

Sleep Duration and Quality: Impact on Lifestyle Behaviors and Cardiometabolic Health

A Scientific Statement From the American Heart Association

ABSTRACT: Sleep is increasingly recognized as an important lifestyle contributor to health. However, this has not always been the case, and an increasing number of Americans choose to curtail sleep in favor of other social, leisure, or work-related activities. This has resulted in a decline in average sleep duration over time. Sleep duration, mostly short sleep, and sleep disorders have emerged as being related to adverse cardiometabolic risk, including obesity, hypertension, type 2 diabetes mellitus, and cardiovascular disease. Here, we review the evidence relating sleep duration and sleep disorders to cardiometabolic risk and call for health organizations to include evidence-based sleep recommendations in their guidelines for optimal health.

The ubiquity of public health reports touting the importance of sleep has led to an increased interest in understanding the extent of sleep problems at the population level and their associated negative effects on various cardiometabolic health outcomes. According to the National Heart, Lung, and Blood Institute of the National Institutes of Health, ~50 to 70 million US adults suffer from a sleep disorder or report insufficient sleep habitually.¹ Although many individuals may opt to curtail their bedtime to pursue personal/professional goals or social obligations, a sizable portion may be experiencing sleep problems originating from a medical or psychosocial cause. Insomnia, the most common sleep disorder, is likely present in 5% to 15% of the US population, with ~30% reporting significant symptoms at any given time.² Sleep apnea, another common sleep disorder defined as at least 5 respiratory events (apnea or hypopnea) per hour of sleep on average, has an estimated prevalence of 27% to 34% among men 30 to 70 years of age and 9% to 28% among women in the same age group.³

The impact of obstructive sleep apnea (OSA) and insomnia on cardiovascular disease (CVD) and metabolic disorders is striking.^{4,5} Population-based studies show that individuals with OSA or insomnia are at significantly greater risk for CVD and cerebrovascular diseases (eg, arrhythmias, atherosclerosis, coronary heart disease [CHD], heart failure, hypertension, and stroke) and metabolic disorders (eg, obesity, type 2 diabetes mellitus, and dyslipidemia).⁶⁻¹¹ Of note, both OSA and insomnia are associated with insufficient sleep durations, which may have independent effects on these chronic diseases.¹² Below, we provide a brief description of sleep disorders and inadequate sleep duration (short sleep, defined as <7 h/night, or long sleep, defined as ≥9 h/night, unless otherwise noted) and provide evidence of the prevalence of these sleep-related problems in the general population and their effects on overall cardiometabolic health. A search of Ovid Medline, Embase, Cochrane, and clinicaltrials.gov was conducted in February 2015 with the following search terms: *sleep duration (sleep, sleep deprivation, sleep duration, sleep restriction), sleep*

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disorders (sleep disorder, insomnia, obstructive sleep apnea, periodic limb movement disorder, restless leg syndrome, sleep disordered breathing), cardiovascular disease (cardiovascular disease, type 2 diabetes mellitus, hypertension), cardiometabolic risk factors (insulin sensitivity, blood pressure, inflammation, inflammatory markers, lipids, lipid profile), energy balance (body weight, obesity, overweight, weight loss, weight reduction, diet, energy intake, energy metabolism, energy balance, energy expenditure, resting metabolic rate, eating, food intake, leptin, ghrelin), and behavior modification (behavior modification, behavior therapy, conditioning therapy, sleep hygiene, sleep restriction therapy, cognitive behavioral therapy for insomnia, barriers, electronic devices, screens, tablets). Our search was restricted to human subjects research published on or after 2005 in the English language.

OSA is the most prevalent sleep disordered breathing (SDB) condition, caused by a repeated narrowing of the upper airway during sleep. The most common characteristics include periods of breathing cessations, oxygen desaturations, and repetitive awakenings during sleep. Individuals with OSA often report a history of snoring, poor memory, morning headaches, and daytime sleepiness.¹³ Some individuals with OSA may also experience comorbid insomnia and short sleep. It is estimated that $\approx 10\%$ of men and 3% of women <50 years of age have OSA, with estimates rising to 17% and 9% for men and women ≥ 50 years of age, respectively.³ Available data also suggest that OSA may be more prevalent among individuals of minority backgrounds.¹⁴ In fact, a study comparing blacks and whites 2 to 86 years of age found that 31% of blacks had OSA compared with 10% of whites.¹⁴

Insomnia, which may be a manifestation of a medical or psychiatric condition, is characterized by 3 primary symptoms: difficulty falling asleep, difficulty staying asleep, and early morning awakenings that occur at least 3 nights a week for at least 3 months; some individuals also complain of feeling tired on awakening. According to a number of epidemiological studies, the prevalence of insomnia symptoms in the previous year ranges from 30% to 45%.¹⁵ Although this condition is relatively common, only 6% of people with insomnia receive a diagnosis.¹⁶ Of note, women and those ≥ 65 years of age are more likely to report insomnia symptoms, although men and women from lower socioeconomic positions and education levels also tend to complain of insomnia.¹⁶

Recent evidence that habitual sleep duration is a risk factor for cardiometabolic conditions has given rise to multiple epidemiological and surveillance studies. The preponderance of evidence emanating from self-reported data suggests a curvilinear relationship between habitual sleep duration and various medical conditions (eg, obesity, hypertension, and diabetes mellitus) such that both short sleep duration (SSD) and long sleep duration are associated with those conditions.¹⁷ Published

data suggest that SSD, defined in the extant literature as sleep durations <7 hours,¹⁸ may have gradually increased over the past 40 years. Using data from the National Health Interview Survey, investigators examined trends in the prevalence of SSD, finding that it has, in fact, gradually increased, with 21.6% of US adults reporting SSD in 1977 compared with 29.1% in 2009.¹⁹ Notably, these prevalence estimates were comparable to estimates derived from the Sleep Heart Health Study.²⁰ Indeed, available data suggest that 27.5% of American adults reported SSD in 2005.²¹

In terms of long sleep, defined as sleep durations ≥ 9 hours, data suggest that its prevalence may have decreased from 11.6% reported in 1977 to 7.8% in 2009.¹⁹ This reduction in the prevalence of long sleep duration could simply be a reflection of the gradual decline in overall sleep duration, as has been demonstrated in other published studies. Examination of data gathered from 1959 to 2005 revealed a decrease in modal sleep durations of ≈ 1.5 hours.²² On balance, a recent systematic review suggested that long sleep may be a greater concern than SSD,²³ although experimental findings strongly indicate that SSD, rather than long sleep, is associated with adverse physiological and immunological consequences.^{24–27} Self-reported sleep durations may be affected by various factors, including demographic profiles, family structure, socioeconomic position, employment status, health risk behavior, and general health status.²⁸ The lack of objective measures of sleep duration in the majority of large-scale epidemiological studies is certainly a limitation that we wish to acknowledge at the forefront of this review. Nonetheless, despite the potential confounders introduced by the use of self-reports, these studies provide a valuable framework that warrants additional investigation using objective measures of sleep.

