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The Relation Between Cognitive Functioning and Self-Reported Sleep Complaints in Nondemented Older Adults: Results From the Bronx Aging Study

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Self-reported sleep complaints and current cognitive functioning were assessed in 375 nondemented participants ages 75 to 85 years (134 men and 241 women) as part of enrollment in the Bronx aging study, an ongoing longitudinal community-based study of cognitive aging. This study only reports on the baseline data collected from 1980 to 1983. Sleep complaints were common, occurring in about 25% of the sample. Furthermore, after controlling for depression, use of hypnotic medication, physical morbidity, age, and education, participants who reported longer sleep onset latencies performed significantly worse on measures of verbal knowledge, long-term memory and fund of information, and visuospatial reasoning. Participants who reported longer sleep durations did significantly worse on a measure of verbal short-term memory. These results suggest that perceived sleep is related to select objective cognitive abilities even when accounting for commonly recognized mediating variables, such as depression, medical comorbidity, age, or use of hypnotic medication. Given the restricted range of this nondemented sample, these results may underestimate the relation between cognitive abilities and sleep.

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Approximately one half of elderly individuals report at least one habitual sleep problem, with insomnia being the most prevalent condition (Foley, Monjan, Simonsick, Wallace, & Blazer, 1999; National Sleep Foundation, 2003). Although many of these cases may be due to an increased incidence of undetected apnea that is often associated with excessive daytime somnolence, late-life insomnia is often undiagnosed and untreated (Mendelson, 1995; National Sleep Foundation, 2003). It has long been believed that insomnia is a natural part of the aging process. However, recent studies have suggested that the sleep of older adults may actually be comparable to that of younger adults after controlling for such secondary causes of sleep disruption as medical–psychiatric comorbidity (Foley, Ancoli-Israel, Britz, & Walsh, 2004; McCrae et al., 2005; Ohayon, 2002; Ohayon & Vecchierini, 2005; Vitiello, Moe, & Pritz, 2002). Nevertheless, a sizable percentage of noncomplaining healthy older adults evidence objective sleep disturbances even after thorough screening for medical comorbidity and CNS active medication use (Vitiello, Larsen, & Moe, 2004).

Late-life insomnia complaints are associated with greater functional impairment and increased use of health care services (Gooneratne et al., 2003), complaints of physical ailments (Foley et al., 1995; Jensen, Dehlin, Hagberg, Samuelsson, & Svensson, 1998), use of hypnotic medication (Foley et al., 1995), and reduced quality of life (Léger, Scheuermaier, Philip, Paillard, & Guilleminault, 2001). Reported sleep problems are also associated with falls in older adults, even after controlling for other risk factors such as hypnotic use (Avidan et al., 2005; Brassington, King, & Bliwise, 2000). Prospective studies have also demonstrated that older adults with insomnia are at increased risk for cardiovascular disease (Leppavuori, Pohjasvaara, Vataja, Kaste, & Erkinjuntti, 2002), depression (Ford & Kamerow, 1989; Livingston, Blizard, & Mann, 1993), suicide (Turvey et al., 2002), early nursing home admission (Pollak, Perlick, Linsner, Wenton, & Hsieh, 1990), and all-cause mortality (Dew et al., 2003; Kojima et al., 2000; Newman et al., 2000).

Despite growing evidence of the effects of insomnia complaints on daytime cognitive performance in younger adults (Fulda & Schultz, 2001; Riedel & Lichstein, 2000), few studies have investigated this relation in older adults. This is particularly important as symptoms of insomnia may act as prodromal indicators for specific neurodegenerative disorders such as Parkinson's disease (Abbott et al., 2005) and be associated with greater memory impairment and accelerated functional decline in Alzheimer's disease (Carpenter, Strauss, & Paterson, 1995; McCurry, Logsdon, & Teri, 1999). Hart, Morin, and Best (1995) found that self-reported sleep problems were associated with poorer performance on measures of vigilance, recall memory, psychomotor speed, and executive functioning in a sample of 78 older adults. Vignola, Lamoureux, Bastien, and Morin (2000) and Bastien et al. (2003) found that certain insomnia complaints were associated with decrements in attention and concentration as well as visual memory. A recent sur-

