Self-Reported Sleep Quality Predicts Poor Cognitive Performance in Healthy Older Adults

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Although cognitive performance declines with increasing age in a variety of domains from intelligence to attention, there is substantial individual variability in the magnitude of these age-associated cognitive decrements (Ardila, 2007). Suggested sources for this variability have focused on individual differences in the amount of age-associated brain dysfunction such as cortical atrophy (Raz, Gunning-Dixon, Head, Dupuis, & Acker, 1998), white matter pathology (Gunning-Dixon & Raz, 2000), reductions in neurotransmitter receptor binding (Bäckman et al., 2000), etc. Another potential contributor to individual variability in age-associated cognitive impairment is a more functional basis—the amount, timing, and quality of sleep. Studies using experimental sleep restriction and sleep deprivation have documented dose-related cognitive impairments even in young adults (e.g., van Dongen, Maislin, Mullington, & Dinges, 2003). Many elderly individuals report chronic problems with sleep onset, duration, and maintenance and with associated adverse daytime consequences such as drowsiness, falls, and functional disability (Buysse et al., 1991; Vaz Fragoso & Gill, 2007). There is also evidence, mainly in younger adults, that poor subjective sleep quality, as seen in insomnia, may also be associated with cognitive impairments (Fulda & Schulz, 2001). Although there is a growing literature on the cognitive performance of older persons recruited as having chronic insomnia (e.g., Haimov, Hanuka, & Horowitz, 2008; Hart, Motin, & Best, 1995; Vignola, Lamoureux, Bastien, & Morin, 2000), there is relatively little information available on the degree to which sleep disturbance may contribute to interindividual variability in cognitive performance in the type of healthy community volunteers used in most normal aging studies. Also, many of the studies investigating the relation between sleep quality and cognitive performance do not control for conditions known to impair both sleep and cognition (e.g., cerebrovascular disease, medications, depression). Such conditions could confound any conclusions drawn about the role that sleep disruption plays in the magnitude of the cognitive decrements associated with normal aging.

Participants in the present study were community-dwelling individuals who were “not” recruited based on their sleep. Rather, they were participating in a naturalistic study designed to examine whether the anticholinergic medications taken by many older persons may contribute to age-associated decrements on tests thought to assess basic cognitive mechanisms (processing speed, attentional control, and working memory). Information on sleep quality was obtained because elderly individuals often use anticholinergic drugs such as antihistamines as a sleep aid (Basu, Dodge, Stoehr, & Ganguli, 2003), and thus, it was possible that sleep disturbance might contribute to any relation found between participants’ cognitive performance and their anticholinergic drug burden. The present analysis examined the relationship between sleep quality and cognitive performance while controlling for the effects of some common confounding conditions including anticholinergic drug burden.

Methods

Participants

Participants were 65–80 years old and were recruited from the community by newspaper advertisements and by letters sent to persons who had previously expressed interest in participating in aging research. The study was described as investigating whether medications (both prescription and over-the-counter [OTC]) taken by many older persons for various medical conditions may play a role in the cognitive problems that occur with increasing...
age. A nurse practitioner skilled in geriatric assessment obtained a medical history and performed a physical and neurological exam. All prescription and OTC medications taken in the past 24 hr were recorded. The participants' current medical conditions and medications list were also obtained from their primary care physicians (PCPs). To participate, participants' medical history could not include evidence of central nervous system pathology (e.g., stroke, Parkinson’s disease) or substance abuse, nor could they be taking prescription psychoactive medications (e.g., benzodiazepines, narcotics). They could not report having been diagnosed with major depression within the past 5 years nor have a present score of 15 or more on the Geriatric Depression Scale (GDS; Yesavage et al., 1983), a self-report instrument that quantitates depressive symptomatology on a 30-point scale. It is important to note that the GDS does not include questions about somatic symptoms, such as sleep disturbance. Three persons were excluded based on their GDS score. To determine whether any participants had a cognitive impairment suggestive of a dementia, participants were given the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS; Randolph, 1998), which assesses performance in a number of cognitive domains. Persons scoring more than 2 SDs below the mean for their age group were excluded from the present analysis.

