Who Are the Long Sleepers? Towards an Understanding of the Mortality Relationship

Michael A. Grandner[1,2,3] and **Sean P. A. Drummond**[2,3]

[1]San Diego State University / University of California, San Diego Joint Doctoral Program in Clinical Psychology

[2]Psychology Service, San Diego VA Healthcare System

[3]Department of Psychiatry, University of California San Diego

Abstract

While much is known about the negative health implications of insufficient sleep, relatively little is known about risks associated with excessive sleep. However, epidemiological studies have repeatedly found a mortality risk associated with reported habitual long sleep. This paper will summarize and describe the numerous studies demonstrating increased mortality risk associated with long sleep. Although these studies establish a mortality link, they do not sufficiently explain why such a relationship might occur. Possible mechanisms for this relationship will be proposed and described, including (1)sleep fragmentation, (2)fatigue, (3)immune function, (4)photoperiodic abnormalities, (5)lack of challenge, (6)depression, or (7)underlying disease process such as (a)sleep apnea, (b)heart disease, or (c)failing health. Following this, we will take a step back and carefully consider all of the historical and current literature regarding long sleep, to determine whether the scientific evidence supports these proposed mechanisms and ascertain what future research directions may clarify or test these hypotheses regarding the relationship between long sleep and mortality.

Keywords

sleep; long sleep; sleep duration; mortality; depression

WHO ARE THE LONG SLEEPERS?

How much sleep do we need?

Whereas the answer to this question usually involves the determination of the minimum amount of sleep required for the maintenance of health, less attention has been directed to the question of the maximum amount of sleep for optimum health. This may be an equally important issue, given research suggesting that there are negative health consequences of excessive sleep[1].

There is growing public awareness of the need for an adequate amount of sleep, and the myriad problems associated with insufficient sleep, which is consistent with a hypothesis

that our society is chronically sleep-deprived as a whole. While this hypothesis has been debated throughout the scientific literature[2,3], it has been widely reported that average sleep durations have decreased substantially[4].

While obtaining too little sleep is certainly problematic, at least some argue that obtaining too much sleep may also be problematic. For example, an analogy between sleep and diet has been made, suggesting that moderation, rather than abundance, may be most healthy[5].

A U-shaped relationship between sleep duration and mortality[5] is consistent with other relationships between overall poor health and variables such as exercise, diet, and light exposure. As in these relationships, moderation in sleep quantity, rather than extreme low or high amounts, may be best for health. If that is the case, the increased desire for sleep beyond 8 or 8.5 hours may be less indicative of genuine sleep debt and more akin to the desire for more high-calorie foods. Thus, both short and long sleep are related to increased mortality risk; this raises the question: How much sleep do we need?

While the available scientific literature is currently unable to provide a definitive answer to this question, and the available studies have limitations, we will summarize and describe the numerous studies demonstrating increased mortality risk associated with long sleep, defined as a habitual reported sleep length of at least 9 hours and characteristically distinct from clinical hypersomnias. It will also be shown that although these studies establish a mortality link, they do not sufficiently explain why such a relationship might occur. Subsequently, possible mechanisms for this relationship will be proposed and described. Following this, we will take a step back and carefully consider all of the historical and current literature regarding long sleep, to determine whether the scientific evidence supports these proposed mechanisms and ascertain what future research directions may clarify or test these hypotheses regarding the relationship between long sleep and mortality.

Regarding this last goal, while there has been relatively little research on long sleep (as compared to restricted sleep), there is a sufficient body of work to formulate and evaluate hypotheses. First, differences between characterized long and short sleepers will be described, from physiological and psychological domains. Second, a summary of findings related to characteristics of variable-length sleepers (those who alternate between long and short sleep) is provided, so that we may understand what variables may relate to changes in habitual sleep pattern, possibly revealing the functions of long sleep. Also, understanding these factors may help clarify hypothesized mechanisms underlying development of long sleep patterns which might play a role in mortality risk. Finally, results of laboratory sleep extension are reviewed to document possible effects of mild and moderate lengthening of sleep time in controlled settings. These findings provide another perspective of physiological and psychological changes that occur when sleep is lengthened, and the changes may also elucidate mechanisms of the mortality relationship.

LONG SLEEP AND INCREASED MORTALITY

Increased mortality associated with long sleep

Although negative psychological and other health outcomes of extreme sleep lengths have been reported for over 45 years[6,7], relatively little attention has been devoted to the study of long sleepers. A resurgence of interest in studying long sleep may be due to several recent studies relating long sleep to increased mortality.

A recent study of over 1 million American adults age 30–102 reported that sleep durations below 6.5 hours and above 7.5 hours were associated with increased mortality hazard[5]. Most of the increased mortality was associated with longer sleep (over 7.5 hours in this study),

which was reported with 2.5 times the frequency of short sleep (less than 6.5 hours in this study). This finding persisted across age and gender, and the analysis controlled for over 30 other variables. A recent study of over 100,000 Japanese adults aged 40–79 found that sleep duration greater than 7 hours was associated with increased mortality. This report further controlled for "mental condition," including stress and depressive symptomatology[8].

Several other recent studies have also reported an association between long sleep and increased mortality[9–18]. These studies replicate similar findings in earlier studies of long sleep and mortality risk[6,19–22]. In fact, there is only one known survival study favoring long sleep over short or moderate sleep amounts,[23] though long sleep was associated with increased risk of cardiovascular death in women in this study. These studies, spanning over 45 years, have repeatedly shown an association of long sleep with increased mortality risk. In addition to a direct relationship with sleep length, there have been a number of studies that show increased mortality risk associated with sleep complaints that are more common in long sleep. For example, Mallon and colleagues[24] found that sleep duration complaints (short or long) and depression were both associated with increased mortality risk. Nilsson and colleagues [25] demonstrated a relationship between subjective sleep disturbance (e.g., problems falling asleep, early awakening) and increased mortality risk. Additionally, Dew and colleagues[26] conducted an electroencephalographic (EEG) study of sleep duration and increased mortality and found that, controlling for psychiatric and medical problems, long sleep latency and poor sleep efficiency (which are more disturbed in long sleepers) were associated with increased mortality. Short sleep duration, however, was not associated with elevated mortality in this study. A summary of the results of all known mortality studies are presented in Table 1.

Problems with epidemiological studies

While many studies have reported a link between increased mortality and long sleep, there are several important limitations to these findings. First, long sleep is sometimes defined as sleep over 8 hours, while others have used different cutoffs, such as 9 hours or a specified extreme end of the distribution of sampled sleep times. While this inconsistency reduces the generalizability of the conclusions across studies (e.g., are they all measuring "long sleep?"), the remarkable convergence of findings strengthens overall reliability of the long sleep - mortality link.

Second, since these studies are primarily epidemiological, they employ survey and self-report measures of sleep duration, which may better reflect time in bed rather than actual sleep time[27]. Additionally, subjective and objective measures of sleep duration are often discrepant, and both measures inherently contain error in their estimation. Together, this means it is not known whether increased mortality risk associated with long sleep directly involves EEG sleep time or time in bed. Nonetheless, studies that have attempted to measure time in bed separately from sleep time[11,14] have found that mortality relationships were consistent across both measures. Additionally, rationales for the relationship between mortality and long sleep also apply to long time in bed, such that many of the reasons that long sleep may contribute to mortality risk apply to extended time in bed as well.

