Published in final edited form as:
*Sleep Med Rev*. 2010 August ; 14(4): 239–247. doi:10.1016/j.smrv.2009.08.001.

# Problems Associated with Short Sleep: Bridging the Gap between Laboratory and Epidemiological Studies

**Michael A. Grandner, Ph.D.**[1,*], **Nirav P. Patel, M.D.**[1,2], **Philip R. Gehrman, PhD.**[1,3], **Michael L. Perlis, Ph.D.**[3], and **Allan I. Pack, M.B., Ch.B., Ph.D.**[1]

[1] Center for Sleep and Respiratory Neurobiology, Division of Sleep Medicine, Department of Medicine, University of Pennsylvania, Philadelphia, PA

[2] Division of Pulmonary, Allergy, and Critical Care Medicine, University of Pennsylvania, Philadelphia, PA

[3] Department of Psychiatry, University of Pennsylvania, Philadelphia, PA

## Abstract

Existing data from laboratory studies suggest a number of negative consequences of acute reductions in sleep time. Also, epidemiological data suggest links between shorter self-reported sleep duration and negative health outcomes. These bodies of work are growing, revealing several key points of convergence and opportunities for future exploration. In addition, they begin to highlight possible problems experienced by "short sleepers," who sleep approximately six hours or less per night. While it is likely that this group is heterogeneous, comprised both of individuals with less need for sleep and those not sleeping enough, the laboratory and epidemiological findings point towards directions that can be more fully explored in verified short sleepers. This paper discusses problems associated with the terminology used to describe "short sleep," summarizes laboratory studies exploring neurobehavioral performance, metabolism and obesity, and psychological health and epidemiological studies exploring mortality risk, obesity and metabolism, cardiovascular disease, and general health/psychosocial stress, describes studies of verified short sleepers and explores areas of convergence, laying out possible future directions.

### Keywords

Sleep; Sleep Deprivation; Epidemiology; Short Sleepers; Neurobehavioral Performance; Cardiovascular; Metabolism; Obesity; Psychological Health; Mortality

## Introduction

There is a substantial debate, both within the scientific community and society at large, about the minimum amount of sleep required for health and well-being. Central to this debate is a concern about the long-term effects of the shortening of sleep opportunity (sleep curtailment). There are no studies that directly address whether sleep curtailment is the same as sleep insufficiency. That is, is it the case that reduced sleep opportunity leads to reduced sleep time?

CORRESPONDING AUTHOR: Michael A. Grandner, Ph.D., University of Pennsylvania Center for Sleep and Respiratory Neurobiology, 125 S. 31st Street, Philadelphia, PA 19104, Phone: 215-746-4815, Fax: 215 -746-4814, grandner@upenn.edu.

While this seems likely, the relationship between changes in sleep opportunity and sleep time are not perfectly linear, as factors such as sleep ability (e.g., insomnia) and sleep homeostasis (e.g., sleep pressure) play a role. Additionally, if sleep time is reduced, is the reduction of sufficient magnitude to lead to negative outcomes? The evidence to suggest that sleep curtailment is associated with negative outcomes comes from two primary sources: (1) studies of acute sleep curtailment in the laboratory setting (sleep deprivation) [1–18], and (2) epidemiological studies relating self-reported sleep to health outcomes [6, 9, 12–13, 19–27].

The goals of his paper are to: (1) Review the existing literature examining "short sleep" using objective, laboratory-based methods, (2) Review the existing literature examining "short sleep" using epidemiological and other self-report methods, (3) Assess the degree to which there is a convergence between these approaches, and (4) Suggest further research required to clarify the underlying issue of whether short sleep is related to negative health outcomes.

## A problem of terms: The lexicon of short sleep

An issue relevant to this discussion is the untangling of the various terms used to describe "short sleep." In the popular press[28] and in the scientific literature[29] apparently synonymous terms (e.g., "short sleep," "insufficient sleep," "sleep loss") are used interchangeably, although these terms have distinct, specific meanings that connote different methods and concepts. For the purposes of this paper, we have outlined working definitions for these and other terms in Table 1.

The question remains: What is "short sleep?" The phrase has been used to refer to all of the various terms in Table 1. It has been used to describe subjective and objective sleep duration, for both chosen and forced sleep opportunities, in both laboratory and home settings. In a way, it can mean all of those things, as, literally, the phrase is ambiguous. It doesn't address how short the sleep is, what the shortness is relative to, and how it was determined. Thus, for scientific purposes, it is a suboptimal descriptor (necessitating the use of other Table 1 terms when they are appropriate).

However, we propose that there is a valid use of the term: To describe habitual sleep duration of <6 hours. Many of the epidemiological studies already use the term this way, and it helps describe subgroups of Short Sleepers in the population. For example, while it is possible that there are true Short Sleepers in the natural environment (those who demonstrate Short Sleep without Sleep Insufficiency), as well as those who demonstrate Short Sleep along with varying levels of Sleep Insufficiency, the existing literature has not specifically explored these groups. A clarification in terms may help build a nosological framework around which some of the heterogeneity among Short Sleepers can be explored.

To better understand what Short Sleepers (true or otherwise) may experience, we look to the existing Laboratory and Self-Report studies and examine their convergence, despite limitations.

## Data from laboratory studies

One of the earliest laboratory studies investigating the role of sleep on performance was carried out almost a century ago by Pieron and colleagues[30], who were searching for biomarkers of sleep in dogs, using sleep deprivation. As the search for such biomarkers continues, the science of studying sleep has matured considerably, growing to explore many other hypotheses and research directions. Current studies employ laboratory-controlled sleep deprivation to examine outcomes associated with sleep loss, which may generalize to short sleep. Three domains in which this has been explored are: neurobehavioral performance, metabolism and obesity, and psychological health.

## Laboratory Studies of Neurobehavioral Performance

Sleep deprivation leads to increased sleepiness. The term "sleepiness" is used to describe a state of increased sleep propensity (likelihood of falling asleep), usually referring to situations in which sleep is not appropriate or desired[31]. This is in contrast with "tiredness," which is a subjective experience similar to fatigue. While they often overlap significantly, they are separate constructs – for example, individuals suffering from insomnia often experience tiredness, yet are not sleepy32. Individuals who experience partial or total sleep deprivation demonstrate increased sleepiness5, 33. This finding has been replicated[2, 5, 34–35] across three methods of objective assessment of sleepiness: the Multiple Sleep Latency Test, Maintenance of Wakefulness Test, and measurements of oculomotor activity[5].

Within the paradigms of sleep deprivation and sleep loss, neurobehavioral performance is one of the most often-measured outcomes4, often operationalized as sustained attention. The dominant measure of sustained attention in paradigms of sleep deprivation is the Psychomotor Vigilance Task4, [36] (PVT). A consistent finding with the PVT is that sustained attention decreases with sleep restriction, and this decrease is exacerbated as sleep opportunity is reduced from 7 to 3 hours[4–5]. Additionally, cumulative deficits across several days are seen when sleep schedules of 3, 4, 5, and 6 hours are maintained4–5. Inferences, though highly important in our understanding of sleep duration and sustained attention, are limited to the subjects and conditions specified in these experiments. PVT performance, which measures one aspect of neuropsychological function, has not been evaluated outside of the laboratory. Other work has demonstrated that sleep deprivation is also associated with deficits in executive function[37], learning[38], and memory[39].

There has been significant discussion of the role of impaired functioning due to sleep loss in auto accidents[40–45]. There is strong evidence that sleepiness is a major factor contributing to automobile accidents[46–48]. Several papers have shown that accidents are related to sleepiness caused by sleep apnea[46, 49–50] and prolonged wakefulness[48, 51–52]. Some evidence also suggests that short sleepers may exhibit more sleepiness than average[53–54], but currently there is no data to support the claim that short sleepers are more likely to experience auto accidents[55].

## Laboratory Studies of Metabolism

The first study to evaluate metabolic consequences of sleep loss was published over 15 years ago[56] in 8 healthy subjects. This study found that sleep deprivation had a negative impact on glucose tolerance and insulin resistance, but problems resolved with recovery sleep. Other classic studies found that rats who were continually deprived of sleep became hyperphagic, even though they demonstrated a depletion of energy reserves[57]. More recent studies have expanded our knowledge about the relationship between sleep and metabolism.