There were no inclusion or exclusion criteria based on sleep. Sleep quality was, however, assessed by a widely used questionnaire (Pittsburgh Sleep Quality Index [PSQI]) with established reliability and validity (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) which assesses subjective sleep quality and quantitative sleep–wake parameters (measures of sleep latency, duration, and efficiency derived from self-report) over the preceding month. Responses to the 19 questions in the PSQI are scaled onto seven component scores, which are totaled to provide a global PSQI score ranging from 0 to 21, with higher scores representing worse sleep. Participants were divided into two groups based on their total PSQI score: good sleepers with a score of 5 or less (N = 108) and poor sleepers with a score of 6 or more (N = 49). Previous studies have shown that this cutoff yields groups significantly different in polysomnographic evidence of sleep disruption (Buysse et al., 1989) and that it discriminates individuals with primary insomnia from controls with a sensitivity of 98.7% and a specificity of 84.4% (Backhaus, Junghans, Broocks, Riemann, & Hohagen, 2002), although this test by itself is not sufficient to make a diagnosis of insomnia.

**Behavioral Measures**

Data were available from tasks commonly used in the cognitive aging literature to assess basic processing operations postulated to underlie the age-related decline in cognitive performance (Braver & West, 2008; Salthouse, 1996)—information-processing speed, working memory, and inhibitory function. There were also results from tests commonly used to assess set shifting, episodic memory, and abstract reasoning, aspects of cognition which also show major declines with increasing age.

**Information processing speed.**—The speed with which participants processed information was measured by the time they took to make two types of simple comparison. In the perceptual comparison task, they had to determine whether two shapes presented side by side on a computer screen were physically identical. They pressed a button with one hand if the two shapes were the same, and with the other if they were different. In the conceptual comparison task, they saw two stimuli (single letters and digits) and had to decide whether they were both from the same category (i.e., both letters or both digits) or whether they were from different categories (e.g., L 5). Mean response time (RT) for “same” trials in both tasks was used in the analyses.

**Working memory.**—Working memory is viewed as a limited-capacity work space which can be flexibly allocated between the processing and temporary storage of information. In the N-Back test (Dobbs & Rule, 1989), participants heard a string of digits presented one digit every 2 s. As they heard each digit, they were to say the digit that was N-Back in the string, where N ranged between 0 and 5. In the N = 0 condition, they repeated the digit they just heard. In the N = 1 condition, they were to say the digit immediately before the digit they just heard; for example, if given the digit string “5, 2, 7 …,” they would say nothing after the first digit; after hearing “2,” they would say “5”; after hearing “7,” they would say “2,” etc.). In the N = 2 condition, using the same example, the participants would say nothing after the “5” or the “2,” whereas after the “7” they would say “5,” etc. The participants were given two digit strings at each N, with each string requiring 10 responses. A string was presented until the participant made an error. The score on each string was the number of correct responses given before making an error, for a maximum correct on a string of 10. The result used in these analyses was the sum of the participant’s highest scoring string in each of the six conditions (i.e., N = 0–5) for a maximum score of 60.

The second working memory task was the Letter–Number Sequencing subtest of the Wechsler Adult Intelligence Scale III (Psychological Corporation, 1997). Participants were shown on a computer screen a string of numbers and letters (e.g., 6, M, 3, K) one item per second. After seeing all the items, they were to first recall the numbers in ascending order and then the letters in alphabetic order (i.e., 3, 6, K, M). The number of letters and numbers in a list started at two and increased up to eight. The age-normed scaled score was used in this analysis.
Inhibitory function.—Two tasks assessed the efficiency with which participants could inhibit prepotent responses that were irrelevant in the current context. In a computerized version of the Stroop test (Spieler, Balota, & Faust, 1996), a stimulus word appeared on a computer monitor and the participant had to name aloud the color of the letters making up the word. The vocal response triggered a voice key stopping a timer started by presentation of the stimulus. In one condition (incongruent), the stimuli were color names (“red,” “green,” “yellow,” or “blue”) presented in an incongruent color (e.g., the word “red” was spelled out with green letters). In the other (neutral), the stimuli were four noncolor words (bad, poor, deep, and legal) also presented in one of the four colors. Trials from the two conditions were randomly intermixed. The measure of inhibitory efficiency was the “interference ratio” equal to the mean RT on incongruent trials divided by the mean RT on neutral trials.