vey of over 7,000 community-dwelling older individuals found that self-reported cognitive impairment was associated with shorter night sleep duration (SD; under 6 hr) and longer daytime naps (Ohayon & Vecchierini, 2005). Further evidence of a link between self-reported sleep complaints and reduced cognitive functioning comes from three recent longitudinal investigations (Cricco, Simonsick, & Foley, 2001; Foley et al., 2001; Jelicic et al., 2002). Significantly, each of these studies used dementia screening measures to assess for cognitive impairment and to our knowledge, no large-scale studies have examined the associations between self-reported sleep complaints and cognitive functioning as assessed by a battery of neurocognitive tests—a goal of this study.

Insomnia complaints are also highly associated with anxiety and depression (Weissman et al., 1996), and sleep quality has been identified as a mediating variable in recovery from geriatric depression (Reynolds et al., 1997). This suggests that depression in older adults may be an independent predisposing factor to cognitive decline and dementia (Bassuk, Berkman, & Wypij, 1998; Geerlins et al., 2000). Although depression in older adults is associated with cognitive deficits and increased mortality risk (Christensen, Griffiths, Mackinnon, & Jacomb, 1997) and insomnia has been demonstrated to be a risk factor for depression in older adults (Ford & Kamerow, 1989; Roberts, Shema, Kaplan, & Strawbridge, 2000), the relation between sleep, depression, and cognitive impairment has been largely overlooked in the studies reviewed here with older adults. Thus, another goal of this investigation was to determine whether the relation between subjective reports of poor sleep and reduced cognitive functioning remains robust after controlling for depression and other covariates that may impact sleep such as medical comorbidities, age, education, and use of hypnotic medications.

The Bronx Aging Study provides a unique opportunity to examine the relation between sleep and cognition in older adults. For over two decades, this community-based study followed an aged cohort with annual clinical interviews and neuropsychological testing (e.g., Aronson et al., 1990; Katzman et al., 1989; Verghese et al., 2002). On entrance to the study in 1980, participants were interviewed with a structured questionnaire that provided detailed information about their sleep as well as all pertinent covariates listed previously. The purpose of our investigation was to examine the relation between self-reported sleep complaints and current cognitive abilities, including processing speed, psychomotor dexterity, visual–spatial reasoning, verbal functioning, attention–concentration, and verbal short-term memory in a nondemented elderly sample. This is while controlling for the possible confounding effects of depression, hypnotic medication use, age, education, and physical morbidity. We hypothesized that although depression, hypnotic use, education, and physical health would partially account for this relation, sleep complaints would be independently associated with reduced cognitive functioning.

METHOD

Participants

The data for this study came from the inception cohort for the Bronx Aging Study, a longitudinal study of cognitive aging. Between 1980 and 1983, a total of 488 English-speaking, community-dwelling adults between 75 and 85 years of age were recruited and initially evaluated. Baseline and annual follow-up evaluations included extensive demographic and clinical interviews, medical and neurological examinations, and neuropsychological testing. Participants were recruited from senior citizen centers, by local newspaper advertisements, and by word of mouth. Potential volunteers were told that they would be offered a comprehensive yearly evaluation. All participants gave informed consent and understood the longitudinal nature of the study. There was no direct payment for participation, but transportation costs, lunch, and certificates of participation were provided for each participant. Whenever possible, a close friend or family member was also interviewed to confirm the history of the participant. Written informed consent was obtained at enrollment, and the local institutional review board approved the study protocol.