In this report, we review the epidemiological and clinical evidence relating sleep duration and some sleep disorders (insomnia/insomnia symptoms, SDB, periodic limb movement disorder, restless leg syndrome) to major risk factors for CVD such as obesity, type 2 diabetes mellitus, and hypertension, as well as CVD.

SLEEP DURATION AND OBESITY RISK

Epidemiological Evidence

Many epidemiological studies have described associations between self-reported habitual SSD and obesity. A meta-analysis by Cappuccio and colleagues²⁹ found that across 23 studies of adults, a pooled odds ratio of 1.55 was found. Furthermore, analysis of 7 studies that examined linear relationships between sleep duration and body mass index as a continuous variable showed that for every increased hour of sleep, body mass index was reduced by 0.35 points.²⁹ These findings are echoed in

several qualitative reviews on this topic,^{30–38} which all agree that individuals with habitual SSD are more likely to be obese than those with normal sleep habits (generally 7–8 hours of sleep a night).

Among these cross-sectional studies, several have identified important moderators of effects. For example, analyses of nationally representative data collected as part of the 2009 Behavioral Risk Factor Surveillance System made 2 notable findings on the sleep-obesity relationship. First, this analysis found that although the majority of individuals with SSD slept in the 5- to 6-hour range, the most pronounced effects were seen among those reporting ≤ 4 hours of sleep per night.³⁹ This finding suggested that risk varied across sleep duration; very short sleep (which more closely resembled sleep duration achieved in acute sleep restriction studies) seemed to carry greater risk than more typical short sleep. The second important finding in this study was that the relationship to obesity was independent of self-reported perceived sleep insufficiency.³⁹ A pair of notable studies that analyzed data from the 2007 to 2008 NHANES (National Health and Nutrition Evaluation Survey) found that the relation between sleep duration and obesity depends on age⁴⁰ and race/ethnicity.⁴¹ Specifically, the relation between SSD and obesity is stronger among younger than among older adults.⁴⁰ This finding is consistent with many studies that show that sleep loss in children and adolescents is a consistent risk factor for obesity in those groups.⁴² Furthermore, the relationship between SSD and obesity varies among blacks and whites,⁴¹ with blacks in the United States being disproportionately affected by SSD with respect to obesity risk.⁴³

Although cross-sectional studies often have the benefit of increased sample size and statistical power, it is impossible to determine the direction of effects. Further complicating this is the wording of survey items, which tends to focus on recent habitual sleep duration relative to obesity, which presumably developed over a much longer period. Thus, studies of incident weight gain and obesity are particularly helpful in this regard. Several key studies have identified habitual SSD as a risk factor for incident weight gain and obesity. In a landmark analysis of data from the Nurses' Health Study, women sleeping ≤ 5 hours a night on average gained 1.14 kg more weight over 16 years and women sleeping 6 hours gained 0.71 kg more weight compared with women sleeping at least 7 hours.⁴⁴ Furthermore, those sleeping ≤ 5 or 6 hours were 32% and 12% more likely to gain 15 kg over the 16-year follow-up period, respectively. These findings were replicated and extended with data from the Québec Family Study showing that over the course of 6 years, habitual short sleepers were more likely to gain weight and to have increased waist circumference and percent body fat compared with normal-duration sleepers. Notably, there was a U-shaped relationship in which a similar pattern was seen for habitual long sleepers.⁴⁵

In a study of male Japanese workers,⁴⁶ habitual sleep duration of < 6 hours was associated with increased likelihood of developing obesity over the course of 1 year. Taken together, these and other studies suggest that habitual SSD leads to weight gain and obesity over time.

Several possible reasons for this exist. As described below, habitual SSD may lead to metabolic changes and cardiometabolic risk factors. In addition, SSD could lead to neurocognitive changes that could result in weight gain, including impaired judgment and decision making, which could presumably alter food choice,⁴⁷ and fatigue and tiredness, which could result in decreased physical activity. Additionally, sleep loss may lead to increased food intake despite little change in energy expenditure, leading to a positive energy balance.⁴⁸

An emerging evidence base suggests that SSD is related to food intake. Among factory workers in Japan, SSD was associated with more snacking between meals, more irregular eating habits, less consumption of vegetables, and a greater preference for strongly flavored food.⁴⁹ In the United States, a study of 459 postmenopausal women enrolled in the Women's Health Initiative examined relationships between dietary records and sleep diary and actigraphy variables.⁵⁰ This study showed that SSD, measured objectively with actigraphy, was strongly associated with higher intakes of many dietary fat-related variables.

From nationally representative data, analysis of the 2007 to 2008 NHANES showed that self-reported long sleepers consumed fewer calories than 7- to 8-hour sleepers.⁵¹ Additionally, very short (< 5 hours), short (5–6 hours), and long sleepers consumed a smaller variety of foods (fewer number of foods consumed on the measurement day) compared with 7- to 8-hour sleepers. In an examination of nutrient profiles, very short sleep and long sleep were associated with lower consumption of protein, carbohydrate, fiber, and fat relative to 7- to 8-hour sleep (long sleepers consumed less sugar, and short sleepers consumed less fiber). These observations were made after adjustment for other dietary intake variables, including total caloric intake.⁵¹ Data thus suggest that SSD is associated with poor dietary quality.

Epidemiological data linking sleep duration with energy expenditure are relatively scarce. The reason may be that the relationship between sleep and energy expenditure is complex. For example, analysis of $> 300\,000$ respondents to the 2009 Behavioral Risk Factor Surveillance System showed that self-reported perceived sleep insufficiency was associated with self-reported physical activity variables but that this relationship was generally nonlinear. Moderate exercise was generally not associated with insufficient sleep for values up to ≈ 1 h/d, and vigorous physical activity was nonlinearly but generally associated with greater insufficient sleep, which was consistent with employment data showing that manual labor was associated with more insufficient sleep than

jobs that included mostly sitting or standing. In contrast, however, those engaging in no activity reported more insufficient sleep than those engaging in some activity.⁵²

This finding is consistent with data from a previous wave of the Behavioral Risk Factor Surveillance System that found that those individuals who reported any exercise in the past 30 days (regardless of amount) were about a third less likely to report sleep disturbances and about half as likely to report daytime tiredness.⁵³ This is consistent with data from the National Sleep Foundation that found that increased sedentary time was associated with poorer sleep quality.⁵⁴ Also consistent with the data on insufficient sleep, there was no relationship between sedentary time and sleep duration. It may even be the case that shorter sleepers get more exercise on average. In data from the 2007 to 2008 NHANES, minutes of moderate and vigorous exercise differed across sleep duration groups; the shortest sleepers reported the most exercise, and the longest sleepers reported the least.⁵¹