The Hayling test (Burgess & Shallice, 1996) compared the time participants took to generate a one-word ending with a sentence whose semantic context was highly constraining (e.g., “He scraped the cold food from his ____”). For one set of sentences (part A), participants were to generate a one-word ending that made sense (e.g., “plate,” “dish”), whereas for another set of sentences (part B) they had to generate a nonsensical ending for each sentence. Thus, part B required that the participant inhibit highly salient responses in order to produce a meaningless ending. Each sentence was presented auditorially by a computer, and in the place where the last word would fall, there was a tone which was the signal to respond. The measure of inhibitory efficiency was the “interference ratio” equal to mean RT on part B divided by part A.

Attention shifting.—The ability of participants to shift between attentional sets was assessed by the Trail Making Test—part B (Reitan & Wolfson, 1995). Here, the participant must as quickly as possible draw a line connecting 25 encircled letters and numbers, alternating between numbers and letters in the appropriate order (i.e., 1, A, 2, B, 3, C, etc.). The score used in the analysis was the age-normed deviation quotient (mean deviation quotient = 100, SD = 15).

Abstract reasoning.—Participants were given the Test of Nonverbal Intelligence (TONI) III (Brown, Sherbenou, & Johnson, 1997), which assesses abstract reasoning using a matrix analogy paradigm. It is considered a measure of fluid intelligence. The score used in the analysis was the age-normed deviation quotient (mean deviation quotient = 100, SD = 15).

Episodic memory.—The Logical Memory Test from the Wechsler Memory Scale–Revised (Wechsler, 1987) was used to measure both immediate and delayed recall of two short stories. Age-normed scaled scores from the MOANS (Ivnik et al., 1996) were used in the analysis.

The last measure of cognitive function examined was the age-corrected index score from the RBANS, which served as a measure of general neuropsychological status.

Finally, in addition to using the GDS to exclude potential cases of clinical depression (scores ≥15), we compared total GDS scores in good and poor sleepers because sleep dysfunction is associated with elevated depressive symptomatology in community-dwelling elderly adults (Newman, Enright, Manolio, Haponik, & Wahl, 1997). The range of scores on this measure is truncated at the upper end (≥15) in the present study due to its use as a depression screen. In addition to the total GDS score, we also examined scores on two subsets of GDS questions. A factor analysis of GDS data from over 300 community-dwelling elderly adults (Sheikh et al., 1991) identified a cluster of nine questions dealing with mood symptoms (e.g., sadness, helplessness, worry) and another six questions dealing with functional symptoms (e.g., difficulty concentrating and making decisions, loss of motivation). Bäckman, Hill, and Forsell (1996) found that in nondepressed elderly individuals, self-reported functional symptoms, especially those related to the ability to focus and maintain attention, were more strongly associated with actual cognitive performance than were mood symptoms.

The PSQI, RBANS, GDS, and Trail Making Test were administered in the first of two testing sessions, with the remaining measures being administered in a second session approximately 2 weeks later. It should be noted that Backhaus et al. (2002) found PSQI scores to be very stable across 45 days, with a test–retest correlation of .86. Thus, it is likely that the PSQI scores derived in the first session are also relevant to performance in the second session.

Potential Confounds

There are a number of conditions common in older individuals that are associated with both sleep disturbance and cognitive impairment, and thus it is important to examine whether any differences found between the good and poor sleep groups could result from a differential prevalence of these conditions in the two groups. Cerebrovascular disease is widespread after age 70 years, as evidenced by white matter hyperintensities (WMHs) on structural magnetic resonance scans (Longstreth et al., 1996). WMHs have been linked to impairments in cognitive function (Gunning-Dixon & Raz, 2000), depressive symptomatology (Nebes et al., 2001), and sleep (Culebras, 2004). In the present study, results from the medical questionnaire, the physical exam, and the participant’s PCP were used in an algorithm (Framingham Stroke Risk Profile) that estimates an individual’s percent risk for having a stroke within 10 years (Wolf, D’Agostino, Belanger, & Kannel, 1991). This score
served as a composite measure of the cerebrovascular risk factors (e.g., hypertension, diabetes) present in that person. The Stroke Risk Profile has been shown to be strongly related (r = .68) to the volume of WMHs present in community-dwelling elders (Jeerkathil et al., 2004), and there is some evidence that higher stroke risk is associated with cognitive decline in an aged sample (Brady, Spiero, McGlincey-Berroth, Milberg, & Gaziano, 2001).