Short sleep is associated with impaired glucose tolerance and insulin resistance. In a landmark study, Spiegel and colleagues[14] studied the effects of sleep curtailment to 4 hours for 6 days, with baseline and recovery sleep opportunities. Compared to baseline, glucose tolerance decreased by 40%, glucose disposal decreased by 30%, acute insulin response decreased by 30%, and insulin sensitivity trended lower. Additionally, the area under the Homeostasis Model Assessment (HOMA) curve was 50% higher than when rested. This study had several limitations including small sample size, an order effect, and no control group. Nonetheless, it engendered significant interest in the field of sleep and metabolism and was followed by numerous studies. In a crossover design[13] in which groups were allocated to receive 4 hours or 10 hours of sleep opportunity for 2 days each Spiegel and associates were able to replicate their earlier findings.

Other hormone systems are affected as well. Studies of sleep deprivation have also examined the hormones leptin (originates in adipose tissue and regulates appetite and energy metabolism) and ghrelin (released from th stomach and pancreas and stimulates appetite). These are hormones that, among other functions, regulate hunger and satiety[58–59]. Total sleep deprivation has been shown to result in a decrease in the amplitude of the leptin rhythm60, and a decrease in the normal rise in ghrelin associated with sleep61. In one study[62], leptin decreased and its circadian phase was shifted earlier by 2 hours compared to baseline. A later study[63] replicated these findings in sleep deprivation and extension. Changes in leptin and ghrelin may be partially responsible for changes in hunger and appetite[63], as previous studies have shown that sleep deprivation is associated with cravings for more calorie-dense foods[63] and habitual shorter actigraphic sleep is associated with greater fat intake[64].

### Laboratory Studies of Cardiovascular Health

While research in this area is relatively limited, there have been several sleep deprivation studies that have linked less sleep with poorer cardiovascular functioning. If there is a link between sleep deprivation and cardiovascular disease, it may be through CRP (described above) and other inflammatory markers[65]. Coronary circulation may also be affected66. Additionally, sleep deprivation results in blood pressure and sympathetic changes67–68.

### Laboratory Studies of Psychological Health

Sleep deprivation has effects on several domains of psychological health, including stress, mood and socialization. An increased stress response has been shown, including increased basal activity of neuroendocrine stress systems, elevations of the sympathetic nervous system, altered hypothalamic-pituitary-adrenal axis function, and increased stress reactivity[16]. Effects are modest, but chronic exposure may have gradual but cumulative effects. Exploring this issue further, Roman and colleagues[69] found that sleep deprivation may lead to changes in certain receptor systems that are either directly or indirectly related to stress response.

In a study by Haack and Mullington[18], “optimism-sociability” declined 15% over consecutive days of sleep loss; additionally, a 3% (statistically significant) increase in bodily discomfort was reported, due to significant increases of generalized body pain, back pain, and stomach pain. These findings support other recent work showing a significant relationship between sleep deprivation and mood dysregulation[17]. These laboratory studies suggest that sleep loss is associated with increased depressive symptoms and stress. However, these findings need to be replicated in studies of verified short sleepers to ascertain whether they are the result of acute sleep deprivation or associated with habitual short sleep.

There is some recent evidence that induced sleep deprivation is associated with impaired decision-making. This may have to do with impaired executive functioning (described above). One study investigated risky decision making following sleep deprivation[70] and found that not only was sleep deprivation associated with increased risky behavior, but an effect was seen for age, such that older subjects were more likely to demonstrate a greater effect. Some studies have reported similar effects71–73, while others have found mixed results74.

## Data from studies of self-reported short sleep

Epidemiological studies remain a valuable method for exploring relationships between sleep and health in the general population. These studies allow for the measurement of sleep and related health variables at a population level, elucidating relationships that may be too subtle to detect in laboratory studies but nonetheless valuable to society[75]. The primary domains addressed by epidemiological studies that considered sleep are: mortality risk, obesity and metabolism, cardiovascular disease, and general health/psychosocial stress.

## Epidemiological Studies of Mortality Risk

Over 40 years of epidemiological studies have examined the association between habitual sleep duration and risk of mortality[19, 76]. These studies, spanning several decades and continents, and including millions of study participants, have replicated the pattern that "short sleep" and "long sleep" (with varied definitions across studies) are associated with increased mortality relative to those in the normative group – usually this consists of those sleeping 7–8 hours. In addition, studies comparing several sleep duration groups have generally found that the further the deviation from the normative range, the greater the increase in mortality risk[19]. A complete review of these studies is published elsewhere77.

A recent meta-analysis[78] combined the results of these studies, and found that pooled relative risk (RR) for all-cause mortality for "short sleep" was 1.10 (95% CI 1.06, 1.15), and RR for all-cause mortality for "long sleep" was 1.23 (95% CI 1.16, 1.30). When cause-specific mortality was explored, the RR for cardiovascular-related was 1.06 (95% CI 0.94, 1.30) and cancer-related RR at 0.99 (95% CI 0.88, 1.13) in short sleep and cardiovascular RR was 1.38 (95% CI 1.13, 1.69) and cancer RR was 1.21 (95% CI 1.11, 1.32) for long sleep. This meta-analysis supports the claim that short sleep (which was defined variously in these studies but usually was defined as <7 or <6 hours) is associated with increased all-cause mortality risk, echoing the theme of many of the epidemiological studies. However, it should be noted that the RR for "long sleep" (usually defined as >8 or ≥9 hours in these studies) reflects a much greater associated risk than that for short sleep. Even if long sleep is associated with greater risk, the importance of the relationship between short sleep and mortality risk is not diminished. It has been asserted that the risks associated with long and short sleep represent two distinct phenomena which are to be considered separately[77, 79]. Thus, these results demonstrate that short sleep is a predictor of mortality and that long sleep is also worthy of study regarding mortality risk.

## Epidemiological Studies of Obesity and Metabolism

Many studies have shown a dose-response relationship of short sleep duration and obesity and metabolic consequences across all age groups[9–10, 21, 25, 80]. Cross-sectional studies have been conducted in adults from Canada[23–24], France[81], Germany[82], Japan[83], Spain[84], the UK[85], and the USA[86–88], showing significant associations between short sleep and obesity.

Recent large studies have found several interesting relationships between self-reported short sleep duration and BMI. Increased BMI was associated with decreased sleep durations among men, but a U-shaped relationship was seen in women[86]. Also, subjective time in bed was associated with BMI in a U-shaped manner after adjusting for age and sex, with the minimum BMI observed at 7.7 hours/night[87–88]. Longitudinal analysis observed that sleep durations <7 hours were associated with increased risk of weight gain. In another study, data collected over 13 years showed that the odds ratio for sleep duration predicting obesity was 0.50, such that every extra hour increase of sleep duration was associated with a 50% reduction in risk of obesity[89]. This finding has also extended from urban into rural samples[90]. Studies outside the US have replicated these findings as well, in France[81] and Canada[24–25].

## Epidemiological Studies of Cardiovascular Health

Recent data suggests that individuals who reported less than 6 hours of sleep were at increased risk for hypertension[91–92], which found that those reporting habitual sleep of ≤5 hours were at greatest risk of incident hypertension; this relationship was attenuated in those reporting 6 hours. Other recent data [93] was able to replicate this finding in women, but not in men. Additionally, women who sleep <7 hours were at increased risk of stroke[94], though the relationship for long sleep was stronger. Regarding myocardial infarction, short sleepers were at significantly increased risk[95]. This relationship was notably stronger for women than men.

### Studies of Subjective Short Sleep and General Health

Short sleep is associated with poor health in general. “Short” sleep (self-reported 6–7 hours or <6 hours) has been associated with lower reports of self-rated overall health than “normal” (7–8 hour) or “long” (8–10 or >10 hour) sleep in young adults[27]. Short sleep has also been associated with low socioeconomic status96–97, as well as living in an urban environment97. These studies, however, did not examine whether these risk factors were associated with decreased health-related quality of life.