The relationships between sleep and energy expenditure are clearly complex and may not be well measured at the population level. For example, there may be subgroups of short sleepers who, by virtue of having more wake time, are more physically active, whereas others who sleep poorly have less energy and ability to be active. It seems to be the case, however, that decreased physical activity plays a role in the relation between sleep and CVD. In an analysis of data from the National Institutes of Health–AARP Diet and Health Study, SSD predicted CVD mortality, especially among overweight and obese people, and the combination of SSD, low physical activity, and overweight status was associated with an increased likelihood of all-cause, cardiovascular, and cancer-related mortality.⁵⁵

Epidemiological evidence thus clearly describes a relation between SSD and increased energy intakes. Much less clear is the relation between sleep duration and physical activity level. Given the association between SSD and obesity, however, the preponderance of evidence suggests a mismatch between energy intake and energy expenditure favoring a positive energy balance. This is assessed in clinical intervention studies.

Clinical Evidence

Impact of Sleep Restriction on Energy Intake

Several studies have assessed energy intake after periods of sleep restriction, usually 4 hours in bed, relative to periods of habitual sleep, usually 8 to 10 hours in bed.^{56–64} With 2 exceptions,^{57,60} studies have generally been performed in normal-weight individuals. In general, studies reported increases in 24-hour energy intake when participants underwent sleep restriction relative to habitual sleep (Table 1), with the degree of overeating ranging from ≈180 to 559 kcal/d. Few studies also

reported no difference in energy intake^{56,63,64} between sleep conditions. One of those studies was a small study with young men and women randomized to either sleep restriction or habitual sleep that found a significant increase in food intake with sleep restriction relative to baseline but no between-group difference.⁵⁶ The other 2 studies were performed exclusively in young, healthy men.^{63,64} One study reported greater hunger ratings but no difference in intake at a single late-afternoon buffet meal after a period of sleep restriction.⁶³ The other study reported greater fat intake but not total energy intake after sleep restriction.⁶⁴

Several mechanisms have been posited to explain the greater energy intake observed after periods of sleep restriction. Studies have reported alterations in appetite-regulating hormones, particularly leptin and ghrelin but also glucagon-like peptide 1 in women. However, data in this area are mixed,³⁴ with studies showing increased,^{57,59,65–68} reduced,^{69,70} or no difference in ^{56,63,64,71,72} leptin levels with sleep restriction. Similarly, ghrelin levels have been reported to be increased^{69,70,72–74} or not different^{56,60,64,71} with sleep restriction relative to habitual sleep. Only 1 study reported glucagon-like peptide 1,⁷² showing reduced levels with sleep restriction relative to habitual sleep in women but not in men. Neuroimaging studies have highlighted a role of the neuronal reward network in response to foods as an attempt to explain enhanced food intake after periods of sleep restriction.^{75–77} Conclusions from those studies are in line with data showing that participants select larger portion sizes, even in the sated state,⁷⁴ and report greater food purchases⁷⁸ in mock portion size and supermarket tasks, respectively, after a night of total sleep deprivation relative to an 8-hour sleep opportunity.

Impact of Sleep Restriction on Energy Expenditure

On the flip side of the energy balance equation, data on the effects of sleep duration on energy expenditure are also mixed.³⁴ This is due in part to the multicomponent nature of energy expenditure: total energy expenditure, resting metabolic rate, sleeping metabolic rate, physical activity energy expenditure, and diet-induced thermogenesis, as well as the multiple ways by which energy expenditure can be assessed: actigraphy/accelerometry, indirect calorimetry, metabolic chamber, and doubly labeled water. Studies that have examined the impact of sleep duration on resting metabolic rate using indirect calorimetry have found either no difference^{57,62,79–81} or a lower resting metabolic rate⁶⁹ with sleep restriction relative to habitual sleep. As part of a weight loss diet, sleep restriction leads to greater reduction in resting metabolic rate than habitual sleep.⁷³

When 24-hour energy expenditure was measured by doubly labeled water, no difference between sleep restriction relative to habitual sleep was observed.^{60,75}

Table 1. Impact of Sleep Restriction on Food Intake

Study	Participants	Sleep Intervention	Outcome (Sleep Restriction)
Benedict et al, ⁶³ 2011	14 Men	24 h with 8 h TIB or TSD	Greater morning hunger
	Normal weight		No difference in energy intake at afternoon buffet
	Mean age, 23 y		
Bosy-Westphal et al, ⁵⁷ 2008	14 Women	2 Nights of >8 h TIB followed by consecutive nights of 7, 6, 5, and 4 h TIB	Energy intake was increased by 415 kcal/d relative to baseline
	8 Normal weight, 3 overweight, 3 obese		
	Mean age, 27.5 y		
Brondel et al, ⁵⁸ 2010	12 Men	48 h with 8 or 4 h TIB	Energy intake was increased by 559 kcal/d
	Normal weight		
	Mean age, 22 y		
Calvin et al, ⁵⁶ 2013*	11 Men, 6 women	Reduce sleep to 2/3 habitual sleep or maintain habitual sleep, 8 d	Energy intake was increased by 559 kcal/d relative to baseline
	Normal weight		
	Mean age, ≈25 y		
Markwald et al, ⁵⁹ 2013	8 Men, 8 women	5 d of 5 or 9 h TIB	Energy intake was increased by 6% (182 kcal/d)
	Normal weight		
	Mean age, 22.4 y		
Nedeltcheva et al, ⁶⁰ 2009	6 Men, 5 women	14 d of 5.5 h or 8.5 h TIB	Total energy intake was increased by 297 kcal/d (NS); snack intake was increased by 221 kcal/d
	Overweight		
	Mean age, 39 y		
Schmid et al, ⁶⁴ 2009	15 Men	2 d with 4 or 8 h TIB	Energy intake was similar, but fat intake was increased
	Normal weight		
	Mean age, 27 y		
Spaeth et al, ⁶¹ 2013*	Sleep restriction: 15 men, 16 women	Sleep restriction: 1 baseline night (8 h TIB) followed by 4 nights of 4 h TIB and 2 nights of 10 h TIB Control, 10 h TIB	Energy intake was increased by ≈511 kcal/d when bedtime was delayed and sleep restricted to 4 h TIB
	Control: 4 men, 2 women		
	Normal weight to overweight		
	Mean age, ≈34 y		
St-Onge et al, ⁶² 2011	13 Men, 13 women	5 Nights of 4 h TIB or 9 h TIB	Energy intake was increased by 296 kcal/d
	Normal weight		
	Mean age, ≈35 y		

NS indicates not significant; TIB, time in bed; and TSD, total sleep deprivation.

*Parallel-arm study.

