


Do all individuals with sleep apnea suffer from daytime sleepiness? A preliminary investigation

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Abstract

We derived descriptive characteristics related to habitual sleep duration and insomnia for individuals newly diagnosed with sleep apnea/hypopnea syndrome and evaluated how sleep apnea/hypopnea syndrome, insomnia, depression, and sleep duration relate to sleepiness and fatigue. In total, 100 participants were divided into three sleep groups: short (<7 hours), long (≥8 hours), and midrange (7–7.9 hours). Polysomnography, insomnia, sleepiness, fatigue, depression, and gender were assessed. Half of the participants were short sleepers. They were more likely to have insomnia than midrange or long sleepers and they were more likely to be sleepy than midrange or long sleepers, regardless of insomnia.

Keywords

age, behavioral medicine, sleepiness, sleep apnea, depression, health psychology, physical symptoms, respiratory problems, risk factors

Introduction

Sleep apnea/hypopnea syndrome, daytime sleepiness, and driving safety

Numerous studies have indicated that individuals with sleep apnea/hypopnea syndrome (SAHS) experience problematic daytime sleepiness. SAHS is a nocturnal breathing disorder that is characterized by episodes of breathing cessation during sleep. Typically diagnosed by overnight polysomnography (PSG), where individuals sleep in a sleep laboratory connected to a variety of sensors, it has both medical and behavioral implications beyond disrupted sleep. Severity of SAHS is often determined by the number of nocturnal arousals per hour and by the degree of nocturnal oxygen desaturation.

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For example, because it is often associated with daytime sleepiness, SAHS has been identified as a prevalent and potentially dangerous condition among both commercial and non-commercial motor vehicle drivers (George, 2007; Hakkanen and Summala, 2000; Howard et al., 2004; Pack et al., 2006; Rodenstein et al., 2008). Individuals with SAHS have commonly been used as the prototype example of how impaired daytime performance caused by sleepiness is manifested in increased vehicle crash risk (Pack et al., 2006). However, it is known that a high percentage of individuals with SAHS are not demonstrably sleepy during the day (Bailes et al., 2011). When actual driving records of patients with SAHS are examined, it has been found that the majority of patients with SAHS have not had a motor vehicle crash in the last 5 years, and the directives for physicians to remove driving privileges because of SAHS vary widely by region (Mayer et al., 2010).

Measurement of sleepiness

Many self-report measures confound the concepts of sleepiness and fatigue (Bailes et al., 2006). It is not yet clear whether “feeling sleepy” can be reliably distinguished from “feeling fatigued,” nor is it clear whether fatigue is linked to driving safety concerns.

Insomnia and daytime sleepiness

Insomnia is highly prevalent in SAHS. For example, Vozoris (2012) recently found that 43 percent of individuals with SAHS had insomnia, compared to 30 percent of individuals without SAHS. A recent review (Fichten and Libman, 2011) demonstrated conclusively that “mid-range” sleepers (typically defined as sleeping somewhere between 7 and 7.9 hours (National Sleep Foundation, 2009) not only live longer but also have fewer comorbidities than both shorter and longer sleepers (Grandner and Drummond, 2007). As for insomnia, it has long been known that patients with insomnia “commonly view themselves as sleepy, fatigued, cognitively impaired, and generally not as ‘mentally sharp’ as they recall themselves to be prior to the onset

of their sleep difficulties” (Edinger et al., 2008). However, the literature has typically shown few objective differences in performance between those with and without insomnia (Bonnet, 2005), unless tasks are carefully crafted (Edinger et al., 2008). Paradoxically, it was also found that individuals with insomnia were less sleepy when given a nap opportunity than controls. Moreover, it has long been known that the key self-reported consequence of insomnia is not sleepiness but fatigue (Chambers and Keller, 1993; Chambers and Kim, 1993; Roth and Roehrs, 2003). In addition, insomnia is closely related to depression, with each condition being a predictor for the other (Gaynes et al., 2005; Goyal et al., 2007). This raises the question as to whether the sleep duration/insomnia/depression constellation relates in some manner to daytime sleepiness or fatigue.