Also, short sleepers have been found to demonstrate shorter melatonin rhythms and more morning activity than long sleepers[19]. While implications of these effects are unclear, underlying circadian rhythm differences may suggest genetic influence. Additionally, self-reported short sleepers report higher rates of difficulty falling asleep, waking too early, waking during the night, waking unrefreshed, and feeling sleepy during the daytime than self-reported 7–8 hour sleepers[98]. A study[99] found that short sleep was associated with drinking six cups or more of coffee per day, taking sleeping medications, difficulty initiating sleep, disrupted sleep, early morning awakening. Also, short sleep was associated with self-reported “Sleeping disorders or insomnia,” “difficulties in getting sleep without sleeping medicines,” “overexertion or exhaustion” and “being awake during the night.” It should also be noted that self-reported short sleepers usually includes people purposefully curtailing their sleep as well as individuals with insomnia who want to sleep more but are not able to do so. It may be the case that a large portion of self-described “short sleepers” are people with insomnia and/or are taking sleeping medications, which may play a role in health outcomes. Currently, there are no studies that have explored the heterogeneity among short sleepers to discern which are true short sleepers, which are experiencing insufficient sleep and which have sleep disorders.

### Studies of Subjective Short Sleep and Psychological Health

Short sleep has been associated with increased anxiety and a coping style characterized as having more of an external locus of control than long sleepers100. This means that short sleepers are more likely to look to external explanations for successes and failures. For example, they will be more likely to blame others for problems, and judge their successes based on the impressions of others. On the Ways of Coping Questionnaire, short sleepers reported higher “Confrontive,” “Self-controlling,” “Accepting responsibility,” and “Positive reappraisal” scores than long sleepers[101]. It is possible that this represents an underlying personality style that is linked to short sleep and related to the experience (or need) of less REM sleep[102], but these effects have not been sufficiently explored in recent studies.

Self-reported short sleep has been associated with increased anxiety[103] and hypomania[104]. In a study of reported stressful events, Sexton-Radek[105] found that short sleepers had more consistent responses to stress. This is consistent with an external locus of control. These studies suggest that preferred sleep schedules are associated with different coping styles. However, these studies did not verify whether the “short sleepers” studied did actually sleep less.

## Data from verified short sleepers

Several studies have investigated health outcomes associated with sleep duration utilizing objective methods (polysomnography and actigraphy) to estimate sleep in subjects reporting habitual short sleep. While this literature is limited, it may clarify some of the findings from laboratory and epidemiological studies.

## Sleep Homeostasis and Circadian Rhythms in Short Sleepers

In early studies of sleep duration[106–107], short sleepers were found to demonstrate more consolidated sleep, less REM (but increased REM density[108]), stage 1 and stage 2 sleep, and equivalent or more slow wave sleep when compared to long sleepers.

There is some evidence that sleep homeostasis and circadian rhythms are fundamentally different in this group. Aeschbach and colleagues[53–54, 109] explored these questions in several studies. First, they found that short sleepers and long sleepers show differences in recovery from sleep deprivation, suggesting a difference in sleep homeostasis[54]. During baseline nights, short sleepers had a shorter sleep onset latency and higher sleep efficiency than the long sleepers. Additional differences appeared when the groups were compared after truncating all sleep recordings to the shortest sleep time, looking at the "longest common sleep interval." When this is done, short sleepers still showed decreased sleep latency, but they also showed increased SWS, suggesting greater homeostatic sleep pressure in this group. Additionally, more REM sleep was seen in the short sleepers using this technique, suggesting chronic sleep restriction (resulting in REM rebound) or differences in circadian phase (resulting in earlier REM onset). Following sleep restriction, the long sleepers demonstrated a greater degree of impact on sleep. This landmark study was extended with later findings supporting the idea that short sleepers live under higher homeostatic sleep pressure, but they are much more adept at tolerating that pressure than long sleepers[53].

This group also examined whether short sleepers exhibit a shorter "biological night" than long sleepers. This study evaluated 10 long sleepers and 9 short sleepers in a paradigm of constant environmental conditions, without sleep, so that the expressions of endogenous biological rhythms could be assessed. This study specifically examined aspects of various circadian markers, including the melatonin, cortisol, and body temperature rhythms. Short sleepers demonstrated a significantly shorter duration of high-level melatonin secretion, cortisol secretion and low body temperature. Also, the well-established co-occurrence of the peak of cortisol secretion with the time of habitual awakening from sleep occurred 2.5 hours earlier in the short sleepers. The authors note that higher homeostatic pressure load may be an explanation of performance deficits seen in sleep deprivation, though it is unknown how this may be present in habitual short sleepers. These findings, as well as those by the earlier studies, suggest that the processes governing sleep homeostasis and circadian rhythms may be altered in habitual short sleepers.

## Sleep Debt in Short Sleepers

Habitual short sleepers may be sleep deprived. In a recent study[110] individuals recorded habitual sleep duration, then spent 3 nights in the laboratory, and were given as much sleep opportunity as they would take. This study examined 17 individuals of varying sleep lengths, and found that throughout the 3 nights in the laboratory, all of the participants slept longer than their habitual sleep patterns indicated, except for some of those with the longest sleep durations who, by the second or third night, were back to (or below) their habitual durations. This study included only 2 individuals with habitual sleep durations of 6 hours or less. When these individuals were given maximal sleep opportunity for three consecutive nights, they both slept for >10 hours all three nights – on the third night, these two subjects slept approximately 12 and 14 hours. This suggests that habitual sleep might harbor significant amounts of sleep debt, even in self-reported short sleepers[111]. However, it should be noted that this was a very small study.

## Coronary Artery Calcification

A recent analysis of the data from the CARDIA study[112], which obtained actigraphic measurements of habitual sleep, found that shorter sleep was associated with increased

coronary artery calcification. In a linear regression analysis, each increase of 1 hour of sleep duration was associated with an OR of 0.67 (95% CI: 0.49–0.91). While a U-shaped relationship was not explored, these data suggest a possible mechanism through which sleep duration may be responsible for increased mortality risk.

### Metabolic Disruption in Short Sleepers

The Wisconsin Sleep Cohort Study utilized sleep diaries, polysomnography and blood samples to examine relationships between sleep and health. Sleep time, when measured by polysomnography, was negatively associated with ghrelin levels (less sleep was associated with more ghrelin). Additionally, when sleep was measured by sleep diary, but not polysomnography, more sleep time was associated with increased levels of leptin[113]. All of these analyses adjusted for BMI. Thus, self-reports of less sleep was associated with decreased leptin (which may result in decreased satiety) and less polysomnographic sleep was associated with more ghrelin (which may result in increased hunger).

### Diet of Short Sleepers

There has been one study of diet associated with both subjective and objective habitual sleep duration114. This study examined a subset of women enrolled in the Women's Health Initiative (WHI), a large, multisite trial of health in postmenopausal women. Sleep and circadian rhythms were investigated in 459 women by collecting 1 week of sleep diaries and actigraphic recordings, and a sleep questionnaire (sleep recordings described previously115). Dietary data were based on the WHI Food Frequency Questionnaire116 that was completed as part of involvement with the WHI and analyzed by WHI nutritionists. Partial correlations adjusted for age, income, education, physical activity, BMI, and total gram amount of diet. Actigraphic total sleep time was significantly and negatively associated with total fat, monounsaturated fat, trans fat, saturated fat, polyunsaturated fat, total calories, gamma-tocopherol (a form of vitamin E, which is ingested almost exclusively in fats), cholesterol and alpha-tocopherol (also a form of vitamin E). There were no significant correlations with sleep diary total sleep time. It should be noted that the primary sources of vitamin E are the diet are oils. This suggests that there is a relationship between more fat intake (irrespective of type) and shorter habitual sleep duration. It should be noted that since this study is correlational, direction of causation cannot be measured. However, these findings support the hypothesis that metabolic obesity is closely related to sleep duration.

## Convergence Between Experimental and Epidemiological Studies

Compared to the amount of data available from laboratory and epidemiological studies, there has been relatively little investigation of verified short sleepers. However, given the findings from these domains, several patterns emerge:

First, short sleep has been associated increased mortality risk. While the risk associated with long sleep may be greater, self-reported short sleep does carry an increased risk, which is unexplained by cancer and cardiovascular events, even though short sleep may be a risk factor for heart attack and stroke. The mortality relationship is a global issue, as studies from 5 decades and several countries have replicated this finding. Sleep may be directly related to mortality, or, more likely, it may mediate or moderate a relationship involving cardiovascular disease, obesity, metabolic dysregulation, stress, immune dysfunction, psychological health, cancer or coping difficulties. Laboratory results suggest that sleep deprivation is associated with impairments in these domains, and epidemiological studies confirm that short sleepers report impaired overall health as well as a number of cardiovascular and metabolic risk factors that support that this pattern may be seen in habitual short sleep. However, future studies need to

link the laboratory findings and epidemiological results to verified short sleepers, so that the extent of the presence of these risks in this group is understood.