Participants were screened to rule out the presence of significant cognitive impairment at baseline. To be included in the study, participants were required to make eight or fewer errors on the Blessed Information–Memory–Concentration test (BIMC; Blessed, Tomlinson, & Roth, 1968). The BIMC is correlated with severity of Alzheimer's disease (Fuld, 1978; Grober et al., 1999), has high test–retest reliability, and has been validated against other mental status tests (Thal, Grundman, & Golden, 1986). Participants who exhibited a significant drop in cognition during annual reevaluations (i.e., total BIMC score of eight or increase of four points from baseline BIMC score) or who exhibited behavior suggestive of dementia observed by significant others or research staff, were referred for a dementia work-up. This included an EEG, a high-resolution computed tomography scan, and further neurological examination including determination of Hachinski (1978) and Rosen, Terry, Fuld, Katzman, & Peck (1980) ischemic scores. Exclusion criteria for the Bronx Aging Study included a diagnosis of any of the following health conditions: dementia, Parkinson's disease, liver disease, a known terminal illness, alcoholism, or severe visual or hearing impairment (e.g., corrected visual acuity greater than 20/200). Other than depression, no other psychiatric conditions were assessed.

Starting in 1986, all available data for participants with suspected cognitive impairment were reviewed and diagnosed during case conferences attended by neurologists and a neuropsychologist. In 2001, all of the suspected cases of cognitive impairment were reevaluated and rediagnosed according to current standards by clinicians who were not involved in the previous case conferences to account for subtypes of dementia and retrospectively excluded from participation in the study.

Accordingly, of the 488 initial Bronx Aging Study participants, 113 were diagnosed with some form of cognitive impairment within 2 years of baseline and excluded from our study, yielding a final sample total of 375 participants (240 women and 135 men). All analyses were conducted on data collected during this initial baseline period. Consequently, this study consists of data for 375 participants from the original cohort who were not diagnosed with dementia or cognitive impairment within 2 years of enrolling.

Measures

Baseline and follow-up evaluations included extensive demographic and clinical interviews including economic and work history (e.g., type of employment), social history (e.g., contact with friends and family), medical history (e.g., previous hospitalizations and operations), and current functional status (e.g., activities of daily living). Participants also received a physical and neurological exam and provided information about medical morbidity including present and past diagnoses of visual or hearing difficulties, hypertension, myocardial infarction, stroke, diabetes, seizure, Parkinson's disease, memory loss, and cigarette use.

Sleep measure. As this study was initiated prior to the advent of many of the standardized sleep measures currently in use, all participants were administered a detailed interview-based, 54-item sleep questionnaire. This was developed specifically for the Bronx Aging Study by Dr. Elliott Weitzman, one of the fathers of sleep medicine, based on extensive clinical observation. Among other questions about sleep, participants were asked about their initial sleep onset latency (SOL; less than 5 min, 5–15 min, 16–30 min, 31–60 min, or greater than 1 hr) and how many hours of sleep they normally get at night (3 hr or less, 3–4 hr, 5–6 hr, 7–8 hr, 9–10 hr, or more than 11 hr). Participants were also asked a single question about whether they had "insomnia or trouble sleeping" and how many times they normally awoke during the night (never or rarely, one to two times a night, three to four times, or 5 times or more). Regrettably, the interview did not distinguish between sleep onset and sleep maintenance insomnia; nor did it specify any specific time frame regarding the frequency or duration of sleep problems. In addition, the sleep measure has no documented psychometrics. Given the inherent difficulty in making valid diagnostic considerations from these data in the absence of more controlled operational definitions, all analyses focused on self-reported SD and SOL rather than the presence of insomnia.

Information was also obtained about use of hypnotic medication for sleep (none, rarely–less than once a month, one to four times per month, one to two times per week, three or more times per week, or daily) as well as regular use of alcohol to help with sleep (yes–no). Participants were asked about the occurrence of a number of dysomnias such as those relating to movement and breathing disturbances (never,

less than once a month, one to four times per month, one to two times a week, three to four times a week, five to six times a week, or seven or more times a week). However, as no objective measures of sleep were taken (e.g., polysomnography data), it was difficult to determine the presence of other sleep disorders, which may impact waking cognitive functioning (e.g., apnea, restless legs syndrome), and as such, data regarding dysomnias were not included in the final analyses.