Sleepiness and fatigue in SAHS

We recently carried out a study designed to explore the role and implications of daytime sleepiness and fatigue as distinct constructs in a sample of individuals with SAHS (Bailes et al., 2006, 2008). The data show not only that many individuals with SAHS are not sleepy but also, as shown by others (Hossain et al., 2005), that fatigue is another very common symptom associated with SAHS (Bailes et al., 2011). Moreover, our study identified four subgroups among individuals with SAHS characterized by combinations of high and low levels of daytime fatigue and daytime sleepiness. Of particular interest are the substantial numbers of individuals with relatively low daytime sleepiness and fatigue scores—below clinical cutoffs—who, despite an unmistakable SAHS diagnosis, appeared not to complain of diminished functioning or quality of life and to be similar to individuals in a healthy comparison group.

Perceived driver risk among individuals with SAHS

The data suggest that individuals with SAHS are not all alike—that there are important differences among them on daytime sleepiness and fatigue.

Although one can demonstrate clear sleep fragmentation with SAHS, a substantial segment of people with this disorder do not manifest impaired daytime functioning. This suggests that the characteristically impaired sleep quality in people with SAHS does not necessarily lead to daytime sleepiness and fatigue. There are some indications in the literature that there may be other physiological processes underlying sleepiness and fatigue in SAHS. For example, there are data to suggest that nocturnal basal oxygen saturation (BSpO₂) may play a role in influencing fatigue levels (Bailes et al., 2011). There are also suggestions that low, sub-clinical levels of depression are implicated in the daytime sleepiness/fatigue experienced in SAHS (Bardwell et al., 1999). There are inconsistent reports in the literature that insomnia may play a role in motor vehicle driving risk (Philip and Akerstedt, 2006). Given these diverse findings, as well as evidence that insomnia and depression are often part of the sleep deprivation/fragmentation constellation, it is important to examine individual differences associated with sleepiness and fatigue to provide a preliminary conceptual basis for perceived driver risk in the SAHS population.

The present study

In view of the relationship between SAHS severity, sleep fragmentation, depressive features, insomnia, and sleep duration, the present study was designed to (1) provide a descriptive profile of a sample of individuals newly diagnosed with SAHS who have not yet received treatment and (2) determine how PSG findings, insomnia, depression, and sleep duration relate to sleepiness and fatigue in individuals with SAHS. We hypothesized that (1) short sleep duration as well as the respiratory disturbance index (RDI) would be related to daytime sleepiness and (2) that insomnia, depression, and low nocturnal BSpO₂ would be related to daytime fatigue. (3) We also predicted that variables which predict sleepiness would differ from those which predict fatigue. Based on the findings, we discuss how distinctive patterns of variables relate to sleepiness and fatigue and possible related driving risk.

Method

Participants

Participants consisted of 100 consecutive individuals (45 females, 55 males; mean age = 61.74 years, standard deviation (SD) = 11.56 years, median = 61.00 years, range = 35–85 years) diagnosed with SAHS at a sleep laboratory before they commenced treatment. They were recruited from primary care and other waiting rooms for a research study on sleep, sleepiness, and fatigue, which involved an overnight stay at a sleep laboratory for PSG. Individuals were encouraged to participate whether or not they had these symptoms. Individuals were excluded on the basis of the presence of another medical illness in which sleepiness/fatigue was an important symptom or taking medication with major sleepiness/fatigue side effects (e.g. sedating antidepressants, anxiolytics, antipsychotics). A comprehensive evaluation was conducted first through interview and questionnaire by the research team and then through medical and overnight PSG assessment by our team respirologist. Those who were subsequently diagnosed with SAHS were selected for this study.

Procedure

The research ethics committees of both the Jewish General Hospital and the Mount Sinai Hospital of Montreal approved the research protocol. Participants signed a consent form advising them of all aspects of the study, including the right to withdraw at any time. All were paid a small honorarium for their participation and were refunded travel and parking expenses.

Participants were screened with a structured sleep history interview for comorbid diagnoses and excluded if they (1) suffered from a current major psychiatric illness; (2) had another known medical condition related to fatigue, sleepiness, or insomnia; or (3) if they were working rotating/split shifts or recently traveled across time zones. Inclusion criteria were being a community resident and having sufficient cognitive and language skills to complete the measures in English or French.