Third, these studies could not accurately describe how risks associated with long sleep vary across the lifespan. While age was used as a covariate, it is likely that the mechanisms by which sleep duration increases mortality risk have varying effects at different stages of life.

Finally, these large studies fail to adequately measure the extent to which those reporting long sleep do so because of a biological need for more sleep, rather than an extension of sleep. Thus, there may be some self-described long sleepers that experience negative effects

of long sleep and others for whom less sleep would be associated with the negative consequences of sleep deprivation.

Possible causes of mortality in long sleep

While the numerous studies mentioned above contain some problems regarding limitations imposed by methodology, they do all converge on the common finding that long sleep is associated with mortality. However, these studies do not sufficiently propose or test mechanisms through which this relationship would manifest. Thus, a review of the possible ways in which long sleep could lead to increased mortality is necessary.

There are a number of possible explanations for the association between long sleep and increased mortality. Possible mechanisms for this relationship will be proposed and described, including (1) sleep fragmentation, (2) fatigue, (3) immune function (e.g., change to immune function), (4) photoperiodic abnormalities, (5) lack of challenge (e.g., lack of physiological challenge), (6) depression, or (7) an underlying disease process such as (a) sleep apnea, (b) heart disease, or (c) failing health. A graphical summary of these proposed mechanisms is shown in Figure 1.

Excessive time in bed have been linked to increased sleep fragmentation, wake after sleep onset (WASO) and sleep latency[1,26], each of which occur when the sleep period is punctuated by numerous intervals of wakefulness. Sleep fragmentation, in particular, has been associated with a number of negative health outcomes, including decreased energy and vitality and increased role limitations [28]. Fragmented sleep may be caused by long time in bed or long sleep by extending the latter portion of sleep associated with more frequent awakenings[29] . It may also be the case that long sleep (or time in bed) leads to reduced sleep efficiency through higher WASO or sleep latency, which may be associated with mortality, rather than fragmentation itself.

Second, long sleep has been associated with feelings of fatigue and lethargy[30,31], as has induced sleep extension[32] . These states may decrease resistance to stress and disease[33], which, in turn, may lead to increased mortality. It should also be noted that fatigue and lethargy may cause long sleep.

Third, long sleep may influence the expression of cytokines (inter- and intra-cellular components of the immune system). Changes in cytokine expression associated with changes in sleep may influence mortality[34–38].

Fourth, long times spent in a darkened environment may be related to a shortened photoperiod (the physiological day based on the light-dark cycle). Such photoperiodic changes and abnormalities have been associated with mortality in mammalian species[39]

Fifth, long sleep (or even long time in bed) may be associated with a lack of physiological challenge. A number of stressors, such as exercise, irradiation, heat and cold shock, and hypergravity, have been linked to longevity in many species, when compared to a relative absence of these stressors[40]. There are two possible ways in which long sleep may decrease longevity by decreasing possibly beneficial mild stressors. First, Mild sleep restriction may itself be one such stressor, associated with increased longevity; second, more time in bed provides fewer opportunities for the experiencing of other such stressors. However, it may be implausible that as little as one hour per day (8.5 hours vs. 7.5 hours) may have much of an effect on overall physiological challenge.

Sixth, there may be an underlying disease processes that mediates the relationship between long sleep and mortality. Although many risk factors were controlled in the statistical

exploration of this relationship in the epidemiological studies reviewed above, it is possible that a high rate of undiagnosed illness may have contributed to both long sleep and mortality. There are at least four such diseases to consider. One is obstructive sleep apnea. Sleep apnea has been associated with increased risk of hypertension and carotid artery disease[17]. Several researchers have suggested that the relationship between sleep duration and mortality risk may be the result of patients with sleep apnea compensating for the large degree of fragmentation associated with the disorder by spending more time asleep or excessive amounts of time in bed[41]. Thus, it may be that mortality is related to variables associated with sleep apnea, rather than long sleep per se.

However, there is not sufficient evidence to suggest that patients with sleep apnea sleep any more or less than the average[27] , though they may spend more time in bed. Additionally, many very reliable predictors of sleep apnea, including age, gender, body mass index and ethnicity, were controlled for in these epidemiological studies[5,17]. Moreover, the relationship between sleep duration and increased mortality risk has been evidenced while controlling for diseases associated with sleep apnea (e.g., cardiovascular disease), as well as with those that are not (e.g., cancer)[5]. Overall, then, it seems unlikely that a disease such as sleep apnea is the mechanism explaining the link between long sleep and mortality.

Another condition which may mediate the relationship between long sleep and increased mortality risk is depression. Depression can be associated with both long and short sleep, as well as over- or under-estimation of sleep time. Most of the previously mentioned epidemiological studies did not adequately measure mood disturbance. However, two of the large epidemiological studies that did measure mood disturbance showed that increased sleep duration was related to increased mortality over and above mood symptoms[8,42]. Moreover, sleep disturbance in depression is usually characterized as insomnia or lack of sleep rather than long sleep, and insomnia has not been consistently associated with increased mortality risk[5].

Some researchers have proposed a relationship between coronary disease and sleep duration. Ayas and colleagues[42] found that short and long sleep are associated with coronary events. A recent study found a U-shaped relationship between sleep duration and hypertension[43]. Glaser and Glaser[44] suggest that mortality due to coronary events in long sleep may be due to blood pressure dipping too low over the course of the night, increased blood clotting or thrombosis. However, Mallon and colleagues [24] found that long and short sleep did not predict coronary artery disease mortality.

Finally, mortality risks of long sleep may be associated with general failing health. Long sleep is most prevalent in those over 60 years old[5] and sleep is known to lengthen in the last weeks or months of life[45]. It may be that sleep duration lengthens gradually during the aging process and/or during periods of failing health. Under this possibility poor health would lead to long sleep, rather than long sleep leading to increased mortality.

To summarize, mortality associated with long sleep may be due to: (1) sleep fragmentation, (2) fatigue, (3) immune function (e.g., change to immune function), (4) photoperiodic abnormalities, (5) lack of challenge (e.g., lack of physiological challenge), (6) depression, or (7) an underlying disease process such as (a) sleep apnea, (b) heart disease, or (c) failing health. Additionally, it should be noted that these mechanisms are not mutually exclusive.

While some empirical support exists for these possibilities, relatively little research has attempted to elucidate the possible causal mechanisms underlying the relationship between long sleep and mortality. Also, relatively little is known about long sleepers, including factors that may influence the development and maintenance of patterns of long sleep, and the consequences of long sleep. These things may play roles in the observed mortality

relationship, and a better understanding of the mechanisms and consequences of long sleep need to be understood in order to formulate and modify hypotheses regarding methods of action in the long sleep/ mortality relationship.

