follow-up analysis revealed that PIs reported increased mental fatigue, as well as reduced activity and motivation. On the sleep diary Likert scale measures, PIs reported worse concentration, greater daytime fatigue, more irritability, more stress, and lower overall physical well-being than did GSs. PIs also reported feeling less well-rested during the day. See Table 3.

Neuropsychological Data

The MANOVAs, using age and education as covariates, found attention: $F_{14,32} = 1.794$, P <0.085, partial $\eta^2 = 0.434$; motor speed: $F_{3, 43} = 0.639$, P<0.594, partial $\eta^2 = 0.043$; verbal fluency: $F_{3, 43} = 2.387$, P <0.082, partial $\eta^2 = 0.143$: and verbal learning and memory: $F_{6, 40} = 1.387$, P <0.244, partial $\eta^2 = 0.172$, were not significantly different between groups. See Table 4.

Canonical Associations

The only significant canonical associations were: a) PSG measures with motor function (canonical correlation 0.853, Wilk's λ = 0.082, $\chi^2(36) = 57.413$, P = 0.013, $\eta^2 = 0.918$); b) PSG measures with daytime complaint measures (canonical correlation of 0.974, Wilk's λ, P<0.001, $\chi^2(132) = 170.933$, P = 0.013, $\eta^2 = 1.000$); and c) diary measures with daytime complaint measures (canonical correlation of 0.948, Wilk's λ= 0.010, $\chi^2(66) = 100.481$, P = 0.004, $\eta^2 = 0.990$). There were no cannonical associations between daytime complaint measures and any domain of neuropsychological functioning.

Table 2—Subjective Sleep Measures (MANOVA Wilks' λ P< 0.001) Covariate = Age

Measure	PI Mean (SE)	GS Mean (SE)	SIG	partial η^2
Retrospective Sleep Meaures				
PSQI SL	45.86 (4.44)	8.01 (6.13)	< 0.001	0.348
PSQI TST	300.93 (10.43)	457.61 (14.41)	< 0.001	0.623
PSQI Total	11.80 (0.45)	1.80 (0.62)	< 0.001	0.786
Epworth	8.22 (0.79)	5.82 (1.09)	0.085	0.063
Prospective Sleep Measures				
SL	49.21 (6.35)	9.77 (8.79)	0.001	0.221
# Awakenings	2.63 (0.20)	1.05 (0.27)	< 0.001	0.318
WASO	60.69 (4.31)	7.63 (5.95)	< 0.001	0.527
TST	322.63 (15.17)	455.59 (20.95)	< 0.001	0.630
Efficiency (%)	71.47 (1.56)	93.28 (2.16)	< 0.001	0.588
Sleep quality (5-pt scale)	2.66 (0.13)	4.79 (0.18)	< 0.001	0.664

Note: Values reported are of the adjusted means based on the covariates. P-values reported above are derived from the post hoc univariate ANOVAs and are not corrected for Type I error. These tests would remain significant based on Bonferroni correction at P <0.0125 (retrospective) and P <0.008 (prospective). PSQI=Pittsburgh Sleep Quality Inventory, SL=Sleep Latency, TST=Total Sleep Time, WASO=Wake After Sleep Onset. SE = standard error

Table 3—Daytime Complaint Measures (MANOVA Wilks' λ P< 0.001) Covariate = Age

Measure	PI Mean (SE)	GS Mean (SE)	SIG	partial η^2
MFI				
Total	50.37 (1.39)	37.54 (1.91)	< 0.001	0.386
Mental fatigue	10.95 (0.61)	5.44 (0.85)	< 0.001	0.373
Reduced activity	8.92 (0.53)	5.10 (0.74)	< 0.001	0.274
Reduced motivation	7.86 (0.46)	5.43 (0.63)	0.003	0.172
Physical fatigue	13.17 (2.65)	9.44 (3.66)	0.418	0.014
General fatigue	12.35 (0.29)	12.75 (0.40)	0.434	0.013
Diary (5-pt Scale)				
Fatigue	2.21 (0.16)	0.91 (0.22)	< 0.001	0.327
Restedness	2.60 (0.15)	4.53 (0.20)	< 0.001	0.558
Phy well-being	3.57 (0.14)	4.42 (0.19)	0.001	0.220
Stress	1.60 (0.16)	0.80 (0.22)	0.005	0.162
Irritability	1.38 (0.16)	0.66 (0.23)	0.013	0.127
Concentration	3.73 (0.19)	4.52 (0.26)	0.018	0.115