Any attempt to link cognitive performance to sleep quality must deal with the possible confound of depression. Depressive symptomatology is associated with cognitive impairment even in older individuals who are not clinically depressed (Bäckman et al., 1996), as well as being associated with increased sleep disturbances (Newman et al., 1997). Thus, it was essential to examine whether depressive symptomatology (here the total GDS score) accounts for any group differences in cognitive performance.

Another potential confound is use of sleep medications. Persons who have trouble sleeping would be expected to use more sleep medications, some of which can affect cognitive performance (Hilmer et al., 2007). Although individuals using prescription sedatives and narcotics were excluded from this study, participants could be using other sleep medications. Information about use of sleep medications is available from the PSQI itself, and that information was used in the present analysis. A related issue is participants’ use of anticholinergic (muscarinic blocking) drugs. Anticholinergic agents are used to treat medical conditions common in older persons (e.g., urinary incontinence, ulcers) and are associated with substantial cognitive impairment (Wesnes, Simpson, & Kidd, 1988). Older individuals also often use OTC drugs with an anticholinergic effect (e.g., diphenhydramine), as a sleep aid (Basu et al., 2003).

This is especially likely when their sleep is disrupted by pain or respiratory illness because anticholinergics are included in many “night time” versions of OTC pain and cold medications. To determine whether differential use of anticholinergics might confound the present results, we measured serum anticholinergic activity (SAA) using a radioreceptor assay which quantifies the cumulative concentration (pmol/ml) of anticholinergic medications in the participants’ blood (Mulsant et al., 2003).

### RESULTS

The participant characteristics and behavioral results for poor sleepers (N = 49) and good sleepers (N = 108) are shown in Table 1, which also reports the results of the two-group t-tests (significant values are indicated in bold). The groups did not differ in age or education. Poor sleepers did perform significantly worse than good sleepers on the RBANS (p = .009), the TONI (p = .001), part B of the Trail Making Test (p = .016), and one of the two working memory measures (N-Back, p = .01). On the other working memory task, while poor sleepers did have lower mean scores than good sleepers, the difference was not significant (p = .17). There was no evidence that reported sleep problems were related to information-processing speed, inhibitory function, or verbal memory. The GDS differed between the two groups with poor sleepers reporting more depressive symptomatology (p = .001). On the subset of GDS questions dealing with functional symptoms (e.g., decreased

### Table 1. Participant Characteristics and Behavioral Results for Good (PSQI Scores ≤5) and Poor (PSQI Scores >6) Sleepers

<table>
<thead>
<tr>
<th></th>
<th>Good Sleepers, N = 108</th>
<th>Poor Sleepers, N = 49</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>72.07 ± 4.03</td>
<td>72.33 ± 4.40</td>
</tr>
<tr>
<td>Education</td>
<td>13.52 ± 2.04</td>
<td>13.04 ± 1.70</td>
</tr>
<tr>
<td>% 10-year stroke risk</td>
<td>11.78 ± 7.70</td>
<td>13.44 ± 10.03</td>
</tr>
<tr>
<td>Body mass index</td>
<td>28.8 ± 6.14</td>
<td>28.6 ± 4.47</td>
</tr>
<tr>
<td>SAA (pmol/ml)</td>
<td>1.56 ± 1.86</td>
<td>1.56 ± 1.61</td>
</tr>
<tr>
<td>GDS total</td>
<td>4.14 ± 3.39</td>
<td>4.14 ± 3.39</td>
</tr>
<tr>
<td>GDS—mood symptoms/9</td>
<td>0.49 ± 0.97</td>
<td>0.69 ± 1.29</td>
</tr>
<tr>
<td>GDS—functional symptoms/6</td>
<td>1.09 ± 1.18</td>
<td>1.90 ± 1.52</td>
</tr>
<tr>
<td>RBANS—index score</td>
<td>98.93 ± 12.62</td>
<td>93.45 ± 10.77</td>
</tr>
<tr>
<td>TONI—deviation quotient</td>
<td>100.34 ± 12.07</td>
<td>92.86 ± 9.25</td>
</tr>
<tr>
<td>Trails B—scaled score</td>
<td>11.43 ± 2.48</td>
<td>10.38 ± 2.51</td>
</tr>
<tr>
<td>N-Back (max = 60)</td>
<td>34.43 ± 13.18</td>
<td>28.57 ± 12.72</td>
</tr>
<tr>
<td>Letter—Number Sequencing scaled score</td>
<td>11.66 ± 2.98</td>
<td>10.92 ± 3.37</td>
</tr>
<tr>
<td>Stroop interference ratio</td>
<td>1.18 ± 0.10</td>
<td>1.16 ± 0.10</td>
</tr>
<tr>
<td>Hayling interference ratio</td>
<td>4.10 ± 3.68</td>
<td>4.76 ± 3.39</td>
</tr>
<tr>
<td>Conceptual comparison (ms)</td>
<td>797.0 ± 125.4</td>
<td>819.3 ± 172.4</td>
</tr>
<tr>
<td>Perceptual comparison (ms)</td>
<td>767.5 ± 142.9</td>
<td>773.7 ± 150.8</td>
</tr>
<tr>
<td>Logical memory IR-scaled score</td>
<td>9.91 ± 2.80</td>
<td>9.56 ± 3.07</td>
</tr>
<tr>
<td>Logical memory DR-scaled score</td>
<td>10.19 ± 2.84</td>
<td>9.79 ± 2.92</td>
</tr>
</tbody>
</table>