Before the mechanisms we propose may be responsible for the long sleep -mortality relationship can be directly tested, long sleepers need to be characterized, so that these hypotheses can be refined to more accurately reflect conditions actually associated with long sleep. While it may be plausible that all of these mechanisms relate long sleep and mortality, we first need to learn whether these mechanisms are associated with long sleep at all. To do this, we now examine the current literature to evaluate current support for these mechanisms and identify areas in which further characterization is needed.

DEFFERENCES BETWEEN LONG AND SHORT SLEEPERS

One way to understand long sleep is to examine differences between long and short sleepers. While differences between long and normal sleepers may be more relevant, most studies have examined the extremes of sleep duration relative to each other, rather than in relation to normal sleep. Despite limitations, these differences may highlight important characteristics of long sleepers suggesting mechanisms of the mortality relationship.

Scientific investigation of the correlates of extreme sleep duration has been conducted for over 80 years. Moore and colleagues[46] found that shorter sleep was associated with muscular inefficiency, but Barry and Bousfield[47] found that short sleep of 5.75 hours or less was associated with increased physical activity. Additionally, this study reported that ratings of "euphoria" were highest at reported sleep durations of 8 to 8.75 hours, though long sleepers of 9 hours or longer did not seem to report less "euphoria." Over the subsequent decades, a number of studies have described differences between long and short sleepers.

Correlates of long versus short sleep

While there is relatively little known about problems associated with long sleep and how these may or may not be related to the mortality relationship, there have been several studies that have described some correlates of long sleep. While causal connections cannot be drawn from these studies, they can suggest possible future research directions by identifying conditions that are associated with long sleep. The paragraphs below will describe some demographic, diagnostic, behavioral and physiological correlates of long sleep.

Several epidemiological studies have examined correlates of sleep duration. A recent report has found numerous correlates of self-reported habitual long sleep from the Nurses Health Study II[48]. This study found that long sleepers (at least 9 hours per night) reported increased likelihood of a history of depressive symptoms, antidepressant use, benzodiazepine use, lack of physical activity, never married or divorced status, living alone, lower income, unemployed status, and lower social status. Lower socioeconomic status may be a primary driving factor in the long sleep-mortality relationship, and should be examined. Specifically, future studies could discern how socioeconomic status alters sleep duration.

Additionally, long sleep was associated with increased likelihood of the diagnosis of a number of medical conditions, including obesity, diabetes, hypothyroidism, seizure, head injury, multiple sclerosis, stroke, coronary disease, hypertension, lupus, rheumatoid arthritis, osteoarthritis, herniated disc, asthma, chronic obstructive pulmonary disease, cancer, and inflammatory bowel disease. Finally, long sleep was associated with restless legs syndrome, snoring and permanent night shift work. However, although all of these factors were associated with long sleep, none of them fully explained the mortality relationship with long sleep.

Depression was the factor that contributed most and may be most likely to confound the long sleep-mortality relationship[48].

A population study from Finland[49] found that long sleepers reported more feelings of being "non-energetic or tired" than mid-range sleepers, but no difference was found for short sleepers. A similar pattern was found for endorsements of feeling "exceptional tiredness." Self-reports of "sleeping disorders or insomnia," "difficulties in getting sleep without sleeping medicines," "overexertion or exhaustion" and "being awake during the night" were reported more in short sleepers than long sleepers.

Another recent report[50] suggests that short sleep (6–7 hours or <6 hours) was associated with lower reports of self-rated overall health than normal (7–8 hours) or long (8–10 or >10 hours) young adult sleepers. There were no differences reported between normal and long sleepers in this study. Interestingly, when depression was entered into statistical models, odds ratios were attenuated but otherwise did not change between sleep duration and health report. This may reflect ontogenetic factors in the relationship between sleep duration and health. A study by Pinheiro and colleagues[51] suggests that long sleep (at least 8 hours) is associated with risk of developing breast cancer, relative to sleep less than 8 hours. Additionally, as sleep duration increases, risk for breast cancer seems to increase, and as sleep duration decreases, risk seems to slightly decrease.

Several studies have demonstrated that increased sleep duration and time in bed are associated with increased weight and daytime sleepiness and decreased mood, sleep quality, health and quality of well-being[52,53]. However, daytime sleepiness has been associated with sleep fragmentation rather than simply sleep duration, and since sleep fragmentation is directly related to time in bed, perhaps increased time in bed is itself harmful[27].

Short sleep duration is associated with increased ghrelin and decreased leptin[54], although long sleepers or a U-shaped relationship were not addressed. Ayas and colleagues[42] found a U-shaped relationship between sleep duration and risk for diabetes, suggesting that long and short sleepers have approximately 1.5 times the risk as normal length (7–8 hours) sleepers. This finding was recently replicated, suggesting a 2.5-fold increase risk for diabetes in short sleepers and a 3-fold risk of diabetes in long sleepers[55]. Motohashi and colleagues[56] found that there was no difference in blood glucose, blood cholesterol, HDL, cholesterol, and triglycerides in long versus short sleepers. Additionally, Hicks and colleagues[57] found that long sleepers tend to eat larger meals and snacks less frequently than shorter sleepers, who tended to have a less regular meal pattern. These findings are in contrast to other recent evidence that partial sleep deprivation is associated with disrupted leptin and ghrelin as well[58]. Also, several recent studies suggest that short sleep, rather than long sleep, is associated with increased obesity[59], as well as intake of fats [60]. It is unclear which of these endocrine abnormalities are associated with sleep duration versus sleep disruption. Additionally, the fact that data suggest endocrine abnormalities seem to play a role in short sleep more than long sleep implies that the mechanisms for health effects on both ends of the spectrum may not be the same. Also, these changes may not reflect a continuous effect – rather, perhaps there are certain long and short sleep thresholds that increase risk.

Ohayon[61] found that long sleep was associated with age of 65–74 years, not working, being underweight, lack of physical activity, disrupted sleep, use of sleeping medications, and anxiety disorder in an older sample. Short sleep was associated with age between 55–64 or 74–84 years, lack of physical activity, drinking six cups or more of coffee per day, taking sleeping medications, difficulty initiating sleep, disrupted sleep, early morning awakening, and anxiety disorder.

Thus, sleep duration has been shown to be related to a litany of variables, some of which may or may not be related to the mortality relationship. Most notably, long sleep may be associated with depression, medication use, lack of activity, lower socioeconomic status, medical problems, dietary problems, heart problems, breathing problems, and sleep disorders. These factors seem to support many of the proposed mechanisms, particularly sleep fragmentation, fatigue, immune function, sleep apnea, depression, and heart disease In addition to these correlates of long and short sleep, long sleep is related to many circadian, sleep-related and psychological factors.

Circadian characteristics of long versus short sleepers

Aschoff[62] reported that perception of time and activity durations may be individual traits and may be different in long and short sleepers. There have been several other studies that describe relationships between circadian and photoperiodic variables and sleep duration.

Long sleepers demonstrate lower average daily temperature than controls[63] and tend to initiate sleep much closer to the circadian body temperature peak than short sleepers[64]. It is interesting to note that this finding is similar to those in temporal isolation studies, where subjects tend to initiate sleep closer to the temperature peak[65]. However, long sleepers may initiate sleep at this point only because it coincides with an earlier bedtime, to accommodate longer sleep while adhering to a regular wake time[8]. Alternatively, it may be that long sleepers have a reduced ability to tolerate sleep pressure, which then leads to increased sleep, as suggested by Aeschbach and colleagues [66,67].