Second, short sleep has been associated with metabolic dysregulation and obesity. Sleep deprivation has been shown to produce short-term changes in a number of endocrine systems, including insulin, glucose, leptin and ghrelin. These differences may persist over the long term, explaining some of the epidemiological finding. Also, short sleepers may be more likely to eat higher-fat foods, which may be causing the obesity repeatedly demonstrated in epidemiological studies and may or may not be driven by endocrine changes.

Third, short sleep has been associated with worse cardiovascular health. While this has primarily been driven by epidemiological studies, with their inherent problems with the measurement of sleep (described below), the evidence suggests that those reporting less sleep are at greater risk of hypertension, stroke and myocardial infarction than those who sleep 7–8 hours. This relationship may be greater in women than men, and may be evident for long sleep as well. This is supported by a number of laboratory studies that suggest that sleep deprivation is associated with heightened blood pressure and sympathetic activity[67–68].

Fourth, short sleep has been associated with impaired neurobehavioral performance and cognitive functioning. These results have been primarily explored in the context of sleep deprivation studies and have not been replicated in naturalistic settings. It is unclear whether performance deficits associated with short-term sleep deprivation describe the experience of habitual short sleepers. These results are supported by epidemiological findings, which suggest that short sleepers report more sleep disturbance, including daytime sleepiness[98].

Fifth, short sleep has been associated with psychological/psychiatric disturbances and poor general health. Sleep deprivation studies show that at least short term neurophysiological changes indicative of stress and depressive symptoms result from sleep deprivation. Studies of self-reported short sleepers mirror these findings, showing that short sleepers exhibit more risk factors for stress and depression, as well as characteristic coping styles.

## Problems with laboratory and epidemiological studies

Laboratory studies have several important limitations. First, these studies usually involve very small sample sizes. Many of these studies were of 10 individuals per group or less. Second, these studies do not study habitual short sleepers. Third, these studies are performed in an artificial setting. In reality, sleep is subject to socio-ecological influences at different levels[77]. For example, caffeine, alcohol, smoking, children, pets or environmental noise may disturb sleep and some may not be present or allowed in a laboratory setting. Work and school schedules may play less of a role in the laboratory, and the experience of being with a typical bed partner and being exposed to his/her routines may influence sleep. Additionally, laboratory measures of sleep usually involve polysomnography, a method that requires equipment that may itself be difficult to adapt to for a night of sleep[117]. While this is most widely-known as the “first night effect,”[118] the act of measuring sleep may disrupt sleep.

Epidemiological studies have also linked short sleep duration with several important health outcomes. However, there are a number of limitations that should be noted. First, inconsistency regarding the definition of “short sleep” reduces the generalizability of the conclusions across studies. Second, since these studies are primarily epidemiological, they capture self-reported measures of sleep duration, which may better reflect time in bed rather than actual sleep time[119]. Third, subjective and objective measures of sleep duration are often discrepant [120], and both measures inherently contain error in their estimation. Fourth, these studies could not accurately describe how risks associated with sleep duration vary across the lifespan. While age was often used as a covariate, it is likely that the mechanisms by which sleep duration

increases health risks have varying effects at different stages of life. Finally, these large studies fail to adequately measure the extent to which those reporting short sleep do so because of less biological need for sleep, rather than a deprivation of sleep[19].

### Future Direction: Investigate Short Sleepers

There is a large volume of research on the negative effects of shorter-term experimental sleep deprivation, and a growing public need for increased understanding of the role of sleep duration in several domains, including the prevention of disease [75], driving accidents [121] and daytime functioning [6]. This literature is extremely valuable as it has elucidated, in controlled environments, maladaptive processes that are seemingly caused by insufficient sleep in healthy subjects. However, it is difficult to extrapolate these findings to populations at large. In this regard, a dearth of literature exists to help us understand the effects of real-world shorter sleep durations.

Important differences exist between the laboratory-based studies and the epidemiologic and translational research needed to answer this important societal question. First, shorter sleep attainment in society is, to a large degree chosen, not imposed except in individuals with insomnia. Second, the exposure to shorter sleep may vary across days, months and years so the snapshot view obtained from most research studies may not be indicative of patterns over time. Third, and related, the effects of the exposure may occur and surface many months or years later. Thus, while the available experimental data are valuable for the generation of hypotheses regarding short sleep as it is experienced by members of the population, they do not sufficiently describe this group and research studying potential differences between short sleepers and sleep-deprived healthy adults needs to be undertaken.

There is a great need to investigate short sleepers, rather than “normal” sleepers who are deprived of sleep within a research setting – they may be at increased risk for a number of negative health outcomes, they comprise a large (and possibly growing) segment of the population, and little research has been devoted to this group. There are few known studies that have studied the sleep of habitual short sleepers and have investigated the performance, metabolic and biopsychosocial (e.g., mood, anxiety, functioning, behavior) characteristics of this group, relative to normative (7–8 hour) sleepers and relative to the sleep-deprived groups investigated in laboratory studies[111].

## Considerations for future studies in short sleepers

### Individual Differences in Habitual Short Sleepers

Not all short sleepers are the same. Recent data suggests that there are a number of reasons people follow a shorter sleep schedule; some believe that this schedule represents their natural way of functioning, and others trade sleep for time spent doing other activities; usually, sleep is traded for commute time and work[122]. Short sleepers are a heterogeneous group, comprising those that perceive impairment and those that do not, as well as those who believe that they are natural short sleepers, and those who choose this schedule to meet other demands. No previous studies have examined these characteristics of short sleepers, relative to objectively-estimated sleep. A future study might investigate who self-identifies as a short, normal and long sleeper, to validate the cutoffs suggested by the literature.

Some people are more sensitive to sleep loss. An important consideration is that sleep loss increases cognitive variability within subjects, such that certain tasks are more vulnerable than others, and significant differences have been reported between subjects [5]. Sensitivity to sleep loss is reliable within subjects, but it depends on the task [123]. Thus, it is important to consider individual differences in response to sleep deprivation in the formulation of hypotheses regarding sleep loss. Regarding other neurocognitive processes, while there has been less

investigation in these areas, there is evidence that sleep loss is associated with impaired memory and academic performance [38].

### Should Short Sleepers Simply Sleep Longer?

If habitual short sleepers demonstrate impairments, two important questions arise:

1. Does short sleep cause problems, or is it a result of them?
2. Does sleep extension alleviate these problems?

The first question is difficult to address: randomization to a short sleep lifestyle is not feasible, though other study designs may approach this question. The second question is the ideal next step for several reasons: it is more easily testable, would demonstrate causality in the other direction (supporting the claim that short sleep causes problems), and may be extended later to apply to acute sleep loss. Thus, with a sleep extension intervention, we could demonstrate if, in short sleepers, sleep extension improves health and functioning.

## Practice Points

1. Terms describing various forms of "short sleep" need to be clarified and used appropriately. There has been much confusion among "sleep deprivation," "sleep loss," "insomnia," and other related terms. This has led to confusion regarding interpretation of data. We propose definitions outlined in Table 1.
2. Laboratory studies have described impairments in neurobehavioral performance, metabolism and obesity, and psychological health associated with acute sleep deprivation.
3. Epidemiological studies have found links between self-reported short sleep duration and mortality risk, obesity and metabolism, cardiovascular disease, and general health/psychosocial stress.
4. Studies of verified short sleepers suggest that this group may experience greater homeostatic sleep pressure and sleep debt, exhibit cardiovascular risks, and are at increased risk for obesity.
5. These domains converge to suggest that habitual short sleep may be associated with increased mortality risk, performance deficits, cardiovascular risk, obesity risk and metabolic dysregulation.