Notes: SAA = serum anticholinergic activity; GDS = Geriatric Depression Scale; RBANS = Repeatable Battery for the Assessment of Neuropsychological Status; TONI = Test of Nonverbal Intelligence; IR = immediate recall; DR = delayed recall.

*Data missing on one person.*
motivation and concentration, problems making decisions), poor sleepers reported more difficulty than did good sleepers ($p = .001$), whereas there was no group difference on the subset of questions dealing with mood symptoms ($p = .28$).

To determine which of the aspects of sleep assessed by the PSQI were most strongly related to cognition, we examined the Pearson bivariate correlation coefficients between those measures showing significant group differences and three specific sleep variables derived from self-reported information in the PSQI. By examining specific participants’ responses to individual questions in the PSQI, we derived measures of sleep-onset latency (in minutes), sleep duration (in hours), and percent sleep efficiency (time spent asleep divided by the time spent in bed). Sleep duration correlated significantly only with total GDS score ($r = -.174, p = .029$); persons who slept less reported more symptoms of depression. Sleep latency correlated with the total GDS ($r = .167, p = .037$); the RBANS ($r = -.241, p = .002$), and the TONI ($r = -.258, p = .001$). For all these measures, a longer time to fall asleep was associated with poorer performance. Sleep efficiency (log transformed) correlated with the total GDS ($r = -.182, p = .022$), the RBANS ($r = .185, p = .020$), the TONI ($r = .197, p = .013$), and $N$-Back ($r = .211, p = .008$), with lower sleep efficiency being associated with poorer performance. Thus, sleep latency and efficiency, two closely linked variables, were more strongly related to those behavioral measures that differed between good and poor sleepers than was the total amount of sleep participants reported. It should be noted, however, that all these correlations were modest in absolute magnitude, accounting for no more than 5% of shared variance between measures.

The impact of potential confounds was examined next. Stroke risk did not differ between the two groups (Table 1), suggesting that the two sleep groups did not differ in their overall risk for cerebrovascular disease. Looking individually at two major risk factors for cerebrovascular disease, 16% of the participants carried a diagnosis of heart disease, whereas 10% carried a diagnosis of diabetes. There were, however, no significant differences between the two sleep groups in prevalence of either disease. Because poor sleepers did show higher GDS scores, it is possible that depression was responsible for the group differences in cognitive performance. We, therefore, carried out an analysis of covariance on the cognitive tasks, covarying for total GDS score (see Table 2). One previously significant finding ($N$-Back) became marginally significant ($p = .062$), whereas the other group differences remained statistically significant. Thus, the greater depressive symptomatology seen in the poor sleepers does not appear responsible for all the cognitive differences between the two groups. To examine the possible contribution of sleeping medications, we examined the question in the PSQI that asks about use of sleeping medications and found greater usage in poor sleepers than in good sleepers (36% vs. 7%, $p < .0001$). We therefore reanalyzed the data using results only from those participants who had not used sleeping pills in the previous month (100 good sleepers and 31 poor sleepers). This did not substantially change the results. The only previously significant result that fell to marginal significance was Trails B ($p = .057$). Thus, differential use of sleep aids does not appear to be a major source of the group differences found in cognition and depressive symptoms. Finally, mean SAA was the same in the two groups, making it unlikely that anticholinergic medication use explains group differences in cognitive performance.