Another physiological process that drives sleep is the regulation of the melatonin system. Melatonin is a hormone that is moderated by light transduction through the retina to the hypothalamus and signals the lack of light or duration of night in the environment – it is released in darkness and is inhibited by light. Endogenous melatonin functions as a sleep-promoting agent in diurnal animals such as humans; longer durations of melatonin excretion (via longer night) are related to increases in sleep, and shorter durations (associated with more light) with less sleep. Aeschbach and colleagues [68] reported that long sleepers have a longer duration of melatonin secretion at night relative to short sleepers. It may be the case that this produces longer sleep durations. Conversely, it may also be the case that more time in bed leads to increased melatonin secretion durations, leading to more sleep. There is experimental data that time in bed (or duration of the dark period) influences melatonin duration, but little experimental evidence for the converse, and melatonin agonists have only very minimal effects on sleep duration[69]. Thus, the increased duration of melatonin secretion in long sleepers may be a consequence, rather than cause, of extended time in bed.

Measures of "morningness" and "eveningness" are often utilized in circadian rhythms research to characterize people based on expressions of endogenous rhythms[70]. Several findings among long and short sleepers have demonstrated differences in this domain. Taillard and colleagues[71] demonstrated that eveningness was associated with a greater need for sleep (which may be characteristic of long sleepers), as well as increased sleep debt. Vera-Villarroel and colleagues [72] showed that long sleepers demonstrated less morning activity than short sleepers, as well as more temperature sensitivity and less EMG activity in the morning than short sleepers.

These findings suggest that long sleepers may demonstrate circadian and photoperiodic differences, including increased homeostatic sleep pressure and melatonin duration, as well as different activity rhythms and perceptions of rhythmicity. These findings support the photoperiodic hypothesis of the long sleep-mortality relationship, although they do not directly assess photoperiodic and circadian variables influences on mortality risk.

Objective sleep in long versus short sleepers

Early studies of sleep differences in long and short sleepers reported that long sleepers demonstrated greater sleep latency and more WASO, REM, stage 1 and stage 2 sleep, and equivalent or less slow wave sleep than short sleepers[29]. Fukuda and colleagues[29] also found that short sleepers had higher REM density in the 2nd and 3rd REM cycle. Aeschbach and colleagues[66] found that following sleep deprivation, sleep latency and REM density went down in short sleepers, and slow wave sleep was affected to a greater extent in long sleepers.

These sleep differences are consistent with the idea that long sleep is not characteristically different from lengthened sleep, such that sleep characteristics typically associated with the end of the night tend to be more present in long sleep and sleep at the beginning of the night seems to be less affected. However, there may be abnormalities associated with slow wave sleep in long sleepers, such that they may experience less overall, despite more time asleep. Slow wave sleep reductions are also associated with aging and health problems associated with a number of the hypotheses regarding the long sleep-mortality link, including reduced immune function and conditions associated with decreased health[73]. Also, long sleep may be associated with REM abnormalities. As REM abnormalities may be related to mood disturbance[74], there may be a possible mood component in long sleep that is consistent with the depression hypothesis underlying the mortality relationship.

Studies of endocrine function during sleep have suggested that long sleep periods are associated with disturbed prolactin rhythms[75] and that patients with growth hormone deficiencies show marked increases in total sleep time following treatment without increases in REM sleep[76]. It is unclear how these findings relate to the larger picture of sleep differences in long sleep, but reinforce the idea that changes in sleep duration are related to endocrine function, as noted in the aforementioned increased diabetes risk.

Subjective sleep in long versus short sleepers

Two large studies have reported increased rates of self-reported insomnia in long sleepers, compared to average sleepers[5,77]. Another study, analyzing data from a census-matched sample polled by the National Sleep Foundation, found that long sleepers report significantly more complaints than average sleepers[31]. Specifically, they reported increased rates of trouble falling asleep, awakenings during the night, early morning awakenings, nonrestorative sleep and daytime sleepiness. One possible explanation for these findings is that increased time in bed leads to fragmentation (described below), which may lead to these complaints.

Behavioral sleep practices in long versus short sleepers

Hicks and Youmans[78] report that long sleepers are more likely to engage in sleep-promoting behaviors than short sleepers, reporting twice as many different sleep-promoting behaviors, and a trend toward increased frequency of behaviors. Long sleepers were more likely to use alcohol (perhaps to induce sleep) and short sleepers more likely to use food before bed to induce sleep. Alcohol intake close to bed time increases sleep fragmentation and daytime sleepiness after waking[79], potentially contributing to the increased reports of insomnia and poor sleep quality in long sleepers reviewed above. These findings, however, may be confounded with the age and social background of subject groups.

Overall, data on objective sleep, subjective sleep, and sleep behaviors suggest that long sleepers normally experience fragmented sleep, report more complaints about their sleep, and engage in more compensatory behaviors in an effort to increase sleep than do others. Long sleepers also show an exaggerated slow wave sleep rebound after sleep loss. Taken

together, these findings support the photoperiodic findings by suggesting there may be homeostatic dysregulation in long sleepers, which may be indicative of depression, sleep fragmentation, sleep apnea, or other conditions that may underlie the mortality relationship.

Psychological characteristics of long versus short sleepers

Research literature on psychological characteristics of long sleepers has shown several interesting patterns, suggestive of impaired functioning and increased risk for negative health outcomes. As such, these data may also aid the development and evaluation of hypotheses regarding the mechanism underlying the link between long sleep and mortality.

Numerous studies have noted some psychological characteristics that may differentiate long and short sleepers. For example, Hartmann and colleagues [80] found that on the California Personality Inventory (CPI), long sleepers reported lower "social presence," "sociability," and "flexibility" than short sleepers. Taub[81] found that long sleepers reported more "aggression and misfortune elements" in dreams. On the Minnesota Multiphasic Personality Inventory (MMPI), long sleepers have scored lower on scales measuring social desirability[7, 80] and mania[82] and higher on scales measuring social introversion[7,80,82] and psychiatric disruption[82].

Long sleepers may also demonstrate impaired coping abilities, which may lead to poor health outcomes. Long sleepers report less of an external locus of control than short sleepers[83,84] . Hicks and colleagues[85] found that following a particularly devastating earthquake in San Francisco, long and short sleepers differed in their responses on the Ways of Coping Questionnaire. Long sleepers reported lower "Confrontive," "Self-controlling," "Accepting responsibility," and "Positive reappraisal" scores than short sleepers. Hicks and Picchioni[86] found that after taking a long sleep, fewer positive coping strategies were reported, including "self-controlling," "responsibility," "problem solving," and "reappraisal." In a study of reported stressful events in long, short and variable length sleepers, Sexton-Radek[87] found that long sleepers had more inconsistent responses to stress. When describing stressful events, long sleepers tended to emphasize performance quality, whereas short sleepers tended to emphasize objective performance and variable sleepers tended to emphasize interpersonal stresses.