Cognitive measure. Participants were evaluated with an extensive battery of neuropsychological tests. These include the BIMC (Blessed, Tomlinson, & Roth, 1968); Fuld object–memory evaluation (Fuld, Masur, Blau, Crystal, & Aronson, 1990); selective reminding task (Buschke & Fuld, 1974); category fluency test (Isaacs & Kennie, 1973); Raven's progressive matrices, Set A (Raven, 1977); Purdue pegboard test (Tiffin & Asher, 1948); and certain subtests from the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955). Participants for whom all measures were not administered were excluded from the study.

In this investigation, the pertinent outcome measures of cognition included the following verbal subtests from the WAIS: *Vocabulary* is a measure of word knowledge and long-term memory; *information* assesses general knowledge and long-term memory, and *similarities* is a measure of abstract reasoning. Attention and concentration were assessed by two measures: *Digit span* is a WAIS subtest in which participants hear progressively longer sequences of random numbers and recite them from memory forward and backward, and *months backward* from the BIMC in which participants were asked to recite from memory the months of the year in reverse. Scores above zero are indicative of concentration errors (2 points for uncorrected errors and 1 point for corrected mistakes). Short-term verbal memory was assessed by the *selective reminding task* in which participants heard 12 unrelated words and immediately attempted to repeat the entire list. Participants were reminded of any words they failed to recite and asked again to recall the entire list of 12 words. Over six trials, participants attempted to recall all 12 words while being reminded only of words they failed to mention each successive trial. Lower scores indicate greater difficulty with verbal learning and short-term verbal memory. Internal consistency for the selective reminding task is .73. (Ruff, Light, & Quayhagen, 1989).

Nonverbal abilities were assessed by the following WAIS subtests: *block design,* a test of visual–spatial reasoning; *digit symbol substitution*, a measure of processing speed and visual scanning; and *object assembly,* which assesses visual–perceptual processing and part–whole integration. Psychomotor skills were assessed by the Purdue pegboard test.

Clinical measure. The Zung (1965) Self-Rating Depression Inventory was administered to assess for the presence and severity of depression. Based on the Zung scoring system, scores ranged from less than 50 (*no depression*), 51 to 60 (*mild*

depression), 61 to 70 (*moderate depression*), and 71 to 80 (*severe depression*). Although the Zung Self-Rating Depression Inventory has now been largely supplanted by other depression measures, it has adequate demonstrated psychometric properties with older populations (McKegney, Aronson, & Ooi, 1988).

Procedure

The initial evaluations required 2 days to complete and included a mental status examination, functional rating, neuropsychological evaluation, and physical and neurological examinations. Standardized clinical–demographic interviews and cognitive assessments were administered either by a neuropsychologist or trained research assistants. Testing occurred between 10:00 a.m. and 2:00 p.m. and was scheduled according to the participants' preference. Interviews were administered after the neuropsychological testing.

Statistical Analyses

Zero-order bivariate correlations were conducted to determine the magnitude of the relation between the measures of cognitive functioning and both the sleep variables (SD and SOL) and the pertinent covariates. To determine the unique association between sleep and cognitive performance after controlling for covariates, separate one-way multivariate analyses of covariance (MANCOVAs) were conducted for SD and SOL. Depression, age, education, hypnotic use, and medical comorbidities were entered as covariates, and all cognitive performance tests were the dependent measures with the exception of selective reminding. Given that this latter measure was included later in the inception of the study, only about one half of the sample completed this test ($n = 210$), thus greatly reducing the power when including this variable in the analyses. Therefore, two separate analyses of variance (ANCOVAs) were conducted to examine the relation with performance on the selective reminding task and sleep while controlling for the same covariates as in the MANCOVAs.

RESULTS

The final sample consisted of 375 participants with a mean age of 79.6 years ($SD = 3.15$). Women represented 64.3% of the sample ($n = 241$), and most participants were Caucasian (90.4%) and primarily of Jewish or Catholic faith and of Italian, Irish, or German ethnic backgrounds, which was representative of the elderly population of the Bronx at the time of the study onset. Table 1 summarizes the main sociodemographics and clinical characteristics of the sample. The sample was relatively healthy with most participants reporting between one and two comorbid