## Research Agenda

1. Findings from laboratory studies need to be replicated in verified short sleepers. This involves assessments of health, performance and psychological functioning.
2. Findings from epidemiological studies need to be replicated in verified short sleepers. Possible links between short sleep and mortality risks need to be explored in experiments rather than just epidemiological studies. Also, questions of how and why short sleepers may be at risk for obesity and diabetes need to be addressed in protocols involving verified short sleepers.
3. The "short sleepers" need to be phenotyped. This will be a heterogeneous group, and methods for differentiating short sleepers with decreased sleep need and those experiencing sleep insufficiency need to be explored.
4. Sleep extension interventions should be explored, once a valid approach for differentiating those with and without impairment is elucidated.

## Acknowledgments

Research supported by T32HL007713 and UL1RR024134. We would like to thank Karen Teff, Ph.D. for input on Metabolism and Obesity, and Nalaka Gooneratne M.D. for assistance in organizing ideas for the manuscript.

## References

1. Belenky G, Wesensten NJ, Thorne DR, Thomas ML, Sing HC, Redmond DP, et al. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. J Sleep Res Mar;2003 12(1):1–12. [PubMed: 12603781]
2. Dinges DF, Pack F, Williams K, Gillen KA, Powell JW, Ott GE, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. Sleep Apr;1997 20(4):267–277. [PubMed: 9231952]
3. Drummond SP, Bischoff-Grethe A, Dinges DF, Ayalon L, Mednick SC, Meloy MJ. The neural basis of the psychomotor vigilance task. Sleep Sep 1;2005 28(9):1059–1068. [PubMed: 16268374]
4. Lim J, Dinges DF. Sleep deprivation and vigilant attention. Ann N Y Acad Sci 2008;1129:305–322. [PubMed: 18591490]
5*. Banks S, Dinges DF. Behavioral and physiological consequences of sleep restriction. J Clin Sleep Med Aug 15;2007 3(5):519–528. [PubMed: 17803017]
6. Bonnet MH, Arand DL. Clinical effects of sleep fragmentation versus sleep deprivation. Sleep Med Rev Aug;2003 7(4):297–310. [PubMed: 14505597]
7. Dinges DF. The state of sleep deprivation: From functional biology to functional consequences. Sleep Med Rev Oct;2006 10(5):303–305. [PubMed: 16919979]
8. Durmer JS, Dinges DF. Neurocognitive consequences of sleep deprivation. Semin Neurol Mar;2005 25(1):117–129. [PubMed: 15798944]
9. Chaput JP, Despres JP, Bouchard C, Tremblay A. Association of sleep duration with type 2 diabetes and impaired glucose tolerance. Diabetologia Nov;2007 50(11):2298–2304. [PubMed: 17717644]
10. Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. Sleep Med Rev Jun;2007 11(3):163–178. [PubMed: 17442599]
11. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. Ann N Y Acad Sci 2008;1129:287–304. [PubMed: 18591489]
12. Schultes B, Schmid S, Peters A, Born J, Fehm HL. Sleep loss and the development of diabetes: a review of current evidence. Exp Clin Endocrinol Diabetes Dec;2005 113(10):563–567. [PubMed: 16320152]
13. Spiegel K, Knutson K, Leproult R, Tasali E, Van Cauter E. Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. J Appl Physiol Nov;2005 99(5):2008–2019. [PubMed: 16227462]
14. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet Oct 23;1999 354(9188):1435–1439. [PubMed: 10543671]
15. Taheri S. Sleep and metabolism: bringing pieces of the jigsaw together. Sleep Med Rev Jun;2007 11 (3):159–162. [PubMed: 17517354]
16. Meerlo P, Sgoifo A, Suchecki D. Restricted and disrupted sleep: effects on autonomic function, neuroendocrine stress systems and stress responsivity. Sleep Med Rev Jun;2008 12(3):197–210. [PubMed: 18222099]
17. Franzen PL, Siegle GJ, Buysse DJ. Relationships between affect, vigilance, and sleepiness following sleep deprivation. J Sleep Res Mar;2008 17(1):34–41. [PubMed: 18275553]
18. Haack M, Mullington JM. Sustained sleep restriction reduces emotional and physical well-being. Pain Dec 15;2005 119(1–3):56–64. [PubMed: 16297554]
19*. Grandner MA, Drummond SP. Who are the long sleepers? Towards an understanding of the mortality relationship. Sleep Med Rev Oct;2007 11(5):341–360. [PubMed: 17625932]
20. Hartz AJ, Daly JM, Kohatsu ND, Stromquist AM, Jogerst GJ, Kukoyi OA. Risk factors for insomnia in a rural population. Ann Epidemiol Dec;2007 17(12):940–947. [PubMed: 17937993]
21. Bjorvatn B, Sagen IM, Oyane N, Waage S, Fetveit A, Pallesen S, et al. The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study. J Sleep Res Mar; 2007 16(1):66–76. [PubMed: 17309765]

22. Cappuccio FP, Taggart FM, Kandala NB, Currie A, Peile E, Stranges S, et al. Meta-analysis of short sleep duration and obesity in children and adults. Sleep May 1;2008 31(5):619–626. [PubMed: 18517032]
23. Chaput JP, Brunet M, Tremblay A. Relationship between short sleeping hours and childhood overweight/obesity: results from the 'Quebec en Forme' Project. Int J Obes (Lond) Jul;2006 30(7): 1080–1085. [PubMed: 16534525]
24. Chaput JP, Despres JP, Bouchard C, Tremblay A. The association between sleep duration and weight gain in adults: a 6-year prospective study from the Quebec Family Study. Sleep Apr 1;2008 31(4): 517–523. [PubMed: 18457239]
25. Chaput JP, Despres JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: Results from the Quebec family study. Obesity (Silver Spring) Jan;2007 15(1):253–261. [PubMed: 17228054]
26. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Sleep duration as a risk factor for diabetes incidence in a large U.S. sample. Sleep Dec 1;2007 30(12):1667–1673. [PubMed: 18246976]
27. Steptoe A, Peacey V, Wardle J. Sleep duration and health in young adults. Arch Intern Med Sep 18;2006 166(16):1689–1692. [PubMed: 16983045]
28. Bakalar N. Extra sleep is found to lower a heart risk. New York Times. December 30;2008 Health.
29. McKnight-Eily LR, Presley-Cantrell LR, Strine TW, Chapman DP, Perry GS, Croft JB. Perceived insufficient rest or sleep--four states, 2006. MMWR Morb Mortal Wkly Rep Feb 29;2008 57(8):200–203. [PubMed: 18305450]
30. Kleitman, N. Sleep and Wakefulness. Chicago: University of Chicago Press; 1939.
31. Kryger, MH.; Roth, T.; Dement, WC. Principles and practice of sleep medicine. 4. Philadelphia, PA: Elsevier/Saunders; 2005.
32. American Academy of Sleep Medicine. International Classification of Sleep Disorders. 2. Westchester, IL: Author; 2005.
33. Pack AI, Dinges DF, Gehrman PR, Staley B, Pack FM, Maislin G. Risk factors for excessive sleepiness in older adults. Ann Neurol Jun;2006 59(6):893–904. [PubMed: 16718691]
34. Guilleminault C, Powell NB, Martinez S, Kushida C, Raffray T, Palombini L, et al. Preliminary observations on the effects of sleep time in a sleep restriction paradigm. Sleep Med May;2003 4(3): 177–184. [PubMed: 14592319]
35. Banks S, Dinges DF. Is The Maintenance Of Wakefulness Test Sensitive To Varying Amounts Of Recovery Sleep After Chronic Sleep Restriction? Sleep 2005;28(Abstract Supplement):A136.
36. Dinges DF, Powell JW. Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. Beh Res Meth Instr Comp 1985;17:652–655.
37. Boonstra TW, Stins JF, Daffertshofer A, Beek PJ. Effects of sleep deprivation on neural functioning: an integrative review. Cell Mol Life Sci Apr;2007 64(7–8):934–946. [PubMed: 17347797]
38. Curcio G, Ferrara M, De Gennaro L. Sleep loss, learning capacity and academic performance. Sleep Med Rev Oct;2006 10(5):323–337. [PubMed: 16564189]
39. Chee MW, Chuah LY, Venkatraman V, Chan WY, Philip P, Dinges DF. Functional imaging of working memory following normal sleep and after 24 and 35 h of sleep deprivation: Correlations of fronto-parietal activation with performance. Neuroimage May 15;2006 31(1):419–428. [PubMed: 16427321]
40. Radun I, Ohisalo J, Radun JE, Summala H, Tolvanen M. Fell asleep and caused a fatal head-on crash? A case study of multidisciplinary in-depth analysis vs. the court. Traffic Inj Prev Mar;2009 10(1): 76–83. [PubMed: 19214881]
41. Anund A, Kecklund G, Vadeby A, Hjalmdahl M, Akerstedt T. The alerting effect of hitting a rumble strip--a simulator study with sleepy drivers. Accid Anal Prev Nov;2008 40(6):1970–1976. [PubMed: 19068302]
42. Ingre M, Akerstedt T, Peters B, Anund A, Kecklund G, Pickles A. Subjective sleepiness and accident risk avoiding the ecological fallacy. J Sleep Res Jun;2006 15(2):142–148. [PubMed: 16704568]
43. MacLean AW, Davies DR, Thiele K. The hazards and prevention of driving while sleepy. Sleep Med Rev Dec;2003 7(6):507–521. [PubMed: 15018093]