Following the initial screening, all the participants completed questionnaires assessing aspects of their nocturnal and daytime functioning. Subsequently, our sleep specialist team member conducted a general medical and sleep disorders assessment including a body mass index (BMI) evaluation. Participants then underwent a single night of overnight PSG in a sleep laboratory.

The diagnosis of SAHS was carried out by a certified respirologist. This was based on the PSG findings and was made in accordance with the *International Classification of Sleep Disorders-2* (American Academy of Sleep Medicine, 2005) and the American Sleep Disorders Association (American Sleep Disorders Association, 1999; American Sleep Disorders Association (ASDA)-Diagnostic Classification Steering Committee, 2005). All participants were provided information about the results of their assessment. Those receiving a sleep disorder diagnosis were offered appropriate treatment and/or referral to a sleep specialist.

Questionnaire responses were used to divide participants into three sleep duration groups (Short: <7 hour/night; Midrange: 7–7.9 hour/night; Long: \geq 8 hour/night). This was also used to divide participants into two Insomnia groups: those with and those without an Insomnia diagnosis. Diagnosis was based on participants' complaint of an insomnia problem coupled with the following sleep parameters: at least 31 minutes of undesired awake time at least 3 times per week, with problem duration at least 1 month. There were 28 individuals with a "mixed" insomnia profile (i.e. complained of insomnia but sleep parameters did not meet the selection criteria or vice versa). They were excluded from the study.

Measures

The BMI was used as an indicator of body fat. A BMI between 18.5 and 24.9 indicates normal weight, a BMI between 25 and 29.9 indicates overweight, and a BMI greater or equal to 30 kg/m² is considered to be obese. PSG assessment was carried out in a supervised sleep laboratory from 10 p.m. to 7 a.m. Monitoring included three leads electroencephalography

(EEG), electrooculography (EOG), bilateral anterior tibialis and chin electromyography (EMG), electrocardiography (ECG), pulse oximetry, nasal and oral airflow with nasal pressure cannulae and thermistor, and inductance plethysmography for measurement of respiratory effort (American Thoracic Society, 1989). All signals were acquired on a digital data management system (Sandman, Ottawa, ON, Canada). A certified PSG technologist with at least 10 years of experience manually scored the studies blind to the results of symptom assessments. Sleep stages were first scored in 30-second epochs according to standard criteria (Rechtschaffen and Kales, 1968). Next, EEG arousals were scored according to standard current consensus criteria (American Sleep Disorders Association, 1992). An apnea event was scored when there was a cessation of breathing for 10 or more seconds. An hypopnea was defined a priori as an event lasting at least 10 seconds with a decrease of >50 percent from a baseline in the amplitude compared to the mean of the largest three breaths over the previous four epochs or a lesser reduction in airflow signal amplitude accompanied by either at least a 3 percent desaturation or an EEG arousal (American Academy of Sleep Medicine Task Force (AASM), 1999). Apneas and hypopneas were summed and then divided by the total sleep time to calculate the RDI. The SAHS severity cutoffs in our laboratory, as in those of others (Young et al., 2002), were mild (RDI 5–14.9 events/hour), moderate (RDI = 15–29.9 events/hour), and severe (RDI \geq 30 events/hour). Leg movements, apnea events, and associated arousals were scored manually according to scoring rules established by the Atlas Task Force of the American Sleep Disorders Association (1993).

A background information form provided data on age, sex, and demographic variables. Retrospective information from the Sleep Questionnaire (Libman et al., 2000) allowed us to diagnose insomnia and to determine habitual sleep duration. The Empirical Sleepiness and Fatigue Scales (Bailes et al., 2006) measured sleepiness and fatigue without confounding the two concepts. The Epworth Sleepiness Scale (Johns, 1991) was

included because it is familiar to most as a self-report of sleep tendency. The Primary Care (PC) Subscale of the *Beck Depression Inventory Manual* (2nd edn; *BDI-II*; Beck et al., 1996) was used to evaluate the affective and cognitive symptoms of depression independent of fatigue, sleepiness, insomnia, and agitation.