Hartmann and colleagues[7] also conducted clinical interviews with all of the long and short sleepers. Long sleepers reported more worries. They were "less definable," but had variable opinions and tended to be "nonconformists and critical." They were found to be "more uncomfortable in many ways" than short sleepers. They "complained" more, were "overly anxious" and inhibited and may even have been "mildly depressed." They were unsure of themselves. They tended toward being creative and artistic. Sometimes they use sleep to escape from an unpleasant experience. They were "definitely worriers."

Long sleepers reported having started work at an older age and for fewer hours per week than short sleepers[80]. More recently, Motohashi and colleagues[56] found that short sleep was associated with increased overtime work.

Later studies demonstrated that short sleepers reported more anxiety[88] than long sleepers and more hypomania symptoms[89] than controls. In contrast, Jean-Louis and colleagues [27] found that increased sleep duration was not related to reported quality of well-being, though the range in the sample was only between 5–9 hours.

Taken together, these findings suggest that long sleepers are particularly susceptible to psychopathology, especially problems with mood and anxiety. Long sleepers might be better characterized as "worriers" and were typically less successful than short sleepers. These

findings are very consistent with those recently reported by Patel and colleagues[48]. Regarding mortality, these findings suggest that long sleepers are predisposed to many stressors, which may be related to increased depression, heart disease, fatigue and lack of challenge.

STUDIES OF VARIABLE SLEEPERS

Variable sleepers are, by definition, people who fluctuate between short sleep, normal sleep, and long sleep, depending on various factors[30]. Understanding variable sleepers may provide insight into the function of sleep[30] and may elucidate mechanisms by which people become long sleepers. Understanding how a person becomes a long sleeper may help clarify the long sleep - mortality connection.

Hartmann and Brewer[90] reported that increased need for sleep was found when subjects reported a period of stress. However, Hicks and Garcia[91] found that high stress leads to reduction in sleep time of about 1 standard deviation and low stress to an increase of about 1 standard deviation of sleep time. Nonetheless, findings from previously mentioned studies[7,80,82,87] argue that lengthening sleep is related more to stress and stressful events than positive events. Sanford and colleagues[92] note that an aversive event can shorten or lengthen sleep in mice for days, depending on the amount of conditioning and training the animal has received. While the neurological mechanisms of this response are currently not completely understood, some investigation has implicated that the noradrenergic locus coeruleus plays a role[93].

Taken together, these results reiterate many of the findings associated with characterological data on long sleepers. Long sleep may be brought on by stress and stressful events. Variable sleepers may respond to stressors with increased sleep but do not maintain this pattern. It is currently unclear why some people develop this pattern for many years and some do not. These findings do, however, support the hypothesis that the relationship between long sleep and mortality may be driven by the factors that lead to long sleep in the first place, such as difficulty handling stress, which may be secondarily associated with depression, fatigue, or poor health.

CONTROLLED SLEEP EXTENSION

While there have been few studies of laboratory sleep extension, this literature may inform hypotheses regarding the long sleep and mortality relationship. These studies document the physiological and psychological effects of artificially lengthening otherwise normal sleep. Clarifying these responses may provide further evidence for mechanisms of the mortality relationship.

By spending additional time in bed, it is possible to increase sleep duration up to 10–12 hours[94] for a few weeks only. A common finding, called the "Rip van Winkle Effect," can be seen when adults sleep longer than usual – they often report a number of symptoms that can persist for hours, including feelings of fatigue, lethargy, irritability and sleepiness for up to 4.4 hours after finally waking[95] . These feelings of sleepiness and fatigue may lead to the increased desire to sleep, creating a vicious cycle of increased sleep and sleepiness.

These results are supported by research[96] which found that average-length sleepers who extend their sleep duration by 2–3 hours report sleepiness, decreased mood, slowed reaction time, lowered vigilance and problems with mathematical tasks. Some studies demonstrated decreases in objective sleepiness[97] and increases in objective sleep[98] following extended sleep, but these studies did not measure subjective experience of sleepiness, fatigue or mood impairment.

Sleep extension that increases time in bed will likely increase sleep fragmentation[99] and this fragmentation is associated with a number of negative health outcomes and sleep complaints. If time in bed is extended to 24 hours, sleep becomes extremely fragmented, resembling that of nursing home patients who cannot stay awake or asleep for more than an hour at a time[100].

LONG SLEEP IN CHILDREN AND ADOLESCENTS

One way in which long sleep risks may be pertinent to pediatric populations is the increasing incidence of sleep apnea in children, which may be related to increasing rates of overweight[101] and is associated with performance deficits[102].

Few studies have specifically identified risks associated with long sleep duration in children. Sampei and colleagues[103] found that in a group of 5–6 year-olds, the quartile with the highest reported sleep duration (>=660 minutes) demonstrated the highest values of systolic blood pressure than the quartile with the lowest reported sleep duration (<=585 minutes). No relationship was found for diastolic blood pressure. A recent study by Gaina and colleagues[104] found that short sleep duration (and increased sleep latency) was associated with evening-type preference in adolescents aged 12–15 years, though Giannotti and colleagues[105] found that in students aged 14–19 years, eveningness was related to short sleep duration during the week, but long sleep duration on weekends, when sleep schedules may better approximate endogenous preference. These students demonstrated more napping during school days, daytime sleepiness, attention problems, and injuries. They also demonstrated poorer school achievement and more emotional upset than the other chronotype. These data illustrate that pediatric populations typically sleep longer than adults, and that sleep duration may be related to similar variables as adults, including health and functional impairment. However, it is unclear whether these relationships are valid for natural long sleepers, short sleepers or both.

CONCLUSION

While there are some reports of correlates and effects of long sleep in the scientific literature, there seems to be a relationship between long sleep and increased mortality risk, which may or may not be related to a number of factors. Six proposed mechanisms of this relationship include: (1) sleep fragmentation, (2) fatigue, (3) immune function, (4) photoperiodic abnormalities, (5) lack of physiological challenge, (6) depression, and (7) an underlying disease process, such as (a) sleep apnea, (b) heart disease, or (c) failing health.

The present literature suggests that long sleepers are characteristically different from short or normal sleepers in a number of domains; they present with demographic, circadian, sleep-related, psychological, and other differences. Taken together, these findings tend to support many of the proposed hypotheses. While most of the available studies did not examine long sleep in the context of mortality, there seems to be a significant overlap between long sleep and depression[1], sleep fragmentation[26,28–29] , and photoperiodic abnormalities[1]. Health issues (e.g., sleep apnea[41] , heart disease[42], and failing health[5]) and lack of challenge were also somewhat supported, though usually indirectly. Thus, much of the evidence supports these hypotheses to varying degrees and future studies should examine long sleep with respect to these domains, especially depression and sleep fragmentation (which itself may be indicative of sleep apnea or other conditions).

Studies of variable sleepers have elucidated some of the possible functions of long sleep and further research in this direction will better determine whether long sleep may be a consequence of stress or coping mechanisms. Sleep extension studies highlight some of the effects of artificially lengthening sleep.

What are the physiological characteristics of long sleepers?

