ORIGINAL ARTICLE

Mortality Associated With Sleep Duration and Insomnia

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Background: Patients often complain about insufficient sleep or chronic insomnia in the belief that they need 8 hours of sleep. Treatment strategies may be guided by what sleep durations predict optimal survival and whether insomnia might signal mortality risks.

Methods: In 1982, the Cancer Prevention Study II of the American Cancer Society asked participants about their sleep duration and frequency of insomnia. Cox proportional hazards survival models were computed to determine whether sleep duration or frequency of insomnia was associated with excess mortality up to 1988, controlling simultaneously for demographics, habits, health factors, and use of various medications.

Results: Participants were more than 1.1 million men and women from 30 to 102 years of age. The best survival was found among those who slept 7 hours per night. Participants who reported sleeping 8 hours or more experienced significantly increased mortality hazard, as did those who slept 6 hours or less. The increased risk exceeded 15% for those reporting more than 8.5 hours sleep or less than 3.5 or 4.5 hours. In contrast, reports of "insomnia" were not associated with excess mortality hazard. As previously described, prescription sleeping pill use was associated with significantly increased mortality after control for reported sleep durations and insomnia.

Conclusions: Patients can be reassured that short sleep and insomnia seem associated with little risk distinct from comorbidities. Slight risks associated with 8 or more hours of sleep and sleeping pill use need further study. Causality is unproven.

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MANY PATIENTS complain to physicians about insufficient sleep and chronic insomnia. Often they request medications for sleep year after year. There has been little evidence-based guidance for the physician indicating when treatment to increase sleep is advisable.[1]

A patient who sleeps 6 to 7 hours may be concerned that she or he is not sleeping long enough, not realizing that 6 to 7 hours is currently the population average.[2,3] Many people believe that 8 hours of sleep is required for health, but there is little medical basis to recommend sleeping 8 hours or more. For example, a classic study found that long sleepers reported less energy and had more psychopathology than did short sleepers.[4]

Insomnia is not synonymous with short sleep. Patients commonly complain of insomnia when their sleep durations are well within the range of those people without sleep symptoms.[5,6] A patient may be concerned by a 20-minute latency to fall asleep, or by awakenings during the night, or by early awakening, when many people with the same sleep latencies and awakenings consider their sleep perfectly satisfactory. Sometimes, such complaints arise from misinformation about what sleep pattern is normal for a person's age. At other times, sleep complaints may reflect a negative self-view and the somatic concerns arising from depression. Insomnia complaints are common symptoms of depression and a large variety of other emotional and medical comorbidities that may not entail particularly short sleep.[1,7-11] Indeed, in the presence of sleep complaints, physicians make a diagnosis of depression more often than a diagnosis of insomnia.[12]

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Epidemiology can inform us what sleep patterns are associated with the lowest mortality risk. In 1959 to 1960, the Cancer Prevention Study I (CPSI) gave health ques-

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SUBJECTS AND METHODS

PARTICIPANTS AND PROCEDURES

Data collection methods of CPSII have been described previously by the American Cancer Society,[24] and many results of CPSII have been published.[25-29] The current analyses were approved by the institutional review board of the University of California, San Diego. Briefly, data were examined from more than 1.1 million participants, mainly friends and relatives of American Cancer Society volunteers, who were a diverse selection of American adults ranging from 30 to 102 years of age. Participants completed health questionnaires in the fall of 1982. The survival or date of death (from death certificates) was ascertained 6 years later for more than 98% of the sample. Because recruitment avoided the institutionalized as well as the most mobile individuals, sample mortality was about 20% lower than for the US population of the same age, but major causes of death resembled the distribution for the population. Data tapes were kindly lent by the American Cancer Society.