However, when 24-hour energy expenditure was measured in a metabolic chamber, which provides a more confined environment, energy expenditure was increased with sleep restriction relative to habitual sleep.^{59,82,83} The increase in energy expenditure associated with sleep restriction could be accounted for by the added cost of maintaining wakefulness.

Finally, free-living physical activity assessed by accelerometry seems to be minimally altered. Some have reported lower high-intensity activity and higher low-intensity activity with sleep restriction,^{62,64} whereas others^{56,57} have found no difference in physical activity energy expenditure. On the other hand, when sleep

duration was reduced as a result of sleep fragmentation, physical activity was increased.⁷¹ Such observations were also made in a crossover sleep restriction study, but the difference between sleep conditions was estimated to be an ≈48-kcal greater expenditure with sleep restriction.⁵⁸ Nonetheless, differences in energy expenditure with sleep restriction do not appear to be sufficient to counter the increase in energy intake reported above.

Data therefore do not support a fundamental alteration in energy metabolism as a result of SSD. In fact, as a result of added time awake, resting energy expenditure is increased when sleep is restricted. However,

clinical intervention studies have not provided sufficient information to conclude on the impact of sleep restriction on physical activity level. This area of research should be expanded.

Impact of Sleep Restriction on Body Weight

Studies that have assessed the impact of sleep restriction on change in body weight in the context of ad libitum feeding have mostly been small, short-term studies.^{57,59–61,73,84} One study⁵⁷ showed an increase of 0.4 kg in body weight in women as sleep was progressively restricted from >8 h/night at baseline to 4 h/night over 4 nights, and another study⁵⁹ found weight gain over a 5-night period of 5 hours in bed relative to 9 hours in normal-weight men and women in a crossover study. In a 2-arm study, individuals undergoing sleep restriction to 4 h/night for 5 nights gained more weight than those provided an 8-hour sleep opportunity, with the weight gain being greater in men than women and in blacks compared with whites.⁶¹ Longer-term studies, however, have not found an impact of sleep restriction on body weight. There was no difference in change in body weight assessed by dual energy x-ray absorptiometry when participants were provided a 5.5-hour sleep opportunity relative to 8.5 hours for 14 days as inpatients,⁶⁰ nor was body weight different from baseline after 3 weeks of a 1.5-hour sleep restriction relative to habitual sleep in an outpatient study conducted exclusively in men.⁸⁴ In the latter study, however, there was a significant treatment-by-week interaction such that weight was initially reduced in the sleep restriction group and rose back to baseline between weeks 2 and 3. It is unknown whether body weight would have continued in an upward trajectory had the study been prolonged beyond 3 weeks. Studies are needed to evaluate the longer-term impact of sleep restriction on energy balance with weight status and body composition as the outcome variables.

One study was conducted as a controlled-feeding weight loss study in which participants concurrently underwent sleep restriction or habitual sleep for 14 days in a crossover design.⁷³ Because of the controlled-feeding nature of the study, participants lost an equivalent amount of weight in both sleep conditions. However, in the sleep restriction condition, participants lost more fat-free mass and less fat mass than when the same weight loss treatment was performed with an 8-hour sleep opportunity. Whether similar results are observed in the context of weight gain remains to be determined.

Overall, there is good agreement that sleep restriction increases energy intake, and evidence shows that this is not accompanied by an adequate compensation via increased energy expenditure. Although data are mixed and somewhat inconclusive in terms of the impact of sleep restriction on energy expenditure, the net result seems to be positive energy balance, with studies show-

ing a strong impact of sleep restriction on increased food intake, particularly from fat and snacks,^{60,62,64} at least in the short term. Studies are needed to assess the longer-term impact of sleep restriction on energy balance with changes in body composition as the main outcome variable to truly determine whether SSD is a causal factor in the development of obesity. Studies should also include more women, older adults, and overweight or obese individuals, all of whom have been underrepresented in studies to date.

SLEEP DURATION, CARDIOMETABOLIC RISK, AND CLINICAL END POINTS

Epidemiological Findings

Epidemiological research has reported the association between sleep duration, quality, and cardiometabolic risk. These topics have been the subject of several meta-analyses, which are described below and summarized in Table 2. In fact, emerging cross-sectional data have shown that SSD is consistently associated with metabolic syndrome (a constellation of symptoms, including elevated waist circumference, lipids, fasting glucose, and blood pressure). Specifically, SSD is positively associated with the odds or risk of developing the metabolic syndrome,^{85–91} with similar metabolic effects being shown in individuals with long sleep duration.^{85,87}

Diabetes Mellitus

Two recent meta-analyses on the association between SSD and development of diabetes mellitus have been published.^{92,93} The findings were similar, showing an $\approx 30\%$ increased risk. The first, based on 7 studies, showed a relative risk (RR) of 1.28 (95% confidence interval [CI], 1.03–1.60) with evidence of heterogeneity; there was a significant effect in men but not women.⁹² The second meta-analysis, based on 10 studies, showed an odds ratio of 1.33 (95% CI, 1.20–1.48) without evidence of

Table 2. Associations Between Sleep Duration and Disorders and Incident CVD: Summary of Recent Meta-Analyses

Disorder	Short Sleep	Long Sleep	Insomnia	SDB
Diabetes mellitus	+	+	+	+
Hypertension	+	X	NA	NA
CHD	+	+	NA	X
Stroke	+	+	NA	+
Total CVD	X	+	+	+

CHD indicates coronary heart disease; CVD, cardiovascular disease; NA, no recently reported meta-analysis identified; SDB, sleep-disordered breathing; +, statistically significant positive relationship; and X, no statistically significant relationship.

heterogeneity.⁹³ Only 1 meta-analysis, based on 6 studies, found an association between long sleep duration and development of diabetes mellitus (RR=1.48; 95% CI, 1.13–1.96) without statistically significant heterogeneity.⁹² Potential confounders were variably adjusted for in the original studies included in these meta-analyses.^{89,90} The minority of studies included adjustment for snoring, but none adjusted for SDB.