TABLE 1
Sociodemographic and Clinical Characteristics of Final Sample

Demographics	
Age (*M* ± *SD*)	79.43 ± 3.02
Gender	
Male	35.7%
Female	64.3%
Race	
Caucasian	90.4%
African American	9.1%
Median education level	7–9 years
Marital status	
Married	35.2%
Widowed	54.9%
Divorced, separated, or never married	9.9%
Clinical characteristics	
Morbidity (*M* ± *SD*)	1.72 ± 1.51
One condition	22.4%
Two conditions	21.6%
Three or more conditions	29.0%
Zung depression (*M* ± *SD*)	46.05 ± 11.69
Hypnotic medication	17.1%
Sleep characteristics	
Sleep onset latency	
< 5 min	30.5%
5–15 min	26.7%
16–30 min	15.3%
31–60 min	13.1%
> 60 min	14.2%
Sleep duration	
≤ 4 hr	7.5%
5–6 hr	33.9%
7–8 hr	51.9%
9–10 hr	6.7%

Note. $N = 375$. Morbidity = total number of current health conditions (range 0–7) and Zung depression (range 20–80); hypnotic medication = use once a month or more.

health problems. The most common medical conditions were hypertension (41.2%) and mild–correctible sensory difficulties (visual = 32.9%; auditory = 27.0%). In addition, 65% of participants scored below threshold for mild depression with women scoring significantly higher than men (*M* difference = 4.87), $t(317) = -3.63$, $p < .001$.

Sleep Variables

Nearly one fourth of the sample answered "yes" to having insomnia or trouble sleeping (22.7%) with a significantly greater proportion of women ($n = 63$) than

men ($n = 22$) responding affirmatively to this item, $\chi^2(1, N = 372) = 4.92$, $p = .03$. However, as there were no significant differences in SOL or SD according to sex, all subsequent analyses were collapsed across this variable. As expected, SD and SOL were inversely associated, $r(353) = –.37$, $p <. 001$. Many participants reported awakening after falling asleep (54.4% reported one or two awakenings a night, 25.6% reported three or four awakenings, and 5.1% reported five or more). Not surprisingly, significant differences for SD were noted according to number of nocturnal awakenings, $F(3, 359) = 6.72$, $p < .001$. Participants who never or rarely wakened during the night slept significantly longer than those who awakened one to two times, three to four times, or five or more times a night. However, there were no differences in SOL between participants who reported nocturnal awakenings and those who did not.

Seventy-nine percent of the sample reported never using hypnotic medication and 3.4% used it rarely (i.e., less than once a month). Only 17.1% reported using hypnotics at least on a monthly basis. This figure dropped to 10.9% on a weekly basis with 5.6% reporting daily use. Although these figures appear low compared to today's standards, it must be remembered that hypnotic medication was far less commonly prescribed at the time of the study inception. As expected, participants who used hypnotic medication once a month or more had significantly longer SOL, $t(340) = –3.30$, $p = .001$, and reported shorter SD, $t(336) = 3.2$, $p = .002$, than those who did not. In addition, 4% of the participants stated they regularly used alcohol to help them fall asleep, although alcohol use was unrelated to SD or SOL. Depression was significantly associated with longer SOL, $r(313) = 0.29$, $p < .001$, and shorter SD, $r(308) = –0.20$, $p < .001$. Table 2 summarizes the zero-order correlations between the two sleep variables and all other measures.

Cognitive Performance Variables

As seen in Table 2, SOL but not SD was significantly and inversely related to all of the verbal-based cognitive measures with longer latencies associated with poorer cognitive functioning. Nonverbal cognitive performance was by and large, unrelated to both sleep indexes. No significant differences were found in cognitive performance according to race, marital status, physical morbidity, alcohol use, or number of nocturnal awakenings. However, depression was moderately associated with both longer SOL, $r(313) = 0.29$, $p < .001$, and shorter SD, $r(308) = –0.20$, $p < .001$. In addition, monthly use of hypnotic medications was associated with longer SOL, $r(342) = 0.18$, $p < .001$, and shorter SD, $r(338) = –0.17$, $p < .001$. Furthermore, scores on certain cognitive tests were significantly related to demographic and clinical variables. Age was inversely associated with poorer concentration as measured by months backward, $r(333) =.15$, $p = .005$, and digit span, $r(306) = –.13$, $p = .02$, as well as verbal short-term memory; selective reminding, $r(258) = –.17$, $p = .007$. Using hypnotic medication once a month or more was also related