**Discussion**

Recent theories of aging have suggested that one or more fundamental mechanisms may underlie the diverse cognitive decrements found with increasing age. The most commonly proposed mechanisms involve a slowing in the rate of information processing (Salthouse, 1996), a decline in working memory (Park & Hedden, 2001), a decrease in the efficiency of inhibition (Hasher, Tone, Lustig, & Zacks, 2001), or a decline in executive function (Braver & West, 2008). The present results produced no evidence that self-reported sleep difficulties were associated with decrements in speed of information processing because the RTs of good and poor sleepers did not differ in two different tasks requiring decisions. Similarly, the two sleep groups were equally effective in inhibiting prepotent but contextually inappropriate responses in the Stroop and Hayling tasks; thus, inhibitory function did not seem related to sleep quality. Nor was there any evidence of a difference in immediate and delayed verbal memory.

Good and poor sleepers did differ, however, on one of the measures of working memory and on a measure of fluid intelligence as measured by abstract problem solving. There were also group differences on a test requiring shifting of attentional set and on a measure of general neuropsychological status (RBANS). Several of the cognitive tasks on which the two groups differed could be seen as measuring “executive” functions. Executive functioning has been formulated in a variety of ways. Miyake, Friedman, Emerson, Witzki, and Howeter (2000) suggest that three basic executive functions are shifting of attentional set,

| Table 2. Reanalyses Assessing the Potential Confounding Effects of Sleep Medications and Depression on Group Differences in Performance |
|---------------------------------|-----------------|-----------------|
|                                | $N = 108/49$    | $N = 100/31$    | $GDSN = 108/49$ |
| GDS total                      | $.001$          | $.001$          | NA              |
| GDS functional                 | $.001$          | $.001$          | NA              |
| RBANS                          | $.009$          | $.023$          | $.039$          |
| Trails B                       | $.016$          | $.057$          | $.042$          |
| TONI                           | $.001$          | $.001$          | $.002$          |
| N-Back                         | $.010$          | $.019$          | $.062$          |

*Note: SAA = serum anticholinergic activity; GDS = Geriatric Depression Scale; NA = not applicable; RBANS = Repeatable Battery for the Assessment of Neuropsychological Status; TONI = Test of Nonverbal Intelligence.*
updating of working memory, and inhibition of prepotent responses. To this listing, other investigators (e.g., Andres & Van der Linden, 2000) have added planning and reasoning. The present results provide evidence for differences between the good and poor sleepers in set shifting (Trails B), abstract reasoning (TONI), and updating of working memory (N-Back). However, inhibitory function was not different in the two groups.

Although the PSQI is a general measure of sleep quality, the participants’ responses to various questions can be used to estimate sleep duration, difficulty initiating sleep (latency), and sleep efficiency (percent of time spent in bed that the individual was actually asleep). The present study found that sleep latency and efficiency were correlated with cognitive performance, whereas total sleep duration was not. Similar results have been found by Schmutte et al. (2007), Hart et al. (1995), and Blackwell et al. (2006). Thus, the more important aspect of sleep appears to be the amount of unwanted intruding wakefulness experienced, rather than the total number of minutes of sleep the individual obtains. This ties in with behavioral treatments for insomnia which often specifically seek to increase sleep efficiency by reducing time in bed. Sleep restriction is a major component of several behavioral insomnia therapies requiring patients to limit the time spent in bed while awake, thus favoring sleep consolidation (Spielman, Saskin, & Thorpy, 1987), often at the expense of a reduction in the number of minutes of actual sleep.

It should be emphasized that the present sample was in no way a clinical sample. None of the participants volunteered because they were seeking help for a sleep problem. Indeed, the mean PSQI score in the poor sleepers was only 8.4 (median = 8, max = 16) in comparison with individuals diagnosed with primary insomnia where PSQI scores typically fall in the 10+ range.