44. McConnell CF, Bretz KM, Dwyer WO. Falling asleep at the wheel: aclose look at 1,269 fatal and serious injury-producing crashes. Behav Sleep Med 2003;1(3):171–183. [PubMed: 15600220]
45. Varughese J, Allen RP. Fatal accidents following changes in daylight savings time: the American experience. Sleep Med Jan;2001 2(1):31–36. [PubMed: 11152980]
46. Pizza F, Contardi S, Mondini S, Trentin L, Cirignotta F. Daytime sleepiness and driving performance in patients with obstructive sleep apnea: comparison of the MSLT, the MWT, and a simulated driving task. Sleep Mar 1;2009 32(3):382–391. [PubMed: 19294958]
47. Powell NB, Schechtman KB, Riley RW, Guilleminault C, Chiang RP, Weaver EM. Sleepy driver near-misses may predict accident risks. Sleep Mar 1;2007 30(3):331–342. [PubMed: 17425230]
48. Philip P, Akerstedt T. Transport and industrial safety, how are they affected by sleepiness and sleep restriction? Sleep Med Rev Oct;2006 10(5):347–356. [PubMed: 16920370]
49. Gurubhagavatula I, Nkwuo JE, Maislin G, Pack AI. Estimated cost of crashes in commercial drivers supports screening and treatment of obstructive sleep apnea. Accid Anal Prev Jan;2008 40(1):104–115. [PubMed: 18215538]
50. Al-Barrak M, Shepertycky MR, Kryger MH. Morbidity and mortality in obstructive sleep apnea syndrome 2: Effect of treatment on neuropsychiatric morbidity and quality of life. Sleep and Biological Rhythms 2003;1:65–74.
51. Sagaspe P, Taillard J, Akerstedt T, Bayon V, Espie S, Chaumet G, et al. Extended driving impairs nocturnal driving performances. PLoS ONE 2008;3(10):e3493. [PubMed: 18941525]
52. Scott LD, Hwang WT, Rogers AE, Nysse T, Dean GE, Dinges DF. The relationship between nurse work schedules, sleep duration, and drowsy driving. Sleep Dec 1;2007 30(12):1801–1807. [PubMed: 18246989]
53. Aeschbach D, Postolache TT, Sher L, Matthews JR, Jackson MA, Wehr TA. Evidence from the waking electroencephalogram that short sleepers live under higher homeostatic sleep pressure than long sleepers. Neuroscience 2001;102(3):493–502. [PubMed: 11226688]
54. Aeschbach D, Cajochen C, Landolt H, Borbely AA. Homeostatic sleep regulation in habitual short sleepers and long sleepers. Am J Physiol Jan;1996 270(1 Pt 2):R41–53. [PubMed: 8769783]
55. Kripke DF, Rex K. Short sleepers are not at higher risk for driving accidents or other violations. Sleep 2002;25(Abstract Supplement):A284–285.
56. Van Cauter E, Blackman JD, Roland D, Spire JP, Refetoff S, Polonsky KS. Modulation of glucose regulation and insulin secretion by circadian rhythmicity and sleep. J Clin Invest Sep;1991 88(3): 934–942. [PubMed: 1885778]
57. Rechtschaffen A, Bergmann BM. Sleep deprivation in the rat: an update of the 1989 paper. Sleep Feb 1;2002 25(1):18–24. [PubMed: 11833856]
58. Gale SM, Castracane VD, Mantzoros CS. Energy homeostasis, obesity and eating disorders: recent advances in endocrinology. J Nutr Feb;2004 134(2):295–298. [PubMed: 14747663]
59. van der Lely AJ, Tschop M, Heiman ML, Ghigo E. Biological, physiological, pathophysiological, and pharmacological aspects of ghrelin. Endocr Rev Jun;2004 25(3):426–457. [PubMed: 15180951]
60. Mullington JM, Chan JL, Van Dongen HP, Szuba MP, Samaras J, Price NJ, et al. Sleep loss reduces diurnal rhythm amplitude of leptin in healthy men. J Neuroendocrinol Sep;2003 15(9):851–854. [PubMed: 12899679]
61. Dzaja A, Dalal MA, Himmerich H, Uhr M, Pollmacher T, Schuld A. Sleep enhances nocturnal plasma ghrelin levels in healthy subjects. Am J Physiol Endocrinol Metab Jun;2004 286(6):E963–967. [PubMed: 14871884]
62. Spiegel K, Leproult R, L'Hermite-Baleriaux M, Copinschi G, Penev PD, Van Cauter E. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. J Clin Endocrinol Metab Nov;2004 89(11):5762–5771. [PubMed: 15531540]
63. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med Dec 7;2004 141(11):846–850. [PubMed: 15583226]
64. Grandner MA, Kripke DF, Langer RD. Correlations among dietary nutrient variables and subjective and objective sleep. Sleep 2005;28(Abstract Supplement):A148–A149.

65. van Leeuwen WM, Lehto M, Karisola P, Lindholm H, Luukkonen R, Sallinen M, et al. Sleep restriction increases the risk of developing cardiovascular diseases by augmenting proinflammatory responses through IL-17 and CRP. PLoS ONE 2009;4(2):e4589. [PubMed: 19240794]

66. Sekine T, Daimon M, Hasegawa R, Toyoda T, Kawata T, Funabashi N, et al. Theimpact of sleep deprivation on the coronary circulation. Int J Cardiol. Feb 7;2009

67. Lusardi P, Vanasia A, Mugellini A, Zoppi A, Preti P, Fogari R. Evaluation of nocturnal blood pressure by the Multi-P Analysis of 24-hour ambulatory monitoring. Z Kardiol 1996;85 (Suppl 3):121–123. [PubMed: 8896314]

68. Lusardi P, Zoppi A, Preti P, Pesce RM, Piazza E, Fogari R. Effects of insufficient sleep on blood pressure in hypertensive patients: a 24-h study. Am J Hypertens Jan;1999 12(1 Pt 1):63–68. [PubMed: 10075386]

69. Roman, V.; Hagewoud, R.; Luiten, PGM.; Meerlo, P. PhD Thesis. University of Groningen; The Netherlands: 2007. Altered serotonergic and corticotropin releasing hormone regulation of the hypothalamo-pituitary-adrenal stress system in chronic partial sleep deprivation.