TABLE 2
Zero-Order Correlations Between Sleep Onset Latency and Sleep Duration With Cognitive Measures and Pertinent Covariates

Variables	*Sleep Onset Latency*	*Sleep Duration*
Verbal		
Months backward	.18***	–.05
Digit span	–.13*	.02
Information	–.19***	.07
Vocabulary	–.18***	.05
Similarities	–.16**	.06
Selective reminding	–.08	–.13*
Nonverbal		
Digit symbol	–.14**	–.03
Block design	–.09	.07
Object assembly	–.03	.02
Purdue pegboard	.05	–.12*
Covariates		
Depression	.29***	–.19***
Age	.01	–.03
Education	–.07	.09
Medical comorbidities	–.02	–.05
Hypnotic use	.18***	–.17**

*$p < .05$. **$p < .01$. ***$p < .001$.

to increased concentration errors during months backward, $t(308) = –2.2$, $p = .027$. As expected, depression negatively correlated with performance on all cognitive measures (ps < .001), and education was associated with better performance on all cognitive measures (ps < .001).

To determine the unique relation between self-reported sleep and cognitive performance, two separate one-way MANCOVAs (SOL and SD) were conducted for all cognitive measures (except selective reminding). There were five categorical levels of SOL (<5 min, 5–15 min, 16–30 min, 31–60 min, and >60 min) and four levels of SD (<4 hr, 5–6 hr, 7–8 hr, >9 hr) as the independent factors. Depression, age, years of education, total number of physical comorbidities, and monthly use of hypnotic medication were entered as covariates. These results, as well as the separate ANCOVA for selective reminding, are presented in Tables 3 and 4 and discussed next.

SOL

Increased SOL remained significantly associated with reduced cognitive performance, even after controlling for the effects of the covariates, $F(27, 630) = 1.81$, $p < .008$. As seen in Table 3, follow-up univariate ANCOVAs reveal significant dif-

TABLE 3
Univariate Analyses for Cognitive Performance Measures by Sleep Onset Latency After Controlling for Depression, Age, Education, Medical Comorbidities, and Hypnotic Use

	< 5 min[a]		5–15 min[b]		16–30 min[c]		31–60 min[c]		> 60 min[d]			
Cognitive Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	*p*
Info	16.94	5.1	16.37	5.0	16.85	5.10	14.60	5.3	12.81	5.7	4.12	.007
Vocab	50.69	16.1	49.77	16.8	51.52	14.64	41.60	16.0	39.30	17.7	5.41	.001
Sim	10.54	6.9	8.42	6.4	9.64	6.30	7.61	5.1	6.59	5.6	1.65	*ns*
SelSum	43.24	9.5	40.00	10.6	42.76	9.61	40.59	9.4	41.07	11.0	0.40	*ns*
MOSB	0.32	0.6	0.32	0.6	0.48	0.80	0.46	0.7	0.63	0.9	0.58	*ns*
DSymb	31.79	11.0	30.60	10.8	28.00	11.70	27.69	10.4	25.07	12.1	1.25	*ns*
BD	18.94	7.9	20.55	8.2	21.55	8.70	18.40	6.8	15.48	6.2	3.53	.02
OA	17.21	8.1	17.24	7.1	18.61	7.50	15.11	6.8	17.74	7.3	1.83	*ns*
Purdue	12.17	1.6	11.90	2.0	11.79	2.00	12.06	1.7	12.48	2.6	1.61	*ns*
DSpan	10.13	2.3	10.00	2.4	10.24	1.90	9.57	1.6	8.96	1.6	1.50	*ns*

Note. MOSB = months backwards; DSymb = digit symbol; BD = block design; OA = object assembly; Purdue = Purdue pegboard; Dspan = digit span; Info = information; Vocab = vocabulary; Sim = similarities; SelSum = selective reminding task.