Diary Scales: higher value equals subjective report of "more" of particular characteristic
Note: Values reported are of the adjusted means based on the covariates. P-values reported above are derived from the post hoc univariate ANOVAs and are not corrected for Type I error.
All tests (except irritability and concentration) would remain significant based on Bonferroni correction at P <0.004. SE = standard error

DISCUSSION

This study investigated how subjective measures of sleep and daytime impairment in patients with primary insomnia and good sleepers are associated with objective performance on neuropsychological testing. Overall, the results indicate that patients with primary insomnia reported worse sleep, diminished activity levels, and a greater number and severity of daytime complaints. The patients with primary insomnia did not, however, show global deficits on neuropsychological tests. Additionally, neuropsychological performance measures were not associated with severity of daytime complaints. Rather, objectively measured sleep was found to be significantly associated with one performance domain (motor speed), while prospective and objective sleep measures were found to be associated with level of daytime complaint. These findings suggest that individuals with primary insomnia who have either an objective bad night of sleep or a subjective bad night of sleep report more daytime functioning complaints, but do not necessarily show impaired performance objectively. The fact that subjective measures of sleep and subjective daytime functioning were found to be significantly associated in this study is not unexpected. The strength of the association likely resulted from the selection criteria required for a diagnosis of primary insomnia. However, the fact that subjective measures of daytime function were not in any way associated with objective measures of daytime performance suggests these constructs measure very different aspects of primary insomnia. While the exact mechanism of these subjective/objective differences remains unclear, there are a number of theoretical considerations that may provide testable hypotheses to help better understand and explain the findings.

Subjective-Objective Discordance in Performance

Patients with primary insomnia often report their sleep to be worse than is measured by PSG. Many ascribe this to simple exaggeration in relation to objective measures of sleep. In extreme cases, this phenomenon can be diagnosed as sleep state misperception insomnia. A similar case can be made for the present lack findings regarding neuropsychologically assessed performance. That is, patients may simply report more perceived deficits during the day than can be determined by objective measures. In this manner one might conceptualize a phenomenon of "daytime performance misperception," which would be defined as a discrep-

Table 4—Neuropsychological Test Measures (MANOVA Wilks' λ P = ns) Covariates = Age and Education

Measure	PI Mean (SE)	GS Mean (SE)
Attention		
Stroop Color/Word –total	60.48 (1.82)	58.34 (2.52)
Stroop Error	2.29 (0.30)	1.28 (0.41)
Digit Span Forward	11.50 (0.39)	11.18 (0.54)
Digit Span Backward	8.07 (0.45)	6.63 (0.62)
Brief Test of Attn-Numbers	8.28 (0.28)	8.83 (0.38)
Brief Test of Attn-Letters	9.09 (0.20)	9.42 (0.28)
Letter-Number Sequencing	12.21 (0.54)	11.42 (0.75)
PVT- Mean reaction time	310.99 (8.23)	302.16 (11.39)
PVT-Mean lapses	0.01 (0.01)	0.06 (0.02)
Motor Speed		
Digit Symbol	81.49 (2.23)	83.09 (3.08)
Trails B (Time)	62.28 (3.66)	63.66 (5.07)
Verbal Fluency		
Cowat total	39.71 (2.31)	42.03 (3.19)
Cowat Intrusions	0.26 (0.12)	0.75 (0.17)
Cowat Perseverations	0.84 (0.26)	0.18 (0.36)
Verbal Learning And Memory		
HVLT Trial 1	7.76 (0.29)	8.09 (0.40)
HVLT Trial 2	10.10 (0.28)	10.29 (0.39)
HVLT Delayed Recall	9.42 (0.32)	10.03 (0.44)
HVLT Recognition	11.57 (0.16)	11.46 (0.22)

Note: Values reported are of the adjusted means based on the covariates. Additional measures for some tests available from authors upon request. PVT=Persistence of Vigilance Test, HVLT=Hopkins Verbal Learning Test. SE = standard error

ancy between a patient's self-perceptions of daytime impairment and objective measures of such impairment.