Data analyses

Fifty-one of the participants were habitual Short Sleepers (<7 hours), 25 were habitual Long sleepers (≥ 8 hours of nocturnal sleep time), and 24 were Midrange Sleepers (7–7.9 hours) (see Fichten and Libman, 2011 for a review). There was no significant difference in sleep duration between males and females, $\chi^2(2, 100) = 0.199$, $p = .905$. Therefore, in subsequent analyses, data from males and females are combined. It should be noted that the correlation between age and total sleep time for the entire sample was not significant, $r(2, 98) = -.05$, $p = .597$.

To examine the relationship between sleep duration and insomnia, on the one hand, and age, daytime sleepiness, daytime fatigue, and PSG findings, on the other, a series of analysis of variance (ANOVA) comparisons were made, with sleep duration and insomnia as the independent variables and sleepiness, fatigue, and two PSG scores (BSpO₂ during sleep and the respiratory distress index (RDI)) as dependent variables.

Our preferred measure of sleepiness is the Empirical Sleepiness Scale (Bailes et al., 2006). Made up of selected items from the Epworth Sleepiness Scale (Johns, 1991), the Empirical Sleepiness Scale more readily distinguishes between fatigue and sleepiness than the Epworth Sleepiness Scale. Nevertheless, because so many readers are familiar with the meaning of the Epworth Sleepiness Scale scores, we provide means and SDs for this measure, although we carried out the bulk of the analyses using the Empirical Sleepiness Scale. It should also be noted that the correlation for this sample between the two measures of sleepiness was very high, $r(2, 85) = .98$, $p < .001$. To examine

predictors of sleepiness and of fatigue in individuals with SAHS, two regression analyses were carried out with age, sex, BMI, depression, insomnia diagnosis, total sleep time, depression, and two PSG findings (BSpO₂ and RDI) as the predictor variables.

Results

Sample characteristics

Demographic aspects of the sample show that the sample was reasonably well educated (mean = 15 years, SD = 5 years) and that slightly less than half were employed and slightly more than half were currently married or living with someone. Approximately three-fourth of the sample had moderate to severe obstructive sleep apnea (OSA) (i.e. RDI ≥ 15). Forty-seven participants had Insomnia. The rest neither complained of Insomnia nor met the quantitative sleep parameter criteria. However, those with Insomnia are overrepresented in the Short Sleeper category (32 of 47), while those with No Insomnia are overrepresented in both the Midrange and Long Sleeper groups (17 of 53 in each group), $\chi^2(2, 100) = 10.40$, $p = .006$.

Sleep duration and insomnia

To examine characteristics of Short, Midrange and Long Sleepers with and without Insomnia, we performed a series of ANOVA comparisons (2 Insomnia (yes/no) \times 3 Sleep Duration (Short/Midrange/Long)). Dependent variables were Sleepiness, Fatigue, RDI, and BSpO₂. Means, SDs, and test results are available in Table 1, which shows that there were no significant main effects or interactions for either Fatigue or RDI. Significant main effects for Sleepiness (on both the Empirical Sleepiness Scale (Bailes et al., 2006) and the Epworth Sleepiness Scale (Johns, 1991)), detailed in Table 1, and post hoc testing show that Short Sleepers, both with and without Insomnia, were more Sleepy than those in the other two groups. Table 1 also shows that there were medium to large effect sizes

Table 1. Sleepiness, fatigue, PSG findings, age, depression, and BMI for participants with different sleep durations, who do and who do not have insomnia.