Responses to the question, "On the average, how many hours do you sleep each night?" were coded in categories from 2 to 9 hours. Fractional-hour responses were coded as rounded integers, ie, 8 hours represented responses from 7.5 to 8.4 hours. All responses from 9.5 to 16.5 hours were combined in a final category. There were too few responses of 2 hours for analysis. Reports of sleeping less than 2 hours or more than 16 hours each night were considered invalid (<0.1% of responses and coded missing). Responses were missing or invalid for 1.4% of men and 1.7% of women. Responses to the question, "On the average, how many times a month do you have insomnia?" were compressed into categories of 0, 1, 2, 3, 4 to 9, and 10 or more times per month. Participants reported past-month use of "prescription sleeping pills,"[30] which probably included a mixture of different classes of medications.

STATISTICAL ANALYSES

To explore whether sleep durations predicted mortality, Cox proportional hazards survival models[31,32] (Cox models) were computed for 636 095 women and 480 841 men, considering the sexes as separate replicates. A total of 32 covariates were entered simultaneously into the models, including sleep duration, insomnia frequency, and variables reflecting demographic risk factors, habits, health, and medication use, selected by preliminary identification of variables substantially predictive of mortality risk (**Table**). The Cox models estimated hazard ratios for each covariate, which indicated the extent to which a covariate was associated with increased mortality as compared with a reference. Hazard ratios for each reported sleep duration were referenced to a hazard ratio of 1.0, which was assigned to the duration of 7 hours. This arbitrary reference duration had been selected on the basis of CPSI results and pilot analyses of CPSII data, suggesting that it would be the minimum. For example, a hazard ratio of 1.12 for a group sleeping 8 hours would indicate that those sleeping 8 hours were 12% more likely to die within the 6-year follow-up than those sleeping 7 hours, other factors being equal. Similarly, hazard ratios for each reported frequency of insomnia were compared with a hazard ratio of 1.0 arbitrarily assigned to the reference of never having insomnia. The 95% confidence intervals for the hazard ratios associated with each reported sleep duration and each reported frequency of insomnia were then estimated from the multivariate model. As a $P<.05$ significance criterion was selected, hazard ratios were considered significant when a ratio of 1.0 was not included within these 95% confidence intervals. Unlike the analyses of prescription sleeping pill hazards reported previously,[30] all ages were considered together in these models. Orthogonal linear, quadratic, and cubic terms were included for age and also for estimated dietary fat and fiber. Categorical variables were entered as several distinct levels. Model selection by backward elimination was used to exclude from the models covariates with $P>.10$, but almost all variables were retained. Inspection of cumulative hazard functions based on Kaplan-Meier survival curves indicated that the proportional hazards assumptions were acceptable.

Similar Cox models were computed for 9 specific causes of death and for all other causes. In addition, to examine interactions among sleep variables, simplified Cox models were computed with SPSS10.0 (SPSS Inc, Chicago, Ill), first including sleep duration, insomnia, sleeping pill use, and age in the models, and then recomputing the models, removing either sleep duration, insomnia, or sleeping pill use, one at a time.

tionnaires to more than 1 million adult Americans who were followed up prospectively for 6 years. The lowest mortality was experienced by women and men who reported sleeping 7 hours.[6,13] More excess mortality was associated with sleep durations of 8 hours or more than with sleep of less than 7 hours. Excess mortality associated with long sleep has also been observed in smaller prospective studies.[14-23] The CPSI results indicated little if any association of "insomnia" with mortality. In contrast, reported "sleeping pill" use "often" was associated with statistically robust increased mortality risk after control for insomnia.[6]

A more recent study, the Cancer Prevention Study II (CPSII) of the American Cancer Society, offered an opportunity to consider whether sleep durations, insomnia, or sleeping pill use predicts mortality after more extensive control for various sources of comorbidity.

RESULTS

The mean (SD) age for women was 57 (11) years, and for men, 58 (10) years at the time when initial questionnaires were completed. Of the more than 98% for whom follow-up was available, 9.4% of the men and 5.1% of the women had died within the 6 years of follow-up. For women and men, respectively, the causes of death were ischemic heart disease in 24% and 33%, other heart disease in 9% and 9%, cerebrovascular accidents in 10% and 7%, breast cancer in 10% and 0%, colon cancer in 4% and 3%, other cancers in 22% and 26%, accidents in 2% and 2%, suicide in 1% and 1%, homicide in 0.2% and 0.2%, and other causes in 18% and 18%.