Hypertension

The association between SSD and hypertension has also been well studied in 3 recent meta-analyses. Two showed similar results without evidence of heterogeneity (RR=1.21; 95% CI, 1.05–1.40 based on 6 studies⁹⁴; and RR=1.23; 95% CI, 1.06–1.42 based on 5 studies⁹⁵). However, the other meta-analysis, based on 6 studies, did not find an association between SSD and the risk of incident hypertension (RR=1.11; 95% CI, 0.84–1.47) with evidence of heterogeneity across studies.⁹⁶ The risk was increased among those <65 years of age but was not increased in those ≥65 years of age without heterogeneity within each sex subgroup.⁹⁶ Potential confounders were variably adjusted for in the original studies included in these meta-analyses.^{91–93} Unaccounted-for variables such as SDB could represent potential confounders of the SSD-hypertension association. Despite associations between long sleep duration and prevalent hypertension,⁹⁶ 3 meta-analyses, each based on 5 studies, have been reported recently, and none found an association between long sleep duration and the development of hypertension.^{94–96} Of note, whereas associations of SSD and some cardiometabolic risk factors are typically significant in young but not older adults, no difference was found between age groups <65 or >65 years of age in the association between long sleep and hypertension.⁹⁶

Cardiovascular Disease

A meta-analysis based on 7 studies demonstrated an increased risk of developing or dying of CHD associated with both SSD (RR=1.48; 95% CI, 1.22–1.80) and long sleep duration (RR=1.38; 95% CI, 1.15–1.66) with some heterogeneity.⁹⁷ Sex, geographical area, and duration of follow-up were explored as potential causes of heterogeneity, but none contributed to this effect. Sleep-inducing medications, perhaps used more frequently in those with SSD, could modify the effect of SSD on CHD or serve as a potential confounder of the SSD-CHD association but were not explored in these meta-analyses. In a separate study,⁹⁸ SSD and CHD mortality were associated among those who used tranquilizers or hypnotics regularly or rarely but not in those who never used them, supporting the possibility of effect modification.

A meta-analysis based on 4 studies identified a modest association between SSD and incident stroke (RR=1.15; 95% CI, 1.00–1.31) with no heterogeneity across studies.⁹⁷ In this meta-analysis, there was also an association between long sleep duration and stroke

(RR=1.65; 95% CI, 1.45–1.87) with no heterogeneity across studies.⁹⁷

A meta-analysis found no association between SSD and total CVD (including any CVD-related cause of death) despite associations with CHD and stroke with no heterogeneity across studies.⁹⁷ There was, however, an association between long sleep duration and the combination of incident CVD and CVD mortality (RR=1.41; 95% CI, 1.19–1.68) with heterogeneity across studies that was not attributable to sex, follow-up duration, or geographical location. Of note, a recent study not included in this meta-analysis showed a significant association between SSD, but not long sleep duration, and CVD in less healthy individuals (hazard ratio [HR]=1.38; 95% CI, 1.12–1.70), defined as having a serious illness at baseline or Medical Outcomes Study Physical Functioning score <75.⁹³ There was no association in healthy individuals (HR=0.92; 95% CI, 0.75–1.14).

Impact of Sleep Restriction on Cardiometabolic Risk Factors

Insulin Resistance

Sleep restriction contributes to and is associated with a variety of adverse metabolic outcomes. Experimental sleep restriction in healthy adults results in increases in insulin resistance⁹⁹ and decreases in insulin sensitivity.⁸¹ SSD is associated with increases in fasting insulin and hemoglobin A_{1c}, which may be mediated by body mass index.¹⁰⁰ Sleep restriction is associated with a 3-fold increase in the odds of having impaired fasting glucose,²⁶ which may be driven by insulin resistance.^{26,84} Under controlled laboratory conditions, consecutive daily sleep restriction of ≈2 hours results in increased insulin resistance and decreased glucose tolerance in response to a glucose challenge.¹⁰¹

Cardiovascular and Proinflammatory Markers

Researchers have examined the effect of sleep restriction on blood pressure outcomes (systolic, diastolic, and nocturnal blood pressure dipping, a disturbance in the normal circadian blood pressure pattern). In a crossover trial, healthy younger and older adults were randomized to either 24 hours including habitual sleep or 24 hours of total sleep deprivation. Normotensive older adults experienced a 13–mmHg increase in systolic blood pressure and a 7–mmHg increase in diastolic blood pressure after sleep deprivation compared with habitual sleep.¹⁰² Nocturnal blood pressure dipping also decreases after restricted sleep (<6.5 hours),^{103,104} which may further increase CVD risk.

Sleep restriction has been shown to affect a variety of other cardiovascular markers. In a randomized, crossover trial of younger healthy adult men, 5 nights of restricted sleep (<5 hours) in a laboratory setting resulted in an increase in sympathetic nervous system activity

(increased low-frequency heart rate variability and decrease in high-frequency heart rate variability), an increase in serum norepinephrine, and a decrease in maximum endothelium-dependent venodilation compared with 5 nights of habitual sleep of >7 hours.¹⁰⁵ Similarly, 40 hours of total sleep deprivation under controlled laboratory conditions resulted in increases in proinflammatory markers (intercellular adhesion molecule-1, e-selectin, interleukin-1 β , interleukin-6, C-reactive protein) and a decrease in interleukin-1 receptor antagonist (an anti-inflammatory marker).¹⁰⁶ Relatedly, healthy adults subjected to restricted sleep (4 hours of time in bed) over 5 consecutive nights experienced an increase in proinflammatory cytokines (interleukin-6, interleukin-1 β , interleukin-17, mRNA) compared with those who underwent 8 hours of habitual time in bed.¹⁰⁷ Finally, in a randomized, crossover trial, young normal-weight adults who underwent 5 nights of SSD (<4 hours) or 5 nights of habitual sleep (>9 hours) showed no significant differences in lipid profile between sleep conditions.

SLEEP DISORDERS AND CARDIOMETABOLIC RISK

Epidemiological Findings

Diabetes Mellitus

No meta-analysis was available to assess the relation between insomnia and type 2 diabetes mellitus. However, a meta-analysis based on 5 studies corroborated an association between insomnia symptoms such as difficulty initiating sleep and the development of diabetes mellitus (RR=1.57; 95% CI, 1.25–1.97) and difficulty maintaining sleep and incident diabetes mellitus (RR=1.84; 95% CI, 1.39–2.43).⁹² No heterogeneity was noted across studies.

A meta-analysis based on 6 studies addressed the association between SDB and diabetes mellitus.¹⁰⁸ This analysis found that moderate to severe SDB (apnea/hypopnea index [AHI] \geq 15) was associated with a greater risk of the development of diabetes mellitus compared with the absence of SDB. Data from 3 studies of individuals with mild SDB compared with those without SDB showed that there was no association with the development of diabetes mellitus (RR=1.22; 95% CI, 0.91–1.63).