Several recent studies have indicated that long sleepers have a characteristically different melatonin rhythm than short sleepers[68]. Other circadian physiological differences also seem to characterize long sleepers, such as temperature, timing and activity[63–67]. Future studies will further elucidate these findings and possibly extend them. For example, there may be CLOCK gene polymorphisms associated with long and short sleepers.

How can we describe the sleep of long sleepers?

Long sleepers seem to present with an extended sleep period that contains more REM, stage 1, and stage 2 sleep, which is to be expected. Long sleepers may have decreased slow wave sleep[29]. They also may experience more sleep fragmentation. More complex polysomnographic techniques may clarify and expand these findings.

What are the psychological characteristics of long sleepers?

Numerous studies have demonstrated that long sleepers experience more anxiety and social introversion and less psychological health than short sleepers[7,80–82] . However, most of these studies were conducted over 30 years ago and have not been able to make use of much advancement in research and medicine since that time. Therefore, these studies need to be reevaluated and their findings need to be replicated.

How do the differences between long and short sleepers relate to the findings of increased mortality risk?

The circadian and photoperiodic findings implicate a photoperiodic mechanism in mortality. Increased incidence of psychological distress, depression, and anxiety, and decreased social interaction, coping skills and organization paint a picture of general poor psychological functioning, which is characteristic of many of the proposed mechanisms, most notably sleep fragmentation, fatigue, lack of challenge and depression. Taken together, many of the findings related to long sleep tend to agree with many of the proposed mechanisms of mortality associated with long sleep. However, many of these studies employed questionable methods. Additionally, most of these studies were purely cross-sectional, disallowing any conclusions of causality. Finally, many studies that would test some underlying mechanisms have not yet been conducted, including specific studies of immune functioning in long sleepers, psychological functioning in long sleepers, and genetic abnormalities associated with long sleep.

What are the clinical implications of these findings?

The findings inform sleep evaluation and treatment in a number of important ways. First and foremost, they establish that sleep evaluation should examine long sleep in addition to sleep insufficiency. Second, these findings have implications on the assessment of sleep quality. Long sleepers report and demonstrate poorer sleep quality than normal sleepers. However, sleep assessment instruments traditionally assign scores based on sleep insufficiency, as opposed to sleep inefficiency. Screening measures might need to include long sleep in addition to short sleep in their assessments. Third, mild sleep restriction for long sleepers may reduce the risks associated with long sleep. Such an intervention will reduce time in bed and total sleep time, which may reduce sleep fragmentation and normalize sleep. While a standardized procedure for such sleep restriction has not yet been developed, one possibility has been proposed by Youngstedt and Kripke[1]. In addition to these examples, the findings highlight the importance of measuring sleep duration (long and short) in patients complaining of sleep disorders, circadian rhythm problems, mood disturbance, anxiety, and other clinical conditions. Simply assessing lack of sleep or general sleep disturbance may be

insufficient. Also, treatments for patients presenting with these conditions may include addressing long sleep.

When is long sleep a life-long trait and when can it be induced?

While some long sleepers report that their sleep pattern has existed for many years, studies of variable-length sleepers show that long sleep can also be induced[30]. Long sleep may be a form of coping with stress[90], and further research into the mechanisms of varying sleep duration will clarify this. It is still currently unknown whether long sleep is generally a trait, what makes a person become a long sleeper when they were previously a short or normal sleeper, and how duration of long sleep pattern may be related to increased mortality risk.

Why is long sleep associated with increased mortality risk?

There are a number of ways in which long sleep may be related to shorter lifespan, such as depression, homeostatic, and photoperiodic disturbance. Also, long sleep may suggest an underlying health condition that may be related to increased mortality risk. Cross-sectional studies that adequately control for these factors need to be conducted and experimental studies need to be undertaken to elucidate causal relationships. Direct mechanism studies may be most appropriate in animal models were potential mechanisms can be more directly manipulated and their consequences more easily followed to fruition. In this manner, the hypotheses proposed here as most promising can be eliminated and/or refined. In undertaking this research, it should be noted that although long sleep is associated with increased mortality risk, these results are based on habitual sleep patterns and it is currently unknown whether reducing sleep time in long sleepers will positively or negatively impact mortality risk. This represents another set of studies that should be conducted.

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PRACTICE POINTS

1. Reported long sleep is associated with increased mortality risk. It is currently unclear whether mortality is associated with long sleep or long time in bed. Nonetheless, there are several testable mechanisms for this relationship that can apply to either marker.

2. Proposed mechanisms for mortality associated with long sleep include: (1) sleep fragmentation, (2) fatigue, (3) immune function, (4) photoperiodic abnormalities, (5) lack of challenge, (6) depression, and (7) an underlying disease process, such as (a) sleep apnea, (b) heart disease, or (c) failing health. The mostly strongly supported mechanisms in the current literature include depression, photoperiodic abnormalities, and health risks.

3. Long sleepers demonstrate some physiological differences from short sleepers. Particularly, long sleepers seem to demonstrate a characteristically different melatonin rhythm, as well as differences in rhythms of temperature, timing and activity. Additionally, long sleepers demonstrate differences in electroencephalographic measurements of sleep.

4. Clinical sleep evaluations should examine long sleep in addition to short sleep. Assessment of sleep quality should be sensitive to the U-shaped relationship between sleep duration and sleep complaints. Screening measures might need to include long sleep in addition to short sleep in their assessments.

RESEARCH AGENDA

1. Cross-sectional studies that adequately control for underlying health conditions, such as coronary disease, sleep apnea and depression, may further explain the roles of these factors in mortality.

2. Cross-sectional studies that sufficiently characterize "long-sleepers" will better ascertain to what extent "long sleep" describes time in bed and to what extent it describes sleep duration.

3. Experimental studies examining whether reducing sleep time in habitually long sleepers will positively or negatively impact mortality risk may provide a preventative measure to counteract the risks. The initial studies may be best conducted in animal models by: (a) shortening sleep in the longest sleeping members of a given species (to the species mean, for example), and/or (b) by shortening sleep in a long sleeping species to the length seen in the wild type species.

4. Experimental studies should further explore genetic and physiological differences among long, normal-length and short sleepers.

5. Experimental studies should examine relationships among the proposed mechanisms of mortality risk in long sleep, with particular emphasis on depression, immune functioning, homeostatic and photoperiodic characteristics, and medical history in long sleepers.

6. Clinical studies should examine the utility of adding long sleep to screening measures and indications of sleep disturbance.

7. Studies of the causes of long sleep (genetic and environmental) may further aid in the understanding of the mortality relationship.

8. Studies should examine the relationship between sleep duration and mortality across the lifespan, as risks may vary with age.

9. Mutant short and long sleepers in both rodents and flies (which may be preferred due to a shorter life span) should be examined to see if 1) there is a mortality difference, and 2) there are physiological or genetic markers associated with sleep duration and mortality. Subsequently, these additional markers should be explored for causal relationships with mortality.

Figure 1.
Hypothesized mechanisms of the relationship between long sleep and mortality.
Mortality associated with long sleep may be caused by a number of factors, which may be
related to each other.