The modal reported sleep duration was 8 hours among both women and men. Almost half the sample reported a sleep duration of 7.5 hours or more. Almost half

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Covariate-Adjusted* Mortality Hazard Ratios From the Cancer Prevention Study II (1982-1988)

	Women (n = 636 095)			Men (n = 480 841)		
	% of Women	Hazard Ratio	95% Confidence Interval	% of Men	Hazard Ratio	95% Confidence Interval
Hours of sleep						
3	0.1	1.33	1.08-1.64	0.1	1.19†	0.96-1.47
4	0.7	1.11	1.01-1.22	0.6	1.17	1.06-1.28
5	3.5	1.07	1.01-1.13	2.9	1.11	1.05-1.18
6	15.9	1.07	1.03-1.11	15.5	1.08	1.04-1.11
7	31.8	1.00	Reference	33.8	1.00	Reference
8	38.8	1.13	1.09-1.16	38.0	1.12	1.09-1.15
9	6.0	1.23	1.17-1.28	5.7	1.17	1.13-1.21
≥10	1.5	1.41	1.34-1.50	2.0	1.34	1.28-1.40
Missing	1.7	1.07	1.01-1.14	1.4	1.08	1.01-1.16
Insomnia						
None	49.4	1.00	Reference	70.4	1.00	Reference
1/mo	7.0	0.81	0.76-0.85	5.8	0.87	0.83-0.91
2/mo	10.4	0.87	0.83-0.90	6.5	0.90	0.86-0.93
3/mo	6.4	0.82	0.78-0.86	3.3	0.91	0.86-0.96
4-9/mo	12.3	0.86	0.83-0.89	5.9	0.94	0.91-0.98
≥10/mo	4.3	0.87	0.82-0.91	2.6	0.90	0.85-0.95
Missing	10.2	0.88	0.85-0.91	5.5	0.96	0.93-1.00
Sleeping pills						
None	46.9	1.00	Reference	48.6	1.00	Reference
1-29/mo	3.4	1.10	1.03-1.17	2.3	1.15	1.08-1.22
≥30/mo	0.5	1.24	1.12-1.38	0.4	1.25	1.14-1.38
Missing	49.2	1.07	1.01-1.14	48.7	1.08	1.01-1.16

*Covariates for Cox models were as follows: demographic risk factors: age, race education, occupation, marital status. Habits: exercise level, smoking at intake, years of smoking, churchgoing, fat in diet, fiber in diet; sleep: reported sleep duration, insomnia frequency; health: "sick now," "upset," body mass index, leg pain, history of heart disease, history of hypertension, history of cancer, history of diabetes, history of stroke, history of bronchitis, history of emphysema, history of kidney disease; and medications: "prescription sleeping pills," Valium [diazepam], Librium [chlordiazepoxide hydrochloride], "blood pressure pills," diuretics, Tylenol [acetaminophen], Tagamet [cimetidine].

†Forced into model for completeness, with $P<.11$.

of the women and more than 70% of men reported that they never had insomnia (Table). Only 4.3% of women and 2.6% of men reported insomnia 10 or more times per month. As might be expected, more frequent insomnia and more frequent sleeping pill use were reported among those sleeping less than 7 hours, but there was also a slightly greater frequency of reported insomnia and sleeping pill use among those reporting more than 8 hours sleep (**Figure 1** and **Figure 2**). Partly because of this U-shaped distribution, the product-moment correlations of reported hours of sleep and frequency of insomnia were only −0.22 ($P<.001$) for women and −0.18 ($P<.001$) for men, reflecting that reported insomnia had little linear association with short reported sleep durations. Among women, the body mass index likewise had a U-shaped relationship to sleep duration (Figure 1), but among men, there was a virtually monotonic trend toward lower body mass indexes among those with longer sleep durations (Figure 2). Note that the mean body mass index for men reporting 3 hours sleep had sufficiently wide confidence intervals to be consistent with this trend.