[a]$n = 68$. [b]$n = 62$. [c]$n = 35$. [d]$n = 27$.

TABLE 4
Univariate Analyses for Cognitive Performance Measures by Sleep Duration After Controlling for Depression, Age, Education, Medical Comorbidities, and Hypnotic Use

	≤ 4[a]		5–6[b]		7–8[c]		> 9[a]			
Cognitive Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	*p*
Info	12.91	5.7	15.31	4.6	16.67	5.50	14.09	5.8	3.92	.009
Vocab	36.09	18.5	47.32	15.0	49.10	17.44	41.27	16.9	3.68	.01
Sim	6.00	5.9	8.71	5.8	9.42	6.60	5.45	5.0	3.99	.001
SelSum	44.36	2.9	43.55	1.1	40.36	0.90	36.28	3.0	2.71	.05
MOSB	0.45	0.7	0.46	0.7	0.33	0.70	0.64	0.7	1.49	*ns*
DSymb	26.18	11.7	29.64	10.8	29.40	11.30	28.36	13.0	0.48	*ns*
BD	14.55	7.2	18.68	7.5	19.99	7.70	20.73	11.7	1.03	*ns*
OA	15.91	5.5	17.04	7.5	17.55	7.60	18.18	8.0	0.03	*ns*
Purdue	11.94	1.8	12.32	2.0	11.91	1.80	12.10	3.0	0.68	*ns*
DSpan	9.18	1.7	9.68	1.9	9.91	2.30	10.36	2.2	0.20	*ns*

Note. MOSB = months backwards; DSymb = digit symbol; BD = block design; OA = object assembly; Purdue = Purdue pegboard; Dspan = digit span; Info = information; Vocab = vocabulary; Sim = similarities; SelSum = selective reminding task.

[a]$n = 11$. [b]$n = 76$. [c]$n = 124$.

ferences for three individual subtests: information ($p < .007$), vocabulary ($p < .001$), and block design ($p = .016$), with cognitive performance demonstrating a sharp drop as sleep latencies exceed 30 min and, particularly, 60 min. The ANCOVA for selective reminding was not significant for SOL when controlling for other covariates.

SD

SD was not associated with reduced cognitive performance in either sphere when pertinent covariates were controlled. However, as seen in Table 4, univariate analyses demonstrate significant effects for three verbal WAIS subtests: information ($p < .01$), vocabulary ($p < .01$), and similarities ($p < .008$). All three groups demonstrated an attenuation of performance with both short and long SDs. In addition, the ANCOVA for selective reminding remained significant, $F(3, 265) = 2.93$, $p < .03$, indicating that longer SD (>9 hr) was associated with reduced verbal short-term memory.

DISCUSSION

Our results are generally consistent with prior research indicating that self-reported sleep complaints are common in older adults, even in a sample prescreened

for prior health problems and dementia and after controlling for depression. Sleep complaints, particularly SOL, were associated with certain cognitive decrements, although the most consistent finding of past studies, problems with attention and concentration, was not replicated in this study. One possible reason for this discrepancy was the use of a composite score for digit span rather than separate scores for digits forward and backward as was used in previous studies (Bastien et al., 2003; Vignola et al., 2000). Although a combined score for digit span is commonly used in research, doing so may obscure differences in participants' level of attention (digit forwards) and concentration (digit backwards; Lezak, 1995).

Three primary findings can be gleaned from our study. First, an inverse linear trend was found between SOL and performance on verbal cognitive tasks. More important, SOL remained significantly related to performance on information, vocabulary, and block design from the WAIS even after accounting for robust main effects for education level, age, hypnotic medication use, and depression. Second, although SD was unrelated to cognitive performance on the multivariate analyses, hours of sleep was inversely associated with a number of measures of verbal cognitive performance, including two of the same measures that were associated with SOL (vocabulary and information). Third, self-reported SD was associated with verbal short-term memory after the covariates were controlled, suggesting that extended sleep is related to decrements in short-term memory.