Hypertension

Studies that have investigated the association between insomnia symptoms and the development of hypertension have shown mixed or nuanced results. Based on subjective symptoms only, a modest association between combined insomnia symptoms (RR=1.05; 95% CI, 1.01–1.08)⁹⁴ or some combination of symptoms such as difficulty falling asleep or waking up repeatedly and the development of hypertension has been found.¹⁰⁹ Although another study in a relatively healthy older cohort showed that insomnia symptoms were protective

against the development of hypertension in adjusted models only in men who were not black, no association was identified in women or blacks.¹¹⁰ The association of sleep disturbance symptoms is complicated by an interaction with objective measures of sleep duration. For instance, chronic insomnia with \geq 6 hours of objective sleep was not associated with the development of hypertension, whereas those symptoms, in combination with SSD, were associated with the development of hypertension when adjusted for potential confounders.¹¹¹ Another study found that insomnia was associated with the development of hypertension after adjustment for sleep duration in individuals 32 to 59 years of age but not in those 60 to 86 years of age.¹¹²

A previous American Heart Association scientific statement reviewed the evidence in support of a relation between SDB and hypertension, referring to SDB as an identifiable cause of hypertension.¹¹³ Longitudinal studies since that time have confirmed an association between severe SDB (AHI \geq 30) and new-onset hypertension in the elderly¹¹⁴ but did not show an association between objectively measured SDB and incident hypertension in middle-aged adults after adjustment.¹¹⁵ Furthermore, the Sleep Heart Health Study demonstrated an association between an AHI \geq 30 and the development of hypertension, but this association was no longer significant after adjustment for body mass index. Associations were present after adjustment in women and those with a body mass index \leq 27.3 kg/m².¹¹⁶

Cardiovascular Disease

Studies that have investigated the association between insomnia symptoms and CHD have generally found an association, although sex differences may exist. Restless, disturbed nights (a combination of response options of “rather more than usual” and “much more than usual”) have been found to be associated with CHD after adjustment for confounders.¹¹⁷ After adjustment for potential confounders, including depression and anxiety, difficulty initiating sleep almost every night, difficulty maintaining sleep almost every night, and a feeling of nonrestorative sleep more than once a week were associated with acute myocardial infarction (MI) compared with never experiencing these sleep difficulties.¹¹⁸ In this study, the number of insomnia symptoms was associated with MI risk in a dose-dependent fashion.¹¹⁸ The relationship between insomnia symptoms and MI appeared to be stronger in women than men.¹¹⁸ In another study with full adjustment, incident MI was associated with difficulty maintaining sleep in women but not men. Difficulty initiating sleep was not significantly associated with CHD in women or men.¹¹⁹ Additionally, in an all-male study, frequent insomnia was no longer significantly associated with CHD after adjustment for potential confounders.¹²⁰

Few studies have addressed the association between insomnia and incident stroke, but in general, the results

have supported an association. In an all-male prospective cohort study, frequent insomnia was associated with incident stroke after adjustment for potential confounders.¹²⁰ Similarly, in a study of working-aged adults, insomnia symptoms were associated with self-reported stroke after adjustment for demographics, anxiety, and depression.¹²¹ Finally, a retrospective cohort study found that insomnia was associated with hospitalization for stroke on the basis of diagnosis codes.¹²²

A meta-analysis based on 13 studies corroborated the association between insomnia symptoms and developing or dying of CVD (RR, 1.45; 95% CI, 1.29–1.62).¹²³ However, 1 study identified an association between insomnia symptoms and CVD only in those with both SSD and poor-quality sleep.¹²⁴

SDB is a group of sleep pathologies that include OSA and is characterized by abnormal respiratory patterns during sleep. Although SDB, detected by polysomnography, has been linked to the development of CHD, the association does not appear to be statistically significant.¹²⁵ Three meta-analyses, each with evidence of at least moderate heterogeneity, showed nonsignificant associations.^{125–127} A cohort study in women published only since the meta-analyses supported the nonsignificant association between untreated SDB and the development of CHD compared with no SDB (AHI <10).¹²⁸

In a meta-analysis, studies using both clinical populations referred for polysomnography because of suspicion of SDB and community-based samples have shown an association between SDB and the development of ischemic stroke.¹²⁹ The overall association was an RR of 2.10 (95% CI, 1.50–2.93) in 10 studies.¹²⁹ Other meta-analyses have shown a similar association between severe SDB and stroke.^{125,126} It has been noted that better evidence exists in men.¹²⁵ In the single study available in women only, the association was not significant.¹²⁵ However, since these meta-analyses were published, a prospective cohort study performed in women found a clear association between SDB and incident stroke (HR=6.44; 95% CI, 1.46–28.3).¹²⁸

For the combined outcome of CVD, moderate and severe SDB, but not mild SDB, was shown to be associated with an elevated risk.¹²⁶ The pooled RR of CVD per 10-unit increase in AHI was 1.17 on the basis of 6 prospective cohort studies.

Another sleep disorder, restless leg syndrome, has been investigated as a CVD risk factor. Although findings from prospective cohort studies have been mixed with respect to the association between restless leg syndrome and the development of CVD, the majority of the studies have been negative. No association between restless leg syndrome and stroke, MI, or major cardiovascular events was found in age-adjusted or multivariable-adjusted analyses from 2 large, single-sex, prospective cohort studies of health professionals, the Physicians Health Study and the Women's Health Study.¹³⁰ In an all-male study, the symptom of restless legs was not associated

with CHD after adjustment for potential confounders.¹²⁰ In the Nurses' Health Study, longer duration of restless leg syndrome diagnosis (>3 years) was associated with CHD and nonfatal MI in adjusted models in women.¹³¹ The diagnosis of restless leg syndrome for <3 years at baseline was not associated with the development of CHD or MI.¹³¹ In an all-male study, the symptom of restless legs (restless legs or bothersome twitches once or twice a week or more) was associated with the development of stroke¹²⁰ after adjustment for potential confounders. In those with end-stage renal disease on hemodialysis, a population with a high prevalence of restless legs, neither continuous restless leg syndrome symptoms nor intermittent symptoms were associated with the development of a new CVD event in adjusted models.¹³²

A retrospective cohort study using administrative data showed that among those who had a polysomnogram for suspected SDB, the number of periodic leg movements per hour (13.4 vs 0) was associated with composite CVD events but not with MI or stroke.¹³³ For CHD, an association was identified for a periodic limb movement index ≥ 30 in an unadjusted model (HR=1.38; 95% CI, 1.07–1.79) but not when adjusted for confounders (HR=1.26; 95% CI, 0.97–1.65). For cerebrovascular events, no association was found in unadjusted or adjusted analyses, although there were few events and thus limited power.¹³⁴ In addition, despite an association between periodic limb movement index and prevalent hypertension, an association was not found with incident hypertension among community-dwelling elderly men.¹³⁴

Preliminary data suggest that sleep extension may have the potential to improve cardiovascular risk factors.¹³⁵ In a small pilot study, 22 subjects with prehypertension or stage 1 hypertension and sleep durations of ≤ 7 hours were randomized to sleep extension or maintenance groups, with a goal of increasing sleep duration by 1 hour over 6 weeks. Although there was a reduction in blood pressure in both groups, it was greater in the group randomized to sleep extension (although the difference between groups was not statistically significant). Larger studies will be necessary to evaluate this approach more rigorously. Similarly, small studies suggest that there may be an effect of treating insomnia by behavioral interventions in reducing the levels of the inflammatory marker C-reactive protein.¹³⁶ Again, larger, more rigorous trials with cardiovascular end points will be necessary to make these findings clinically relevant.