Variables	Insomnia				Effect size, Cohen's <i>d</i>				Test results						
	Yes		No		Insomnia		Yes		No		Effect	df	F	p	Post hoc test
	M	SD	M	SD	Yes	No	Yes	No							
Empirical Sleepiness Scale^a															
Short sleep duration	8.47	4.73	10.11	4.57	↕0.48	↕0.43	↕↕	↕↕	↕↕	Sleep duration	2,94	8.26	.000	Short > medium, long	
Medium sleep duration	6.00	5.45	4.71	2.73	↕0.48	↕1.43	↕↕	↕↕	↕↕	Insomnia	1,94	0.00	.983	See Figure 1	
Long sleep duration	5.75	3.73	5.47	4.77	↕0.64	↕0.85	↕↕	↕↕	↕↕	Interaction	2,94	0.89	.414		
Epworth Sleepiness Scale^a															
Short sleep duration	10.16	5.61	12.38	4.50	↕↕	↕↕	↕↕	↕↕	↕↕	Sleep duration	2,79	5.13	.000	Short > medium, long	
Medium sleep duration	7.50	6.80	7.21	3.35	↕0.43	↕1.23	↕↕	↕↕	↕↕	Insomnia	1,79	0.06	.816		
Long sleep duration	8.25	4.71	7.19	5.53	↕0.37	↕0.82	↕↕	↕↕	↕↕	Interaction	2,79	0.83	.441		
Empirical Fatigue Scale^a															
Short sleep duration	10.06	4.38	11.42	3.61						Sleep duration	2,94	1.79	.173		
Medium sleep duration	10.14	2.41	7.53	3.79						Insomnia	1,94	1.04	.309		
Long sleep duration	11.63	2.72	10.06	4.88						Interaction	2,94	2.09	.129		
BSpO₂^b															
Short sleep duration	95.99	1.89	95.96	2.98						Sleep duration	2,94	1.89	.157		
Medium sleep duration	95.61	1.68	96.06	1.99						Insomnia	1,94	4.74	.032		
Long sleep duration	93.14	5.06	96.39	1.53						Interaction	2,94	3.43	.037		
RDI^a															
Short sleep duration	25.91	18.05	28.54	20.48						Sleep duration	2,94	0.96	.387		
Medium sleep duration	42.80	38.83	25.86	14.80						Insomnia	1,94	1.15	.287		
Long sleep duration	33.88	18.62	32.05	28.33						Interaction	2,94	1.39	.253		
Age															
Short sleep duration	62.88	11.13	59.68	11.76						Sleep duration	2,94	0.32	.730		
Medium sleep duration	68.57	15.80	58.65	11.15						Insomnia	1,94	1.38	.244		
Long sleep duration	59.38	9.75	63.29	10.67						Interaction	2,94	1.91	.154		

(Continued)

Table 1. (Continued)

Variables	Insomnia		Effect size, Cohen's <i>d</i>		Test results				
			Insomnia		Effect	df	F	p	Post hoc test
	Yes	No	Yes	No					
M	SD	M	SD	Yes	No				
BMI									
Short sleep duration	29.72	6.19	28.65	6.28			1.07	.348	
Medium sleep duration	28.22	7.02	30.21	7.28			1.55	.216	
Long sleep duration	29.29	8.49	28.97	5.70			2.81	.065	
Depression									
Short sleep duration	3.59	3.98	3.47	3.92	Sleep duration	2.94	0.83	.441	
Medium sleep duration	3.00	3.06	1.65	2.69	Insomnia	1.94	1.12	.292	
Long sleep duration	3.63	4.72	2.53	2.24	Interaction	2.94	0.27	.761	

PSG: polysomnography; SD: standard deviation; df: degree of freedom; BSpO₂: basal oxygen saturation during sleep; RDI: respiratory distress index; BMI: body mass index.

^aHigher means indicate worse functioning.

^bHigher means indicate better functioning.

^cAfter a Bonferroni correction to the alpha level, only those comparisons with *p* = .000 remain significant.

(Cohen's *d*) for these differences. The significant findings, based on the large number of Short Sleepers, are best seen in Figure 1, which also shows that Insomnia was not related to Sleepiness in either Midrange or Long Sleepers. Notably, in spite of the very large age range in the sample, there was no significant Sleep Duration or Insomnia main effect or interaction for Age. Similarly, there were no significant findings for Depression or the BMI.