70. Killgore WD, Balkin TJ, Wesensten NJ. Impaired decision making following 49 h of sleep deprivation. J Sleep Res Mar;2006 15(1):7–13. [PubMed: 16489997]

71. Venkatraman V, Chuah YM, Huettel SA, Chee MW. Sleep deprivation elevates expectation of gains and attenuates response to losses following risky decisions. Sleep May 1;2007 30(5):603–609. [PubMed: 17552375]

72. Roehrs T, Greenwald M, Roth T. Risk-taking behavior: effects of ethanol, caffeine, and basal sleepiness. Sleep Aug 1;2004 27(5):887–893. [PubMed: 15453546]

73. McKenna BS, Dicjinson DL, Orff HJ, Drummond SP. The effects of one night of sleep deprivation on known-risk and ambiguous-risk decisions. J Sleep Res Sep;2007 16(3):245–252. [PubMed: 17716272]

74. Acheson A, Richards JB, de Wit H. Effects of sleep deprivation on impulsive behaviors in men and women. Physiol Behav Aug 15;2007 91(5):579–587. [PubMed: 17477941]

75. Young T. Increasing sleep duration for a healthier (and less obese?) population tomorrow. Sleep May 1;2008 31(5):593–594. [PubMed: 18517028]

76. Hammond EC. Some Preliminary Findings on Physical Complaints from a Prospective Study of 1,064,004 Men and Women. Am J Public Health Nations Health Jan;1964 54:11–23. [PubMed: 14117648]

77*. Grandner MA, Patel NP, Hale L, Moore M. Mortality associated with sleep duration: The evidence, the possible mechanisms, and the future. Sleep Med Rev. 2009

78. Gallicchio L, Kalesan B. Sleep Duration and Mortality: A Systematic Review and Meta-analysis. J Sleep Res 2009;18(2):148–158. [PubMed: 19645960]

79. Bliwise DL, Young TB. The parable of parabola: what the U-shaped curve can and cannot tell us about sleep. Sleep Dec 1;2007 30(12):1614–1615. [PubMed: 18246971]

80. Hall MH, Muldoon MF, Jennings JR, Buysse DJ, Flory JD, Manuck SB. Self-reported sleep duration is associated with the metabolic syndrome in midlife adults. Sleep May 1;2008 31(5):635–643. [PubMed: 18517034]

81. Locard E, Mamelle N, Billette A, Miginiac M, Munoz F, Rey S. Risk factors of obesity in a five year old population. Parental versus environmental factors. Int J Obes Relat Metab Disord Oct;1992 16 (10):721–729. [PubMed: 1330951]

82. von Kries R, Toschke AM, Wurmser H, Sauerwald T, Koletzko B. Reduced risk for overweight and obesity in 5- and 6-y-old children by duration of sleep--a cross-sectional study. Int J Obes Relat Metab Disord May;2002 26(5):710–716. [PubMed: 12032757]

83. Shigeta H, Shigeta M, Nakazawa A, Nakamura N, Yoshikawa T. Lifestyle, obesity, and insulin resistance. Diabetes Care Mar;2001 24(3):608. [PubMed: 11289495]

84. Vioque J, Torres A, Quiles J. Time spent watching television, sleep duration and obesity in adults living in Valencia, Spain. Int J Obes Relat Metab Disord Dec;2000 24(12):1683–1688. [PubMed: 11126224]

85. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, et al. Early liferisk factors for obesity in childhood: cohort study. BMJ Jun 11;2005 330(7504):1357. [PubMed: 15908441]

86. Kripke DF, Garfinkel L, Wingard DL, Klauber MR, Marler MR. Mortality associated with sleep duration and insomnia. Arch Gen Psychiatry Feb;2002 59(2):131–136. [PubMed: 11825133]

87. Littman AJ, Vitiello MV, Foster-Schubert K, Ulrich CM, Tworoger SS, Potter JD, et al. Sleep, ghrelin, leptin and changes in body weight during a 1-year moderate-intensity physical activity intervention. Int J Obes (Lond) Mar;2007 31(3):466–475. [PubMed: 16909130]

88. Patel SR, Ayas NT, Malhotra MR, White DP, Schernhammer ES, Speizer FE, et al. A prospective study of sleep duration and mortality risk in women. Sleep May 1;2004 27(3):440–444. [PubMed: 15164896]

89. Hasler G, Buysse DJ, Klaghofer R, Gamma A, Ajdacic V, Eich D, et al. The association between short sleep duration and obesity in young adults: a 13-year prospective study. Sleep Jun 15;2004 27 (4):661–666. [PubMed: 15283000]

90. Kohatsu ND, Tsai R, Young T, Vangilder R, Burmeister LF, Stromquist AM, et al. Sleep duration and body mass index in a rural population. Arch Intern Med Sep 18;2006 166(16):1701–1705. [PubMed: 16983047]

91. Gottlieb DJ, Redline S, Nieto FJ, Baldwin CM, Newman AB, Resnick HE, et al. Association of usual sleep duration with hypertension: the Sleep Heart Health Study. Sleep Aug 1;2006 29(8):1009–1014. [PubMed: 16944668]

92. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. Hypertension May;2006 47(5):833–839. [PubMed: 16585410]

93. Cappuccio FP, Stranges S, Kandala NB, Miller MA, Taggart FM, Kumari M, et al. Gender-specific associations of short sleep duration with prevalent and incident hypertension: the Whitehall II Study. Hypertension Oct;2007 50(4):693–700. [PubMed: 17785629]

94. Chen JC, Brunner RL, Ren H, Wassertheil-Smoller S, Larson JC, Levine DW, et al. Sleep duration and risk of ischemic stroke in postmenopausal women. Stroke. Jul 17;2008

95. Meisinger C, Heier M, Lowel H, Schneider A, Doring A. Sleep duration and sleep complaints and risk of myocardial infarction in middle-aged men and women from the general population: the MONICA/KORA Augsburg cohort study. Sleep Sep 1;2007 30(9):1121–1127. [PubMed: 17910384]

96. Stamatakis KA, Kaplan GA, Roberts RE. Short sleep duration across income, education, and race/ethnic groups: population prevalence and growing disparities during 34 years of follow-up. Ann Epidemiol Dec;2007 17(12):948–955. [PubMed: 17855122]

97. Hale L, Do DP. Racial differences in self-reports of sleep duration in a population-based study. Sleep Sep 1;2007 30(9):1096–1103. [PubMed: 17910381]

98. Grandner MA, Kripke DF. Self-reported sleep complaints with long and short sleep: a nationally representative sample. Psychosom Med Mar-Apr;2004 66(2):239–241. [PubMed: 15039509]

99. Ohayon MM, Vecchierini MF. Normative sleep data, cognitive function and daily living activities in older adults in the community. Sleep Aug 1;2005 28(8):981–989. [PubMed: 16218081]

100. Kumar A, Vaidya AK. Locus of control in short and long sleepers. Br J Psychiatry Jun;1986 148:739–740. [PubMed: 3779256]

101. Hicks RA, Marical CM, Conti PA. Coping with a major stressor: differences between habitual short- and longer-sleepers. Percept Mot Skills Apr;1991 72(2):631–636. [PubMed: 1852575]

102*. Hartmann E, Baekeland F, Zwilling GR. Psychological differences between long and short sleepers. Arch Gen Psychiatry May;1972 26(5):463–468. [PubMed: 5019885]

103. Kumar A, Vaidya AK. Anxiety as a personality dimension of short and long sleepers. J Clin Psychol Jan;1984 40(1):197–198. [PubMed: 6746927]

104. Monk TH, Buysse DJ, Welsh DK, Kennedy KS, Rose LR. A sleep diary and questionnaire study of naturally short sleepers. J Sleep Res Sep;2001 10(3):173–179. [PubMed: 11696070]

105. Sexton-Radek K. Stress triggers of long, short, and variable sleep patterns. Percept Mot Skills Aug; 1998 87(1):225–226. [PubMed: 9760649]

106. Webb WB, Friel J. Sleep stage and personality characteristics of “natural” long and short sleepers. Science Feb 12;1971 171(971):587–588. [PubMed: 4395451]

107. Webb WB, Agnew HW Jr. Sleep stage characteristics of long and short sleepers. Science Apr 3;1970 168(927):146–147. [PubMed: 4313684]

108. Fukuda K, Miyasita A, Inugami M. Sleep onset REM periods observed after sleep interruption in normal short and normal long sleeping subjects. Electroencephalogr Clin Neurophysiol Dec;1987 67(6):508–513. [PubMed: 2445542]

109*. Aeschbach D, Sher L, Postolache TT, Matthews JR, Jackson MA, Wehr TA. A longer biological night in long sleepers than in short sleepers. J Clin Endocrinol Metab Jan;2003 88(1):26–30. [PubMed: 12519823]

110*. Klerman EB, Dijk DJ. Interindividual variation in sleep duration and its association with sleep debt in young adults. Sleep Oct 1;2005 28(10):1253–1259. [PubMed: 16295210]