LIFESTYLE INTERVENTIONS FOR SLEEP DISORDERS

Impact of Weight Loss on Sleep Disorders

The results of 4 randomized, controlled trials have shown that weight loss achieved through behavioral or surgical interventions may be effective in the man-

agement or resolution of SDB (measured by change in AHI).^{137–140} In 1 trial, researchers found that a 20-kg (95% CI, 18–21) weight loss achieved by a very-low-calorie diet resulted in a significant reduction in AHI at 9 weeks (-25 ± 17 events/h) compared with usual diet controls (-2 ± 11 events/h).¹³⁹ In a prospective observational follow-up of the same trial participants during a weight maintenance period, significant improvements in AHI were maintained at 52 weeks (-17 events/hour; 95% CI, -13 to -21 events/h) compared with baseline.¹³⁹ Ten percent of those in the intervention group achieved complete resolution of OSA at 12 months, with 48% no longer requiring continuous positive airway pressure (CPAP). Furthermore, individuals with severe OSA (AHI >30 events/h) achieved greater reductions in AHI after weight loss (-38 events/h) compared with those with moderate OSA (-12 events/h).¹³⁹ Results of another trial (ancillary to the LookAhead trial) found that intensive lifestyle intervention produced a significantly greater reduction in AHI (-9.7 ± 2 , -8 ± 2 , and -7.7 ± 2.3 events/h at 12, 24, and 48 months, respectively), with complete resolution of OSA being 5 times more likely in the intervention group compared with the control group.¹³⁷ In another trial, individuals with mild OSA (AHI=5–15 events/h) received 12 weeks of a very-low-calorie diet plus lifestyle counseling (n=26) or lifestyle counseling alone (n=26).¹⁴⁰ Although these researchers found a 5.4-kg/m² difference in body mass index between the intervention and control groups at follow-up, no significant difference was shown in AHI or associated nasal resistance. Notably, these authors did not report attrition rates or whether intent-to-treat analyses were performed, which limits the interpretation of these findings. Lastly, in a comparative-effectiveness trial, obese adults with moderate OSA (AHI=17±21 events/h) randomized to lifestyle intervention or roux-en-y gastric bypass surgery lost 8% and 30% of baseline weight respectively, at 12 months. Significant reductions in AHI were also observed, with larger reductions achieved after roux-en-y gastric bypass.¹³⁸ However, baseline body mass index was lower in the surgery group compared with the lifestyle group, and only 63% of participants had OSA (AHI >5 events/h) at baseline, which limits the conclusions that can be drawn.

In a series of observational cohort studies, weight loss has also shown promise for the resolution or improvement in SDB. Behavioral weight loss interventions¹⁴¹ supplemented with subutramine¹⁴² have resulted in significant reductions in both weight and AHI after 12 weeks¹⁴¹ and 24 weeks¹⁴² of intervention. In contrast, a 16-week intensive lifestyle intervention supplemented with low-calorie meal replacements did not result in significant improvements in AHI for obese adults with moderate OSA (AHI=24±12 events/h).¹⁴³ In the population of obese adults with OSA who have undergone bariatric surgery, researchers have evaluated the effects of a variety of procedures, includ-

ing the laparoscopic adjustable gastric band, roux-en-y gastric bypass, vertical sleeve gastrectomy, or intragastric balloon. Laparoscopic adjustable gastric band has resulted in the resolution of OSA in 60%¹⁴⁴ and 83%¹⁴⁵ of cases, with others reporting 100% of cases discontinuing the use of CPAP¹⁴⁶ and others reporting clinically significant improvements in AHI.^{147–150} Roux-en-y gastric bypass has resulted in the resolution of OSA in 75%¹⁵¹ and 92%¹⁴⁵ of cases, with significant improvements in AHI similarly being achieved.^{145,149,151–154} Vertical sleeve gastrectomy has resulted in the resolution of OSA in 92%,¹⁴⁵ 76%,¹⁵⁵ and 52%¹⁵⁶ of cases, with others reporting clinically significant improvements in AHI.^{148,149} Lastly, in a cohort of men who received the intragastric balloon, a significant improvement in AHI was reported: 52 events/h at baseline versus 12 events/h after 6 months.¹⁵⁷ Although several of these studies have reported improvements in SDB after weight loss, some did not report the observed changes in AHI.^{144–146,148,150,151,156,158}

Together, this emerging body of literature suggests that weight loss achieved through behavioral or surgical interventions may be effective for improving and in some cases resolving SDB. The findings from behavioral interventions are supported by 2 recent meta-analyses.^{159,160} This finding may be particularly true for those with severe OSA. However, well-designed and -executed comparative-effectiveness trials are needed that will further explore the underlying mechanisms between weight loss and SDB and are transparent in the reporting of objectively measured SDB outcomes (ie, changes from baseline to posttreatment follow-up in AHI).

Impact of Treatment of Sleep Disorders on Cardiometabolic Risk Factors

Although observational studies have found an association between the treatment of sleep disorders and an improvement in cardiometabolic risk factors, primarily hypertension,^{161–164} the randomized data are still rather limited. The HeartBEAT study (Heart Biomarker Evaluation in Apnea Treatment) was a randomized trial of 318 patients at 4 academic sites that assessed the effects of CPAP versus nocturnal supplemental oxygen versus usual care on cardiovascular risk factors in patients who had CVD or multiple cardiac risk factors who were found to have OSA.¹⁶⁵ Patients in cardiovascular practices were screened for sleep apnea with the Berlin questionnaire,¹⁶⁶ and those with an AHI of 15 to 50 events/h were randomized. Patients with severe sleep apnea were excluded. The primary end point of 24-hour mean arterial pressure at 12 weeks was significantly reduced in the CPAP group compared with the control group (-2.4 mmHg) or the supplemental oxygen group (-2.8 mmHg). However, there was no significant reduction in the supplemental oxygen group compared with

the control group. Of note, the nocturnal systolic blood pressure was significantly reduced for each additional hour of CPAP use (-0.93 mmHg/h). Furthermore, the odds of nondipping nocturnal blood pressure were reduced for each additional hour of CPAP use. Interestingly, compared with the control arm, CPAP use was associated with a lower adjusted level of C-reactive protein. There was no difference between CPAP treatment and supplemental oxygen groups in C-reactive protein levels at 12 weeks.

The results of HeartBEAT show the potential value of screening for sleep apnea in patients at elevated cardiovascular risk, including those whose cardiovascular risk factors are otherwise well treated.¹⁶⁵ In this setting, identification and treatment of OSA with CPAP reduce blood pressure by an amount that would be expected to translate into a reduction in cardiovascular events in a large enough population followed up for sufficient time. Additional analyses from HeartBEAT such as an examination of the impact of treatment on quality-of-life measures are ongoing.