The hazard ratios for various sleep durations adjusted for 32 covariates are given in the Table. Figures 1 and 2 illustrate that, among both women and men, the best survival was experienced by those reporting a usual sleep duration of 7 hours, which was 1 hour less than the modal sleep duration. Participants who reported sleeping 8 hours or more had distinctly and significantly increased mortality hazard: the longer the reported sleep, the higher the mortality hazard. When reported sleep exceeded 8.5 hours (as occurred among 7.5% of women and 7.7% of men), the added risk associated with long sleep exceeded 15%. Reported sleep had to be less than 3.5 hours among women (as occurred among only 0.1%) or less than 4.5 hours among men (0.7% of men) for the added risk associated with short sleep to exceed 15%.

Women reporting insomnia 1, 2, 3, 4 to 9, or 10 or more times per month had covariate-adjusted hazard ratios between 0.81 and 0.87, all of which were significantly less than the reference hazard of those reporting no insomnia (Table). Men reporting insomnia 1, 2, 3, 4 to 9, or 10 or more times per month had hazard ratios of 0.87 to 0.94, all of which were likewise significantly less than the reference. Women and men whose answer was missing also had ratios significantly less than the reference, which would be expected if some with missing responses had insomnia.

As previously reported,[30] participants who reported prescription sleeping pill use had significantly elevated mortality hazards, with control for sleep duration, insomnia, and other covariates (Table). The sleeping pill × insomnia interaction terms, when tested, were not significant additions to the models.

In the simplified models without full covariate control for comorbidities, hazard ratios (95% confidence interval) associated with 3 hours and 10 hours of sleep reached 2.05 (1.68-2.50) and 2.14 (2.04-2.28) for women and 2.49 (2.05-3.01) and 2.19 (2.09-2.29) for men, re-

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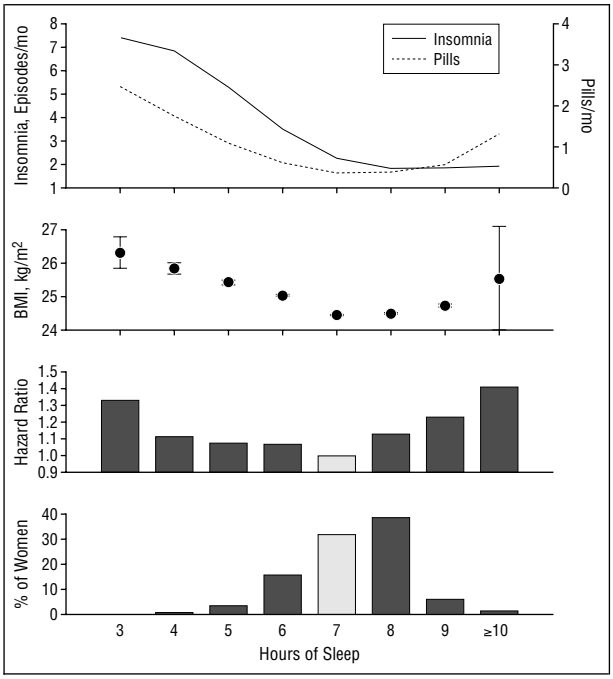

Figure 1. For 636 095 women, the average reported frequency of insomnia, the average number of sleeping pills used per month, and the mean body mass index (BMI) according to reported hours of sleep. The 95% confidence intervals of the BMI are shown. Also shown are the hazard ratios from the 32-covariate Cox models and the percentage of women reporting each sleep duration. The reference duration of 7 hours is represented by the lighter bars.