Predicting sleepiness and fatigue

Stepwise regression analyses were performed to identify predictors of Sleepiness (Empirical Sleepiness Scale) and of Fatigue (Empirical Fatigue Scale). Age, Sex, Depression (BDI-II-PC), Sleep Duration (total sleep time), Insomnia, BMI, BSpO₂ and the RDI were entered as predictor variables. Results show that the best predictor of Sleepiness was shorter Sleep Duration, with younger Age adding

significantly to the prediction. These variables, although significant, $F(2, 97) = 6.33, p < .005$, accounted for a relatively modest proportion of the variance (11%) in Sleepiness. It should be noted that higher BMI was also an important univariate predictor, $r(2, 98) = .19, p = .057$. All other variables entered in the equation were not significantly correlated to Sleepiness.

To evaluate an estimate for the cutoff for age, we compared the ages of participants who were Sleepy and those who were Not Sleepy. Because there are no other typically used cut-offs in the literature, we used Epworth Sleepiness Scale (Johns, 1991) scores to do this (Not Sleepy ≤ 10 , Sleepy > 10 ; Barbe et al., 2001). The significant *t* test shows that the 41 Sleepy participants were, on average, 6 years younger (mean age = 60.24 years, SD = 10.77 years) than the 44 participants who were Not Sleepy (mean age = 66.52 years, SD = 9.89 years), $t(83) = 2.80, p = .007$. Graphing the relationship between Age and Sleepiness, however,

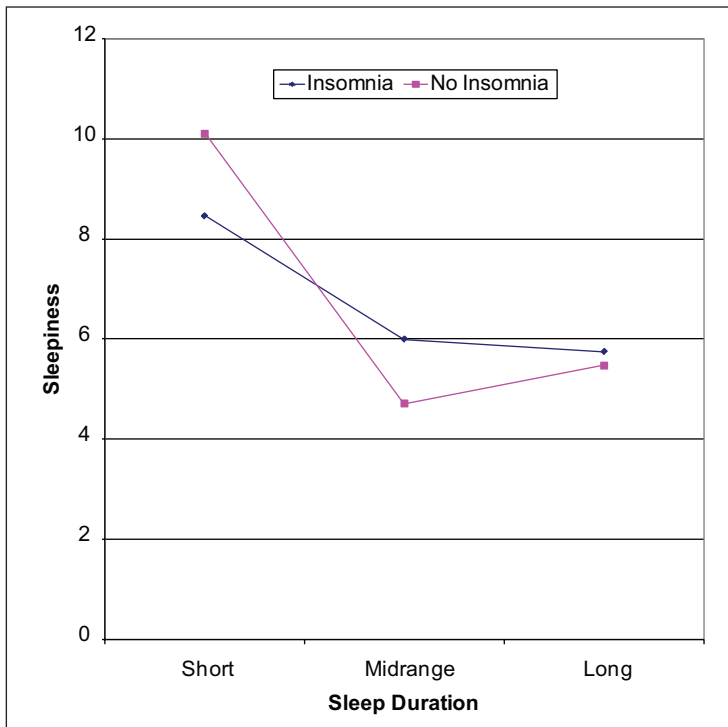


Figure 1. Sleepiness in Short, Midrange and Long Sleepers.

shows that it is not possible to specify an appropriate age cutoff. The regression to predict Fatigue shows that the best model was composed of lower nocturnal BSpO₂ and higher Depression (BDI-II-PCI). Again, although significant, $F(2, 97) = 8.15, p < .001$, the predictors did not explain a large proportion of the variance (14%) in Fatigue. All other variables entered in the equation were not significantly correlated to Fatigue.

Discussion

Our findings indicate that, contrary to popular belief, it was the “younger” individuals with SAHS and those who habitually slept less than 7 hours per night, regardless of whether they had insomnia or not, who were most likely to be sleepy during the day. Sex, RDI, nocturnal BSpO₂, and daytime fatigue were unrelated to sleepiness. That there is no clear relationship between SAHS severity and daytime sleepiness has also been shown by others (Kaminska et al., 2010; Sauter et al., 2000) as has the linkage between short sleep duration in apnea samples and daytime sleepiness (Pack et al., 2006).

In the context of other studies examining sleep and sex differences, some studies have reported that men sleep longer than women (Faubel et al., 2009; Habte-Gabr et al., 1991; Ohayon, 2004), others have reported that women sleep more than men (Kronholm et al., 2006; Natale et al., 2009), while still others reported no sex difference in sleep duration (Bliwise et al., 1994). Our sample of individuals newly diagnosed with SAHS showed no significant differences in sleep duration between males and females.