111. Dinges DF. Can habitual sleep duration harbor sleep debt? Sleep Oct 1;2005 28(10):1209–1210. [PubMed: 16295200]

112. King CR, Knutson KL, Rathouz PJ, Sidney S, Liu K, Lauderdale DS. Short sleep duration and incident coronary artery calcification. JAMA Dec 24;2008 300(24):2859–2866. [PubMed: 19109114]

113*. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med Dec;2004 1(3):e62. [PubMed: 15602591]

114. Grandner MA, Kripke DF, Langer RD. Light Exposure is Related to Social and Emotional Functioning and to Quality of Life in older women. Psychiatry Res Jun 30;2006 143(1):35–42. [PubMed: 16725207]

115. Kripke DF, Jean-Louis G, Elliott JA, Klauber MR, Rex KM, Tuunainen A, et al. Ethnicity, sleep, mood, and illumination in postmenopausal women. BMC Psychiatry Apr 7;2004 4:8. [PubMed: 15070419]

116. Patterson RE, Kristal AR, Tinker LF, Carter RA, Bolton MP, Agurs-Collins T. Measurement characteristics of the Women's Health Initiative food frequency questionnaire. Ann Epidemiol Apr; 1999 9(3):178–187. [PubMed: 10192650]

117. Brown LK. Quantum physics and polysomnography: can we prevent the act of measuring sleep from changing sleep? J Clin Sleep Med Apr 15;2005 1(2):133–135. [PubMed: 17561626]

118. Agnew HW Jr, Webb WB, Williams RL. The first night effect: an EEG study of sleep. Psychophysiology Jan;1966 2(3):263–266. [PubMed: 5903579]

119*. Jean-Louis G, Kripke DF, Ancoli-Israel S. Sleep and quality of well-being. Sleep Dec 15;2000 23 (8):1115–1121. [PubMed: 11145326]

120. Lauderdale DS, Knutson KL, Yan LL, Rathouz PJ, Hulley SB, Sidney S, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. Am J Epidemiol Jul 1;2006 164(1):5–16. [PubMed: 16740591]

121. Horne J, Reyner L. Vehicle accidents related to sleep: a review. Occup Environ Med May;1999 56 (5):289–294. [PubMed: 10472301]

122*. Basner M, Fomberstein KM, Razavi FM, Banks S, William JH, Rosa RR, et al. American time use survey: sleep time and its relationship to waking activities. Sleep Sep 1;2007 30(9):1085–1095. [PubMed: 17910380]

123*. Van Dongen HP, Baynard MD, Maislin G, Dinges DF. Systematic interindividual differences in neurobehavioral impairment from sleep loss: evidence of trait-like differential vulnerability. Sleep May 1;2004 27(3):423–433. [PubMed: 15164894]

124. Schutte-Rodin S, Broch L, Buysse D, Dorsey C, Sateia M. Clinical guideline for the evaluation and management of chronic insomnia in adults. J Clin Sleep Med Oct 15;2008 4(5):487–504. [PubMed: 18853708]

125. Moser D, Anderer P, Gruber G, Parapatics S, Loretz E, Boeck M, et al. Sleep classification according to AASM and Rechtschaffen & Kales: effects on sleep scoring parameters. Sleep Feb 1;2009 32 (2):139–149. [PubMed: 19238800]

126. Krueger PM, Friedman EM. Sleep duration in the United States: a cross-sectional population-based study. Am J Epidemiol May 1;2009 169(9):1052–1063. [PubMed: 19299406]

127. Patel SR, Zhu X, Storfer-Isser A, Mehra R, Jenny NS, Tracy R, et al. Sleep duration and biomarkers of inflammation. Sleep Feb 1;2009 32(2):200–204. [PubMed: 19238807]

128. Winkelman, JW.; Kotagal, S.; Olson, CM.; Scammell, T.; Schenck, C.; Spielman, A. The International Classification of Sleep Disorders. 2. Westchester, IL: American Academy of Sleep Medicine; 2006.

129. Elmenhorst EM, Elmenhorst D, Luks N, Maass H, Vejvoda M, Samel A. Partial sleep deprivation: impact on the architecture and quality of sleep. Sleep Med Dec;2008 9(8):840–850. [PubMed: 17921066]

130. Dew MA, Hoch CC, Buysse DJ, Monk TH, Begley AE, Houck PR, et al. Healthy older adults' sleep predicts all-cause mortality at 4 to 19 years of follow-up. Psychosom Med Jan–Feb;2003 65(1):63–73. [PubMed: 12554816]

131. Grandner MA, Patel NP. From sleep duration to mortality: implications of meta-analysis and future directions. J Sleep Res 2009;18(2):145–147. [PubMed: 19645959]

132. Grandner MA, Patel NP, Gehrman PR, Xie D, Sha D, Weaver T, et al. Gender differences in sleep disturbance patterns associated with aging. Sleep 2009;32(Abstract Supplement):A122.

133. Grandner MA, Patel NP, Gehrman PR, Xie D, Sha D, Weaver T, et al. Who sleeps better? Socioeconomic differences in reports of sleep disturbance. Sleep 2009;32(Abstract Supplement):A422–423.

134. Nedeltcheva AV, Kilkus JM, Imperial J, Kasza K, Schoeller DA, Penev PD. Sleep curtailment is accompanied by increased intake of calories from snacks. Am J Clin Nutr Jan;2009 89(1):126–133. [PubMed: 19056602]

135. Riemann D, Perlis ML. The treatments of chronic insomnia: A review of benzodiazepine receptor agonists and psychological and behavioral therapies. Sleep Med Rev. Feb 7;2009

136. Strine TW, Chapman DP. Associations of frequent sleep insufficiency with health-related quality of life and health behaviors. Sleep Med Jan;2005 6(1):23–27. [PubMed: 15680291]

137. Balkin TJ, Rupp T, Picchioni D, Wesensten NJ. Sleep loss and sleepiness: current issues. Chest Sep; 2008 134(3):653–660. [PubMed: 18779203]

138. Goel N, Banks S, Mignot E, Dinges DF. PER3 polymorphism predicts cumulative sleep homeostatic but not neurobehavioral changes to chronic partial sleep deprivation. PLoS ONE 2009;4(6):e5874. [PubMed: 19516903]

## Table 1

Working lexicon of Short Sleep terms

| Term | Working Definition |
|---|---|
| Sleep Opportunity[124]: | Amount of time in which sleep is possible; including time in bed awake. |
| Sleep Time[125] Sleep Duration [126–127]: | Amount of time spent sleeping. |
| Insomnia[128]: | A clinical diagnosis with specific diagnostic criteria that describe a condition associated with pathologically long sleep latency, large amounts of wake time after sleep onset, and/or early morning awakenings associated with distress or impairment in functioning. |
| Partial Sleep Deprivation[129]: | Sleep deprivation in the laboratory setting, up to 5 hours sleep time; some sleep is achieved. |
| Self-Reported Short Sleep[86] | Subjective, retrospective estimate of habitual sleep time, or mean sleep time from prospective sleep measures (e.g., sleep diaries). |
| Short Sleep[130]: | Habitual sleep time of 6 hours or less. |
| Sleep Attainment[131]: | Habitual sleep quantity of sufficient quality. |
| Sleep Curtailment[122]: | The deliberate shortening of sleep opportunity. |
| Sleep Deprivation[4]: | Acute sleep curtailment in the laboratory setting. |
| Sleep Disturbance[132–133]: | Any problems associated with sleep, usually attributed to symptoms of diagnosable sleep disorders, such as insomnia, sleep apnea, narcolepsy, sleep-related movement disorders, and other sleep disorders; may apply to other clinical conditions, including affective disorder, anxiety disorder, or pain. |
| Sleep Restriction[134]: | Sleep deprivation (in or out of the laboratory), >5 hours sleep time but less than habitual sleep time. May in other contexts refer to a treatment for insomnia[135]. |
| Sleep Insufficiency[136]: | Reduction in sleep time of a magnitude associated with negative outcomes |
| Sleep Loss[137]: | Decrease in sleep time relative to previous sleep time |
| Total Sleep Deprivation[138]: | Complete elimination of sleep for 24 hours or more |
| Verified Short Sleep[64]: | Verification of self-reported habitual sleep duration using polysomnography or actigraphy |