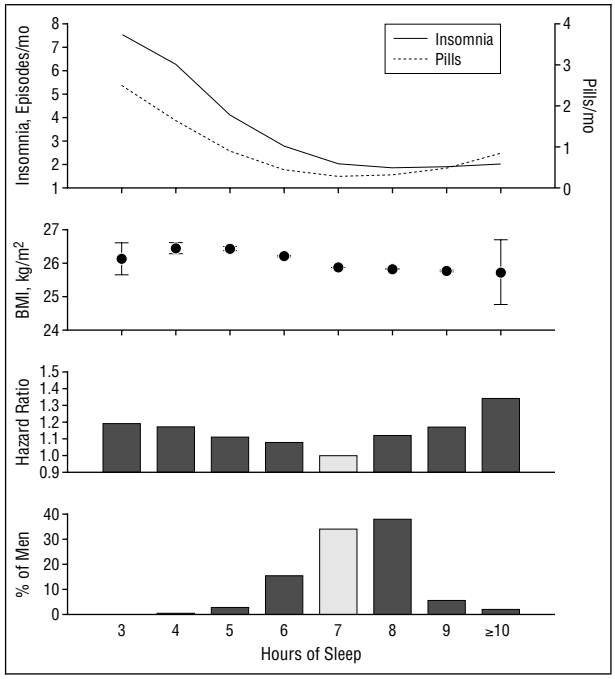

Figure 2. For 480 841 men, data comparable to those shown in Figure 1. BMI indicates body mass index.

spectively. However, removing insomnia or sleeping pill use from these simplified Cox models had only negligible effects (well within the confidence intervals) on the hazards ratios associated with sleep duration. When sleeping pill use was removed from the simplified models (ie, not controlled), the hazard ratio associated with insomnia increased slightly; eg, for insomnia 10 or more times per month, the ratio increased from 0.99 (0.94-1.05) to 1.05 (0.99-1.10) for women and from 1.15 (1.09-1.21) to 1.24 (1.17-1.30) for men. Removal of sleep duration had even smaller effects on insomnia hazard ratios. Moreover, there were only negligible effects on the hazard ratios associated with sleeping pill use when insomnia or sleep duration were removed from the simplified models.

For 8, 9, and 10 or more hours of sleep, the hazard ratios for cerebrovascular deaths were elevated over the hazard ratios for death from all causes among both women and men ($P<.05$ in 3 of 6 comparisons in data not shown). Hazard ratios for deaths from accidents, cancers, heart disease, suicide, and homicide were not consistently higher than the all-cause hazard ratios associated with short and long sleep.

COMMENT

A physician can reassure a patient that it is no longer average to sleep 8 hours. The lowest mortality hazard was experienced by participants reporting usual sleep of 7 hours (6.5-7.4 hours) per night. Sleep durations as short as 4.5 hours were associated with mortality hazards lower than that of almost half the sample. Comparison of the 32-covariate models with the simplified CPSII models and the less-controlled CPSI tabulations[6] showed that most mortality risk associated with short sleep could be explained by comorbidities. Also, a recent population sampling found that short sleep durations were not related to impaired health-related quality of well-being.[33]

Insomnia was not well defined in CPSII. There was only a weak correlation of reported insomnia with short sleep and little interaction. Reported insomnia was associated with no excess mortality hazard whatsoever, once sleeping pill use and other comorbidities were controlled. The absence of significant insomnia hazard noted in CPSI and CPSII has likewise been observed in smaller epidemiologic studies that controlled for comorbidities.[9,10,19,23,34-38] There is evidence that sleep complaints of various forms (not necessarily insomnia) predict coronary heart disease, but the odds ratios may be reduced with control for medications.[39] Moreover, some studies indicate that primary insomnia causes no substantial impairment of function.[1,11] For example, patients with insomnia may have no demonstrable loss of daytime alertness.[1,40] Less than 25% of patients referred for insomnia have primary insomnia as a first diagnosis.[41] Although there may be risks in depression, anxiety, heart disease, cancer, lack of exercise, sleep apnea, and other conditions in which insomnia is often present, patients with insomnia without underlying comorbidities can be reassured that there appears to be no survival risk, as long as the patients refrain from long-term use of sleeping pills. In one study, awakening during the night predicted decreased mortality,[8] similar to the hazard ratios less than 1.0 shown in the Table. However, we are not persuaded that insomnia is beneficial, because we found no dose-response relationship of insomnia frequency to decreasing hazard ratio, and a protective effect of insomnia was not suggested by the simplified models before 32-covariate adjustment. By comparison, mortality in-

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creased progressively in both men and women from 7 to 10 hours of sleep and with increasing sleeping pill use, and these associations were even stronger before adjustment for 32 covariates.