Hyperarousal and Performance

Overestimation of performance deficits may be explained by physiological hyperarousal. Most of the physiological models investigated to date have assumed a priori that arousal and sleep are mutually exclusive. Studies evaluating physiological arousal in insomnia have used a variety of techniques, including whole body metabolic rate,[26,27] heart rate variability,[28] neuroendocrine measures,[29] and functional neuroimaging,[30] and each has found evidence for increased physiological activation in individuals with insomnia relative to controls. It is possible that if this hyperarousal extends to daytime hours, it aids in maintaining performance (despite sleep loss or other consequences of the disorder) and simultaneously results in fatigue, dysphoria, and memory problems. In this model, the daytime arousal, the resultant symptoms, or both are manifested as the self-reported daytime complaints.

Selective Attention and Performance

Recent research has proposed that patients with insomnia may over attend or selectively attend to the potential consequences of insomnia.[31,32] That is, irrespective of whether patients with insomnia experience more fatigue and/or worse than average performance, they nonetheless attend to these phenomena as more salient and/or memorable. Moreover, the detection of "deficits" reinforces the tendency to attend to (and interpret such events as being related to) insomnia. Good sleepers, in contrast, pay little attention to minor cognitive errors or physical challenges that occur during the day, and to the extent that such phenomena are "noticed," they are not interpreted as being the result of poor sleep. Espie and colleagues have developed cognitive tests to assess for the proposed attentional bias in insomnia and have shown that PIs display greater bias for sleep related stimuli than other groups.[33,34]

Compensatory Recruitment and Performance

The disconnect between patients' self-reported daytime difficulties and their actual performance on objective tests may be due to increased neurophysiologic workload. If this is the case, then "effort" rather than "output" may be the true underpinning of the daytime complaint associated with insomnia. Here we use the term "effort" descriptively to explain a process by which individuals with insomnia work against the neurophysiological gradient imposed by the disorder to maintain performance. If increased effort represents the underlying complaint associated with performance, then one must ask how effort can be conceptualized. Specifically, the inconsistent results seen in objective testing in this population may be due to the fact that individuals with insomnia actively recruit additional cerebral resources in response to a cognitive challenge. Such results would be consistent with observations of individuals with obstructive sleep apnea (OSA),[35] those who undergo total sleep deprivation protocols,[36] and patients with a variety of neuropsychiatric disorders.[37-40] While caution should be used in extrapolating findings from in OSA and sleep deprivation to insomnia, these and other literatures provide testable hypotheses regarding how maintenance of daytime performance in insomnia could be explained. For example, if insomnia patients show patterns of increased activation in functional neuroimaging studies similar to those reported in these other literatures, then one could argue that it is the internalized aspect of such compensatory recruitment that leads to the insomnia patient's perception of difficulties with performance. In such a model, neuropsychological tests may fail to adequately capture the true underlying problem because these tests measure the behavioral output rather than the associated neuropathophysiology. Hence, the associated compensatory recruitment may account for both the ability of an insomnia patient to perform well and the subjective impression of difficulty in performing cognitive tasks.

LIMITATIONS

One potential caveat and concern for this study is the failure to see objective differences in sleep between PIs and GSs. The issue of the influence of the prior night's sleep is interesting, and one that affects all of the literature in this field. Given the instability of insomnia severity across nights and typical reaction of patients to the laboratory environment (i.e., "first night effects" in GS and "reverse first night effects" in PI) it is hard to say what impact night-to-night changes in sleep may have on daytime performance.