Table 1

Epidemiologic association of self-reported long sleep with mortality

Citation	Population studied (1. N, 2. Ages, 3. Years Study Duration)	Number of covariates besides age	Age-adjusted mortality rates RR: Relative Risk (95% confidence interval, when available)	Mortality rates adjusted for all covariates RR: Relative Risk (95% confidence interval)
Amagai et al.[18]	1 11,325 (4419 men, 6906 women) 2 19–93 3 9	8	RR (ref 7–7.9 h; men, women) <6h: 2.3 (1.3, 3.9), 0.8 (0.3, 2.2) 6–6.9h: 1.1 (0.7, 1.8), 1.3 (0.8, 2.0) 7–7.9h: 1.0, 1.0 8–8.9h: 1.0 (0.7, 1.3), 1.0 (0.7, 1.5) >=9h: 1.3 (0.9, 1.8), 1.5 (1.0, 2.3)	RR (ref 7–7.9 h; men, women) <6h: 2.4 (1.3, 4.2), 0.7 (0.2, 2.3) 6–6.9h: 1.1 (0.7, 1.8), 1.3 (0.8, 2.1) 7–7.9h: 1.0, 1.0 8–8.9h: 0.9 (0.6, 1.2), 1.1 (0.8, 1.6) >=9h: 1.1 (0.8, 1.6), 1.5 (1.0, 2.4)
Ayas et al.[42]	1 71,617 women 2 45–65 3 10	15	Fatal Coronary Heart Disease RR (Ref 8 h) <=5h: 1.67 (1.02–2.74) 6h: 1.05 (0.75–1.46) 7h: 0.80 (0.58–1.09) 8h: 1.00 >=9h: 1.71 (1.05–2.77)	Fatal Coronary Heart Disease RR (Ref 8 h) <=5h: 1.12 (0.68–1.84) 6h: 0.91 (0.65–1.28) 7h: 0.83 (0.60–1.14) 8h: 1.00 >=9h: 1.45 (0.89–2.36)
Breslow & Enstrom[19]	1 6,928 2 >18 3 9.5	0	Mortality Rates 　　　　Women　Men <=6h: 0.097　0.156 7h: 0.068　0.115 8h: 0.084　0.111 >=9h: 0.104　0.139	
Burazeri et al.[9]	1 1,842 (1,001 women, 841 men) 2 >=50 3 9–11	24	All-Cause Mortality Night Sleep RR (ref 6h) Women　Men <6h: 1.00　1.00 6–8h: 0.82　1.21 (0.57–1.17)　(0.83–1.76) >8h: 0.95　1.86 (0.63–1.42)　(1.19–2.91) Night Sleep + Napping RR (ref 6h) Women　Men <6h: 1.00　1.00	All-Cause Mortality Night Sleep RR (ref 6h) Women　Men <6h: 1.00　1.00 6–8h: 0.80　1.25 (0.54–1.17)　(0.83–1.87) >8h: 1.08　1.91 (0.70–1.66)　(1.16–3.13) Night Sleep + Napping RR (ref 6h) Women　Men <6h: 1.00　1.00

Citation	Population studied (1. N, 2. Ages, 3. Years Study Duration)		Number of covariates besides age	Age-adjusted mortality rates RR: Relative Risk (95% confidence interval, when available)		Mortality rates adjusted for all covariates RR: Relative Risk (95% confidence interval)	
				6–8h: 0.68 (0.46–1.01)	1.03 (0.65–1.64)	6–8h: 0.64 (0.42–0.97)	1.41 (0.83–2.39)
				>8h: 0.84 (0.56–1.27)	1.81 (1.12–2.93)	>8h: 0.80 (0.51–1.24)	2.13 (1.23–3.71)
Chen & Foley[10]	1	10,287	6	With no control for age or other covariates: All-Cause Mortality Mortality Rate (ref: 6–9h) Subjects >=60 years <6h or >9h = 20% 6–9hr = 14.7% Subjects <60 yrs: No Significant Effect		No significant effect. Data not reported	
	2	32–86					
	3	4					
Gale & Martyn[11]	1	1,299	9	All-Cause Mortality. RR (ref 9h) <=7h: 1.1 (0.8–1.4) 8h: 0.9 (0.7–1.0) 9h: 1.0 10h: 1.2 (1.0–1.4) 11h: 1.5 (1.2–1.8) >=12h: 1.9 (1.4–2.5)		All-Cause Mortality RR (ref 9h) <=7h: 1.0 (0.7–1.4) 8h: 0.8 (0.7–1.0) 9h: 1.0 10 h: 1.2 (1.0–1.4) 11 h: 1.3 (1.0–1.7) >=12h: 1.7 (1.2–2.5)	
	2	>=65					
	3	23					
Gottlieb et al.[12]	1	4,541 (2,532 women, 2009 men)	5	Not Reported		All-Cause Mortality RR (ref 7–8h) Women / Men <6=1.70 / 1.40 6=1.20 / 0.80 7–8=1.00 / 1.00 9=0.95 / 1.30 >9=1.8 / 1.5	
	2	not reported					
	3	14					
Hammond[6]	1	1,064,004	0	Death rate per 100 people lowest for 7 hrs in 17/18 age/gender groups			
	2	<30 to >=80					
	3	3					
Heslop et al.[23]	1	3030 (442 women, 2588 men)	10	Cardiovascular deaths		Cardiovascular deaths	

Citation	Population studied (1. N, 2. Ages, 3. Years Study Duration)		Number of covariates besides age	Age-adjusted mortality rates RR: Relative Risk (95% confidence interval, when available)		Mortality rates adjusted for all covariates RR: Relative Risk (95% confidence interval)	
	2	Men <65, women <60		RR (ref "always 7–8h")		RR (ref "always 7–8h")	
	3	25		Women	Men	Women	Men
				Always <7h: 2.32 (0.99, 5.42)	1.33 (1.01,1.76)	Always <7h: 2.30 (0.94,5.60)	1.19 (0.90,1.58)
				Always >8h: 1.67 (0.22,1.25)	0.73 (0.35,15.5)	Always >8h: 1.53 (0.20,1.18)	0.63 (0.30,13.4)
				Increase over time: 0.27 (0.04,2.00)	0.82 (0.59,1.13)	Increase over time: 0.34 (0.04,2.56)	0.72(0.52,1.01)
				Decrease over time: 1.15 (0.47,2.84)	1.07 (0.82,1.39)	Decrease over time: 1.34 (0.53,1.25)	0.88 (0.67,1.16)
				All-cause mortality RR (ref 7–8h)		All-cause mortality RR (ref 7–8h)	
				Always <7h: 1.99 (1.16,3.41)	1.30 (1.06,1.61)	Always <7h: 1.73 (0.99,3.03)	1.15(0.99,1.42)
				Always >8h: 0.60 (0.08,4.37)	1.04 (0.65,1.66)	Always >8h: 0.58 (0.08,4.22)	0.91 (0.57,1.46)
				Increase over time: 0.49 (0.20,1.23)	0.91 (0.72,1.15)	Increase over time: 0.47 (0.19,1.20)	0.81 (0.64,1.02)
				Decrease over time: 0.91 (0.50,1.66)	1.17 (0.46,1.42)	Decrease over time: 0.98 (0.53,1.79)	1.00 (0.82,1.21)
Huppert & Whittington[13]	**1**	6,096	Not reported	All-Cause Mortality RR (Ref 6–9h)			
	2	>=18		Women: NS, data not reported			
	3	7		Men <6h: 0.95; 6–9h: 1.0; >9h: 2.25			
Kaplan[19]	**1**	6,928	6			All-Cause Mortality RR (ref 7–8h)	
	2	60–94				Sleeping >7–8 hr or <7–8 hrs	
	3	17				Ages 38–49=1.23 (0.88–1.70)	
						Ages 50–59=1.44 (1.08–1.91)	
						Ages 60–69=0.95 (0.73–1.24)	
						Ages >=70=1.05 (0.85–1.29)	
						7–8 hrs per night not associated with different risk ratio vs not sleeping 7– 8 hrs across all groups.	