It is likely that control for additional risk factors or qualitatively improved control would further reduce apparent sleep-associated risks. However, there is a possibility that controlling statistically for comorbidities underestimates the mortality risk associated with short or long sleep. For example, if short or long sleep causes heart disease,[42] then correcting for a history of heart disease might obscure an underlying effect of sleep duration.

Above 7.5 hours, the longer participants reported sleeping, the greater their mortality hazard. Slightly elevated mortality hazard was noted even among those with the modal sleep duration (8 hours), suggesting that sleep duration is not merely a proxy for nonspecific hazardous deviations from modal health. This result was highly consistent between replications for women and men. It was also entirely consistent among age groups (unpublished observations from Cox models described previously, examining 4 age ranges from age 30-50 to >70 years).[30] Moreover, CPSII results were consistent with those of smaller studies[14-23] and with the previous results of CPSI.[6]

Currently, we do not know why sleep exceeding 7.5 hours was associated with excess mortality. Sleep duration in itself may not be a causal factor. Our group has suggested that sleep apnea might be a crucial underlying abnormality.[43] The elevated hazard for cerebrovascular deaths might be consistent with an apnea effect. Nevertheless, in a population study, we more recently found no significant relationship of long sleep to sleep disordered breathing.[44] Furthermore, since obesity is a key cause of sleep apnea, if sleep apnea were the major explanation, we would have expected more association of high body mass indexes with long sleep than is shown in Figure 2. Also, the hazard ratios were controlled for body mass index. Depression was not measured effectively in CPSII questionnaires, although depression might be associated with short and long sleep. However, because most insomnia complaints are accompanied by depression, we would have expected insomnia to predict excess mortality if depression explained the mortality hazard associated with short and long sleep. In addition, suicide deaths were not associated with short or long sleep out of proportion. Lacking understanding of causality, we cannot answer the crucial question of whether patients who sleep long could extend their survival by curtailing their sleep.

Long sleepers composed more than 90% of those whose sleep duration was associated with 15% or greater excess risk, although even for those sleeping 10 hours or more, the excess risk was modest. We may estimate that the sample excess fraction of deaths related to sleep duration was 6.3% for women and 5.3% for men.[45] If we allow the unproven assumption that long and short sleep cause the excess hazards with which they are associated, these would be the attributable risk fractions. In women, 83% of the excess and 78% of the excess in men were associated with sleep of 8 hours or more. Although polling and other data suggest a reduction in US population sleep durations in the latter part of the 20th century since CPSI and CPSII questionnaires were collected,[2,46-48] the health of the population has been improving. These data do not support any speculation that the population is sleeping too little on average.

As previously observed[30] and reiterated in the Table, the CPSII risk associated with sleeping pill use was greater than any risk associated with insomnia. Important limitations are that CPSII did not associate this mild mortality risk with particular hypnotic compounds or prove causality. The most popular hypnotics at the time CPSII data were collected, temazepam, triazolam, and flurazepam hydrochloride, have been largely supplanted by new benzodiazepine agonists with greater receptor specificity. It is unknown whether contemporary hypnotics are associated with comparable risks.[30]

A multitude of factors are associated with insomnia, short sleep, and the use of sleeping pills. No epidemiologic approach could leave us completely confident that statistical control for comorbidities would be sufficient but not excessive. To define causality, following the example of large randomizing trials examining diet, exercise, and critical medications, it might be possible to ascertain experimentally whether voluntary curtailment of sleep can prolong life or influence comorbid conditions. Likewise, it should be possible to determine experimentally whether any long-term treatment of chronic insomnia is safe and efficacious.

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Correction

Error in Figure Legend. In the article titled "Time Course of Effects of Testosterone Administration on Sexual Arousal in Women" (*Arch Gen Psychiatry*. 2000;57:149-153), the word *undecanoate* was mistakenly added to the legends for Figure 1 and Figure 3, as well as in the fifth paragraph of the "Participants and Methods" section. The ARCHIVES regrets the error.

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