In contrast to population data, which indicate that the majority of community-based individuals fall into the midrange sleep duration category, while 28 percent sleep 8 hours or longer per night (National Sleep Foundation, 2009), it is noteworthy that half of our participants with newly diagnosed SAHS were short sleepers. This finding, which has not, to the

best of our knowledge, been reported in the literature, needs replication since it may reflect the recruitment process or the age range of our sample.

Some studies in the literature have reported no difference in the prevalence of either insomnia or sleepiness between long and midrange sleepers (Bliwise et al., 1994). Our findings on individuals with SAHS are consistent with this. However, most community-based studies have suggested greater sleep fragmentation and more daytime sleepiness among long sleepers, especially long sleepers with insomnia, relative to midrange sleepers (Fichten and Libman, 2011; Grandner and Kripke, 2004; Kripke et al., 2001, 2002), as well as a higher incidence of insomnia and nonrefreshing sleep (Grandner and Kripke, 2004). In the present study, it was the short sleepers with SAHS who were more likely to have insomnia and who were more likely to be sleepy, whether they had insomnia or not. Whether this is unique to individuals with untreated SAHS is a question that requires further study.

In the present investigation, although explaining only a small portion of the variance in scores, sleepiness was best predicted by shorter sleep duration, followed by younger age. The age variable was counterintuitive, as many studies have found increasing sleepiness with increasing age (Gander et al., 2005). Yet, some studies have also shown that excessive sleepiness was related to younger age (Bixler et al., 2005). Whether the finding of lower sleepiness with age in our sample is a result of our sampling or age distribution or whether it is related to SAHS is an empirical question.

Fatigue, on the other hand, was best predicted by low nocturnal BSpO₂ and depression, even when only the cognitive–affective aspects of depression (e.g. feeling sad, discouraged) were evaluated, without the somatic symptoms (e.g. fatigue, lack of energy). This suggests that fatigue and sleepiness are conceptually separate conditions and that the role of fatigue, independent of sleepiness, needs to be further studied in individuals with SAHS.

Some limitations of the present study include the following. The BMI of participants was somewhat lower than that typically reported in the literature. Although sample size was relatively large, there were few midrange and long sleeper participants who had insomnia. Also, the regression analyses predicted only relatively small portions of variability in sleepiness and fatigue scores.

Driver risk profile for individuals with SAHS

We know from previous studies that approximately 20 percent of individuals with SAHS have few daytime fatigue or sleepiness complaints (Bailes et al., 2011). Indeed, on a variety of psychological and daytime functioning variables, these individuals were similar to a healthy comparison group. In the present study, we found that it was shorter sleep duration (i.e. less than 7 hours of nocturnal sleep), coupled with relatively younger age for an SAHS sample (i.e. 60 years and younger), that appeared to be associated with increased daytime sleepiness in individuals with SAHS. Those reporting midrange (7–7.9 hours) and long (i.e. ≥ 8 hours) sleep duration had significantly lower sleepiness scores. Since only 50 percent of the sample fell into the short sleeper category, and since it was mainly the “younger” individuals who manifested significant daytime sleepiness, our findings clearly imply that daytime sleepiness, as an analogue of driver risk, is confined to a limited segment of the SAHS population. This suggests that instead of classifying an individual with SAHS as an automatic driver risk, one should first take into account age and whether he or she sleeps less than 7 hours per night. These variables can then be situated in the previously demonstrated sleepiness/fatigue risk profile in individuals with SAHS—a profile that might imply driver risk. Nevertheless, we need to reiterate that our results show that age and sleep duration together explain only a modest proportion of the variance in daytime sleepiness and suggest that further research is needed

to identify additional predictors of sleepiness in individuals with SAHS. Although driver sleepiness has been related to driving risk, there is very little known about the independent contribution of driver fatigue to the risk profile. Therefore, the importance of fatigue levels in driving safety of individuals with SAHS remains speculative until the appropriate future studies are done.

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