Citation	Population studied (1. N, 2. Ages, 3. Years Study Duration)			Number of covariates besides age	Age-adjusted mortality rates RR: Relative Risk (95% confidence interval, when available)			Mortality rates adjusted for all covariates RR: Relative Risk (95% confidence interval)		
Kojima et al.[14]	1	5,322		10	All-Cause Mortality RR (ref 7–8.9h)			All-Cause Mortality RR (ref:7–8.9h)		
	2	20–67				Women	Men		Women	Men
	3	12				<6.9h: 0.92	1.90		<6.9h: 0.90	1.93
						(0.53–1.62)	(1.10–3.29)		(0.50–1 61)	(1.12–3.35)
						7–8.9h: 1.00	1.00		7–8.9h: 1.00	1.00
						9–9.9h: 1.10	1.13		9–9.9h: 1.07	1.15
						(0.61–2.00)	(0.73–1.74)		(0.58–1.95)	(0.74–1.77)
						>=10h: 0.42	1.94		>=10h: 0.40	1.77
						(0.06–3.02)	(1.01–3.76)		(0.06–2.92)	(0.88–3.54)
Kripke et al.[21]	1	823,065		5				All-Cause Mortality RR (ref 7–7.9h)		
	2	30 to >90 yrs							Women	Men
	3	6							<4h: 1.48	2.80
									4–4.9h: 1.40	1.59
									5–5.9h: 1.20	1.38
									6–6.9h: 1.13	1.11
									7–7.9h: 1.00	1.00
									8–8.9h: 1.13	1.10
									9–9.9h: 1.27	1.29
									>=10 h: 1.82	1.77
								Similar trends for Mortality Associated with CHD, stroke, cancer, suicide. Similar trends across multiple age-groups		
Kripke et al.[5]	1	1,116,936 (636,095 women, 480,841 men)		32				All-Cause Mortality RR (ref 7 hr)		
	2	30–102							Women	Men
	3	6							2.5–3.4h: 1.33	1.19
									(1.08–1.64)	(0.96–1.47)
									3.5–4.4h: 1.11	1.17

Citation	Population studied (1. N, 2. Ages, 3. Years Study Duration)	Number of covariates besides age	Age-adjusted mortality rates RR: Relative Risk (95% confidence interval, when available)	Mortality rates adjusted for all covariates RR: Relative Risk (95% confidence interval)
			(1.01–1.22)	(1.06–1.28)
			4.5–5.4h: 1.07	1.11
			(1.01–1.13)	(1.05–1.18)
			5.5–6.4h: 1.07	1.08
			(1.03–1.11)	(1.04–1.11)
			6.5–7.4h: 1.00	1.00
			7.5–8.4h: 1.13	1.12
			(1.09–1.16)	(1.09–1.15)
			8.5–9.4h: 1.23	1.34
			(1.17–1.28)	(1.28–1.40)
			>=10 h: 1.41	1.34
			(1.34–1.50)	(1.28–1.40)
Mallon et al.[24]	1 1,870 (964 women, 906 men) 2 45–65 3 12	17	All-Cause Mortality RR (ref 6–8h) Women Men <6h: 1.0 1.1 (0.6–1.8) (0.6–7.0) 6–8h: 1.0 1.0 >8h: 1.3 2.0 (0.6–2.6) (1.2–3.2)	"Not Significant" Data Not Reported Similar patterns for mortality associated with coronary artery disease, cancer, and all other causes.
Patel et al.[15]	1 82,969 women 2 40–65 3 10	11	All-Cause Mortality RR (ref 7h) <5h: 1.41 (1.25–1.58) 6h: 1.07 (1.00–1.15) 7h: 1.00 8h: 1.18 (1.10–1.26) >=9h: 1.72 (1.55–1.91)	All-Cause Mortality RR (ref 7h) <5h: 1.08 (0.96–1.22) 6h: 0.99 (0.92–1.06) 7h: 1.00 8h: 1.11 (1.03–1.19) >=9h: 1.40 (1.25–1.55)
ollak et al.[16]	1 1,855 (1,510 women, 783 men) 2 65–98 3 3.5	10	All-Cause Mortality Mortality Rate (per 100) Women Men <=4h: 19 24 5h: 7 25	Cox regression models not significant for Males or females. Data not available for specific hrs of sleep.

Citation	Population studied (1. N, 2. Ages, 3. Years Study Duration)	Number of covariates besides age	Age-adjusted mortality rates RR: Relative Risk (95% confidence interval, when available)	Mortality rates adjusted for all covariates RR: Relative Risk (95% confidence interval)
			6h: 14 16 7h: 8 16.5 8h: 13 22 >=9h: 18 30	
Qureshi et al.[17]	1. 7,844 2. 32 to >72 3. 10	9		All-Cause Mortality. RR (ref 6–8h) 6–8h: 1.0 >8h: 1.3 (1.1–1.5) Risk of Stroke RR (ref 6–8 hr) 6–8h: 1.0 >8h: 1.9 (1.2–3.1)
Tamakoshi & Ohno[8]	1. 104,298 (60,276 women, 44,022 men) 2. 40–79 3. 10	14	All-Cause Mortality RR (ref 7–7.9 hr) Women Men <7h: 1.08 1.13 (0.99–1.17) (1.04–1.22) 7–7.9h: 1.00 1.00 8–8.9h: 1.24 1.13 (1.14–1.34) (1.06–1.20) >=9h: 1.64 1.52 (1.49–1.80) (1.42–1.64)	All-Cause Mortality RR (ref 7–7.9 hr) Women Men <7h: 1.14 1.12 (0.99–1.32) (0.98–1.28) 7–7.9h: 1.00 1.00 8–8.9h: 1.20 1.18 (1.05–1.38) (1.06–1.31) >=9h: 1.62 1.49 (1.36–1.92) (1.31–1.70)
Wingard et al.[22]	1. 6,928 (2,496 women, 2,229 men) 2. 30–69 3. 9	14	All-Cause Mortality % Mortality (ref 7–8h) Women Men 7–8 h: 5.5% 8.2% <=6 h or >=9: 8.8% 13.6%	In multiple logistic analysis with all covariates, sleep had significant independent association with mortality.

This table is adapted from the summary table previously published by Youngstedt and Kripke[1].