A meta-analysis of 31 randomized trials comparing CPAP with various passive and active controls confirmed a significant effect of this treatment on blood pressure.¹⁶⁷ There was a highly significant net difference in systolic blood pressure (2.6 mmHg) and in diastolic blood pressure (2.0 mmHg) for CPAP compared with the control. In the studies that had 24-hour ambulatory blood pressure monitoring data available, the difference between treatment arms in systolic blood pressure was 2.2 mmHg and in diastolic blood pressure was 1.9 mmHg during the daytime period and 3.8 and 1.8 mmHg, respectively, during the nighttime period. A higher AHI appeared to be associated with a greater decrease in systolic blood pressure. The results of this meta-analysis support that CPAP has a significant, albeit modest, effect on blood pressure.

Although most (but not all) studies and the above meta-analysis support a modest effect of CPAP on blood pressure, the effect on biomarkers associated with cardiovascular risk such as C-reactive protein has been more variable.^{165,168–170} A recent randomized trial of CPAP versus best supportive care found that CPAP was associated with significant reductions in total and low-density lipoprotein cholesterol levels at 3 months but not at 12 months.¹⁷¹ Such potentially beneficial effects on lipid-related parameters have not been consistently observed.¹⁷² Further randomized study is needed of the effects of CPAP on cardiovascular risk factors and risk markers, although the most consistent and robust effect of CPAP to date appears to be on blood pressure.

Racial Disparities

Despite many advances in medicine, significant health disparities in the population remain. Many individuals be-

longing to racial/ethnic minority groups, and those who are socioeconomically disadvantaged face systematic discrepancies in their risk of adverse health outcomes and decreased life expectancy.^{173,174} For example, the prevalence of obesity is higher among blacks than non-Hispanic whites.^{175,176} In addition, rates of hypertension are much higher among blacks^{177,178} and rates of diabetes mellitus are disproportionately higher among blacks and Hispanics/Latinos¹⁷⁹ than non-Hispanic whites. Race/ethnicity and socioeconomic status are important risk factors for cardiometabolic disease morbidity and mortality. As mentioned, sleep is associated with aspects of cardiometabolic disease and may represent an important risk factor for morbidity and mortality in these domains. It is important to note that in addition to cardiometabolic health disparities, racial/ethnic minorities are more likely to experience sleep duration outside of 7 to 8 hours, especially an SSD of ≤ 6 hours,^{180–185} as well as increased prevalence of sleep disturbances,^{186,187} and these relationships interact with socioeconomic status.^{184,186,188,189} These relationships are consistent with the socio-ecological model of sleep and health, which places sleep at the interface of downstream cardiometabolic effects and upstream social/behavioral determinants.^{188,190,191}

There is also emerging evidence that the relations between sleep and cardiometabolic disease risk factors may depend on race/ethnicity. For example, data from the CARDIA study (Coronary Artery Risk Development in Young Adults) showed that 5-year blood pressure change was mediated by race/ethnicity differences in sleep duration.²¹ Additionally, data from NHANES showed that the U-shaped relationship between sleep duration and C-reactive protein levels was different across race/ethnicity groups, with a U-shaped relationship seen in non-Hispanic whites, elevations in short sleep seen in blacks, a pseudolinear relationship (lower levels in short sleep and higher levels in long sleep) seen in Asians/others, and no relation seen in Hispanics/Latinos.¹⁹² Further data from NHANES showed that these 4 groups also differed in their relationships between habitual sleep duration and both subjectively and objectively determined prevalence of obesity, hypertension, hypercholesterolemia, and diabetes mellitus.⁴¹ Although the specific reasons for these differences are not clear, previous work has shown that factors such as exposure to racism^{193–198} and low childhood socioeconomic status,¹⁹⁹ as well as other factors,¹⁸⁸ may play roles.

Taken together, these results indicate that sleep may play an important role in health disparities and may represent a modifiable risk factor (along with diet and physical activity) for cardiometabolic risk in general and cardiometabolic health disparities specifically. Further research is needed to clarify these potential roles.

STATEMENT SUMMARY

Our review of the epidemiological data on the impact of sleep duration and disorders on cardiovascular health suggests the following:

1. Both short- and long-duration sleep and sleep disorders such as SDB and insomnia are associated with adverse cardiometabolic risk profiles and outcomes.
2. Sleep restriction has a negative impact on energy balance, but it is less clear whether treating sleep disorders has a positive impact on obesity risk.
3. Treating those with sleep disorders may provide clinical benefits, particularly for blood pressure.

CLINICAL RECOMMENDATIONS

The American Academy of Sleep Medicine and the Sleep Research Society recently released a statement in favor of ≥ 7 hours of sleep per night for adults “to promote optimal health.”¹⁹¹ Similarly, Healthy People 2020 has released a series of sleep health goals, including to “increase the proportion of adults who get sufficient sleep.” To increase the clinical awareness of and action on sleep-related issues and disorders, the following steps should be considered:

1. The American Heart Association should directly address sleep behavior in a public health campaign to promote ideal cardiac health (akin to its Simple 7 campaign addressing blood pressure, cholesterol, blood sugar, physical activity, diet, weight, and smoking cessation).
2. A public health campaign addressing sleep behavior should include explicit guidelines for adequate sleep and suggestions for how to include screening for sleep duration and sleep disorders in routine clinical care and public health settings.
3. Existing simple assessment tools to screen for sleep apnea risk should be better integrated into routine clinical care and public health settings.

FUTURE RESEARCH PRIORITIES

The increase in observational and clinical studies examining the link between sleep duration and disorders and cardiometabolic health increases the likelihood that we may soon have clearer recommendations and guidelines that will influence clinical practice and public health campaigns. In addition, more research is needed, and research priorities include the following:

1. Inclusion of more diverse populations in research studies (ie, minorities, women, and overweight and obese participants)
2. Longer-term follow-up of participants in observational and clinical studies
3. Accurate and objective measures of sleep behavior, along with sleep architecture

4. Evaluations of the impact of other sleep disorders, notably restless leg syndrome and periodic limb movement disorder, on cardiometabolic risk
5. Development and evaluation of simple sleep behavior screening tools that could be used in busy clinical or public health settings
6. Evaluation of brief intervention strategies in busy clinical or public health settings

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FOOTNOTES

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

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*Modest.

†Significant.

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*Modest.

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**Sleep Duration and Quality: Impact on Lifestyle Behaviors and Cardiometabolic Health:
A Scientific Statement From the American Heart Association**

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On behalf of the American Heart Association Obesity, Behavior Change, Diabetes, and
Nutrition Committees of the Council on Lifestyle and Cardiometabolic Health; Council on
Cardiovascular Disease in the Young; Council on Clinical Cardiology; and Stroke Council

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