We examined a number of potential confounds (risk of cerebrovascular disease, use of sleeping pills, and anticholinergic medications) and found no evidence that they accounted for group differences in performance. Poor sleepers did, however, report more symptoms of depression than good sleepers, although total GDS scores were fairly low. Covarying for total depressive symptoms failed to eliminate most of the intergroup differences in cognition, and thus, it does not appear likely that depression was responsible for the differences in cognitive performance found between good and poor sleepers (participants with a GDS ≥15 were excluded).

Further examination of the GDS results suggested that the elevated GDS scores in the poor sleepers did not result from their responses to questions about crying, hopelessness, anxiety, and feeling blue, with positive responses to these questions being infrequent in both groups. Rather, it was questions about functional symptoms that differentiated the two groups with reports of poor concentration, low motivation, and problems making decisions being significantly more common in the poor sleepers. Although such functional symptoms are prominent in depressed individuals, it is possible that in the present group of nondepressed elders, the increased reports of functional symptoms by poor sleepers may accurately reflect concentration and motivation problems they are experiencing due to their chronic sleep problems.

There are a number of limitations to the present results. Sleep was assessed with a general retrospective self-report measure. There was no log of actual sleep behavior or polysomnography. However, the PSQI does usefully distinguish sleep-disordered and non-sleep-disordered individuals and is sensitive to the treatment of sleep disorders (Germain et al., 2006). Also, self-reported sleep problems have previously been associated with cognitive decrements and depressive symptomatology in older participants in several studies (Schmutte et al., 2007; Tworoger, Lee, Schernhammer, & Grodstein, 2006). There was also no measure of sleep-disordered breathing (SDB), a condition associated with substantial cognitive dysfunction (Fulda & Schulz, 2001). However, the relation of SDB to cognitive impairment may be mediated by increased cerebrovascular disease (Culebras, 2004), and the Stroke Risk Profile of the good and poor sleepers in the present study did not differ significantly nor did their mean body mass index (BMI), an important risk factor for SDB (Table 1). The percentage of persons with a BMI greater than 30 (a clinically relevant cutoff for SDB risk) was 31% in good sleepers and 37% in poor sleepers, a nonsignificant difference. Another limitation is that this study was not originally designed to test specific hypotheses about the relation of sleep quality to specific cognitive operations, and thus, it did not necessarily assess the most relevant cognitive abilities or potential confounds. The present analyses linking sleep quality and cognitive measures must, therefore, be viewed as preliminary, as must any conclusions about the underlying cognitive nature of the performance decrements. Finally, a major issue that cannot be resolved by this study is whether the relation found between sleep quality and cognitive performance is causal (i.e., poor sleep leads to cognitive impairment) or whether some common factor is responsible for a conjoint decline in both sleep and cognition. The present results do, however, indicate that, within the limits of the methodology used to measure them, certain potential common causes such as cerebrovascular disease, depression, and use of sleeping pills and anticholinergic medications do not appear to account for the cognitive differences between good and poor sleepers.

Overall, this study demonstrated that reports of poor sleep by healthy older adults are associated with individual differences in cognitive performance. However, this was true only for certain cognitive tasks. The declines in cognitive performance associated with aging have been postulated to arise from a reduction in general-purpose processing resources, commonly conceptualized as a
slowing in the rate of information processing or a limitation in working memory capacity. The present results showed no evidence that good and poor sleepers differed on measures assessing the speed with which subjects could make simple decisions and only limited evidence for a difference on measures thought to measure working memory (the N-Back test but not Letter–Number Sequencing). An alternative to the processing resources hypothesis is that aging produces a decrease in the efficiency with which subjects can inhibit irrelevant information. Again, good and poor sleepers performed equally well on two commonly given measures claimed to measure inhibitory function. A difference was found between good and poor sleepers on tests that required abstract problem solving and set shifting. Such measures may place a greater demand on subjects’ ability to maintain their concentration on a task, a difficulty noted by the subjects in their responses to the GDS, although without specific tasks examining this aspect of attentional function, this remains speculative. Overall, although sleep problems may contribute to variability in cognitive performance between elderly individuals, their effect appears restricted to certain cognitive domains.

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