A second concern is that the tests selected to assess impairment might have been too easy or simply the wrong tasks to probe cognitive deficits associated with insomnia. Our test selection was guided by the desire to a) measure a variety of cognitive domains, including those with and without evidence of impairment in insomnia; b) use validated, standardized tests whenever possible; and c) administer tests that have been shown to be sensitive to perturbations in sleep. To address this concern, future studies should attempt to employ tests that include 1) the ability to analyze time-on-task effects, 2) parametric manipulation of difficulty levels, and 3) tests designed specifically to be sensitive to insomnia.

A third concern is that there may have been factors, such as IQ and socioeconomic status, that affect performance and were not controlled for in this study. As many factors may contribute to insomnia and to deficits in or maintenance of performance, future research should attempt to identify and address these issues.

Finally, the subjective ratings in the diaries used to assess daytime complaints were derived from our conceptualization of the concerns expressed by patients with insomnia, as well as clinical experience with this population. Since these measures were not validated, it is possible that these questions did not accurately reflect daytime sequelae. Future studies would benefit from development of standardized measures of daytime function that could be used by researchers in the field.

CONCLUSIONS

This study assessed the statistical relationship between subjective and objective measures of performance in insomnia. The inability to find any significant associations between these factors suggests that self-reports of the daytime consequences of insomnia do not reflect the same constructs as objective measures of daytime consequences. While several theoretical frameworks may be used to explain these differences, future studies are needed to help test and refine those frameworks as the field moves towards a better understanding of the cognitive consequences of insomnia.