A principal strength of this investigation was that variables known to affect cognitive performance and sleep were controlled for in analyses. This point was underscored by the fact that all of the covariates, except number of medical comorbidities, demonstrated robust main effects for both verbal and nonverbal cognitive functioning ($ps < .001$). In addition, both depression and hypnotic medication use were highly associated with impaired sleep. Previous studies investigating cognitive functioning and sleep parameters in older adults have been inconsistent in how these variables have been treated. For example, Hart et al. (1995) provided little information on depression severity or hypnotic use in their sample, other than suggesting that it was unrelated to cognitive performance. Similarly, Vignola et al. (2000) and Bastien et al. (2003) did not control for depression, although both of their groups of poor sleepers had significantly higher scores on a depression measure than their control group of good sleepers. Foley et al. (2001) found that insomnia complaints did not predict cognitive decline after controlling for depression, although participants depressed at baseline had a significantly increased likelihood of dementia or cognitive decline (or both) at follow-up. In contrast, Cricco et al. (2001) found chronic insomnia was an independent risk factor for cognitive decline but only in nondepressed men. Jerelic et al. (2002) reported that subjective sleep complaints no longer predicted cognitive decline after depression was controlled.

Although depression in later life often affects cognitive functioning, not all facets of cognition are equally affected. Pronounced deficits tend to occur in motor performance, processing speed, and attention, whereas many verbal abilities and remote

memory are less impacted (Boone et al., 1995; Christensen et al., 1997). Impaired processing speed, working memory, and other executive functions account for most of the variance in cognitive deficits associated with late-life depression, especially in nonverbal abilities such as visuospatial functioning (Lockwood, Alexopoulos, & van Gorp, 2002; Nebes et al., 2000). Thus, our finding that verbal ability performance (vocabulary and information) remains significantly related to SOL after controlling for depression may reflect the fact that these tasks are not as dependent on processing speed or working memory and are relatively resistant to the effects of depression. Nonverbal tasks such as block design do require processing speed for efficient performance, and yet remained related to sleep onset latencies after controlling for depression. Furthermore, memory tasks such as the selective reminding task are highly vulnerable to the effects of late-life depression (Christensen et al., 1997) but remained significantly related to SD after controlling for depression and other covariates. Thus, our findings suggest that the relation between sleep and select cognitive abilities exists independently of depressive symptomatology.

Age-related changes in sleep architecture often reveal reduced slow wave sleep and delta activity (Carrier, Land, Buysse, Kupfer, & Monk, 2001; Landolt & Borbely, 2001), which is believed to provide recovery for the prefrontal cortex and facilitate cortical reorganization (Steriade & Amzica, 1998; Werth, Achermann, & Borbely, 1997). Although younger adults rebound from sleep loss with increased delta activity during NREM in frontal and parietal brain regions, normal older adults exhibit diminished "frontal predominance" and recovery of slow wave sleep (Dijik, Duffy, & Czeisler, 2000; Munch et al., 2004). Significant associations have also been reported between delta power in the prefrontal cortex during the first NREM episode and waking performance on tasks of nonverbal planning and verbal fluency in healthy older adults (Anderson & Horne, 2003). Taken together, age-related declines in prefrontal cortex functioning and slow wave sleep may result in cognitive decrements if trouble falling or staying asleep results in missed delta activity during the first NREM episode.

Our results should be interpreted with caution given a number of important caveats. Our sleep measure was created for the study and has no documented psychometric properties. Furthermore, the absence of any objective corroborating data such as PSG recordings makes it impossible to verify these self-reports and precludes the detection of other sleep disorders, such as sleep apnea and restless legs syndrome, which may account for obtained cognitive decrements. In addition, the cross-sectional nature of the study makes it difficult to determine causal relations between sleep problems and cognitive functioning. Also, as our participants were predominantly Caucasian and comparatively healthy, the sample used in this study may not represent the general aged population. Similarly, given the restricted range of cognitive variables in our nondemented sample of older adults, findings may underestimate the relation between sleep and cognitive performance. In addition, our measure of depression, the Zung Depression Scale, is largely based on so-

matic symptoms of depression, and as such, may have overstated its association with sleep-based complaints.

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