REFERENCES

1. American Academy of Sleep Medicine. International classification of sleep disorders, 2nd ed.: diagnostic and coding manual. Westchester, IL: American Academy of Sleep Medicine, 2005.
2. Roth T, Roehrs T. Insomnia: epidemiology, characteristics, and consequences. Clin Cornerstone 2003;5:5-15.
3. Grunstein R. Insomnia. Diagnosis and management. Aust Fam Physician 2002;31: 995-1000.
4. Balter MB, Uhlenhuth EH. The beneficial and adverse effects of hypnotics. J Clin Psychiatry 1991;52 Suppl:16-23.
5. Ohayon MM, Lemoine P. [Daytime consequences of insomnia complaints in the French general population]. Encephale 2004;30:222-7.
6. Carey TJ, Moul DE, Pilkonis P, Germain A, Buysse DJ. Focusing on the experience of insomnia. Behav Sleep Med 2005;3:73-86.
7. Hauri PJ. Cognitive deficits in insomnia patients. Acta Neurol Belg 1997;97:113-7.
8. Schneider C, Fulda S, Schulz H. Daytime variation in performance and tiredness/sleepiness ratings in patients with insomnia, narcolepsy, sleep apnea and normal controls. J Sleep Res 2004;13:373-83.
9. Edinger JD, Glenn DM, Bastian LA, et al. Daytime testing after laboratory or home-based polysomnography: comparisons of middle-aged insomnia sufferers and normal sleepers. J Sleep Res 2003;12:43-52.
10. Vignola A, Lamoureux C, Bastien CH, Morin CM. Effects of chronic insomnia and use of benzodiazepines on daytime performance in older adults. J Gerontol B Psychol Sci Soc Sci 2000;55:P54-62.
11. Varkevisser M, Kerkhof GA. Chronic insomnia and performance in a 24-h constant routine study. J Sleep Res 2005;14:49-59.
12. Bonnet MH. Recovery of performance during sleep following sleep deprivation in older normal and insomniac adult males. Percept Mot Skills 1985;60:323-34.
13. Szelenberger W, Niemcewicz S. Severity of insomnia correlates with cognitive impairment. Acta Neurobiol Exp (Wars) 2000;60:373.
14. Mendelson WB, Garnett D, Gillin JC, Weingartner H. The experience of insomnia and daytime and nighttime functioning. Psych Res 1984;12:235-50.
15. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193-213.
16. Smets EM, Garssen B, Bonke B, De Haes JC. The Multidimensional Fatigue Inventory (MFI) psychometric qualities of an instrument to assess fatigue. J Psychosom Res 1995;39:315-25.
17. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14:540-5.
18. Strogatz SH, Kronauer RE, Czeisler CA. Circadian pacemaker interferes with sleep onset at specific times each day: role in insomnia. Am J Physiol 1987;253(1 Pt 2): R172-8.
19. Golden CJ. Stroop Color and Word Test: a manual for clinical and experimental uses. IL: Stoelting Company, 1978.
20. Wechsler D. Wechsler Adult Intelligence Scale - Revised manual. New York, NY: Harcourt Brace Jovanovich, 1981.
21. Schretlen D. Brief Test of Attention: professional manual. Odessa, FL: Psychological Assessment Resources, 1997.
22. Smith A. A Symbol-digit modalities test manual. Los Angeles, CA: Western Psychological Services Publishing, 1982.
23. Lezak MD. Neuropsychological assessment. 4th ed. New York, NY: Oxford University Press, 2004.
24. Spreen O, Benton A. Neurosensory Center for Comprehensive Examination of Aphasia (NCCEA) - Revised edition. Victoria, Canada: University of Victoria, 1977.
25. Benedict RHB, Schretlen D, Brandt J. Hopkins Verbal Learning Test - Revised: Instructions for Administration and Scoring, 1997.
26. Bonnet MH, Arand DL. 24-Hour metabolic rate in insomniacs and matched normal sleepers. Sleep 1995;18:581-8.
27. Bonnet MH, Arand DL. Hyperarousal and insomnia. Sleep Med Rev 1997;1:97-108.
28. Bonnet MH, Arand DL. Heart rate variability in insomniacs and matched normal sleepers. Psychosom Med 1998;60:610-5.
29. Vgontzas AN, Tsigos C, Bixler EO, et al. Chronic insomnia and activity of the stress system: a preliminary study. J Psychosom Res 1998;45(1 Spec No):21-31.
30. Nofzinger EA, Buysse DJ, Germain A, Price JC, Miewald JM, Kupfer DJ. Functional neuroimaging evidence for hyperarousal in insomnia. Am J Psychiatry 2004;161:2126-8.
31. Espie CA. Insomnia: conceptual issues in the development, persistence, and treatment of sleep disorder in adults. Annu Rev Psychol 2002;53:215-43.
32. Harvey AG: A cognitive model of insomnia. Beh Res Ther 2002;40:869-93.
33. Taylor LM, Espie CA, White CA. Attentional bias in people with acute versus persistent insomnia secondary to cancer. Behav Sleep Med 2003;1:200-12.
34. Marchetti LM, Biello SM, Broomfield NM, Macmahon KM, Espie CA. Who is pre-occupied with sleep? A comparison of attention bias in people with psychophysiological insomnia, delayed sleep phase syndrome and good sleepers using the induced change blindness paradigm. J Sleep Res 2006;15:212-21.
35. Ayalon L, Ancoli-Israel S, Klemfuss Z, Shalauta MD, Drummond SP. Increased brain activation during verbal learning in obstructive sleep apnea. Neuroimage 2006;31:1817-25.
36. Drummond SP, Meloy MJ, Yanagi MA, Orff HJ, Brown GG. Compensatory recruitment after sleep deprivation and the relationship with performance. Psychiatry Res 2005;140:211-23.
37. Woodard JL, Grafton ST, Votaw JR, Green RC, Dobraski ME, Hoffman JM. Compensatory recruitment of neural resources during overt rehearsal of word lists in Alzheimer's disease. Neuropsychology 1998;12:491-504.
38. Tapert SF, Schweinsburg AD, Barlett VC, et al. Blood oxygen level dependent response and spatial working memory in adolescents with alcohol use disorders. Alcohol Clin Exp Res 2004;28:1577-86.
39. Clark VP, Lai S, Deckel AW. Altered functional MRI responses in Huntington's disease. Neuroreport 2002;13:703-6.
40. Ramsey NF, Koning HA, Welles P, Cahn W, van der Linden JA, Kahn RS: Excessive recruitment of neural systems subserving logical reasoning in schizophrenia. Brain 2002;125(Pt 8):1793-807.