

Sleep and Cardiometabolic Health: A Narrative Review of Epidemiological Evidence, Mechanisms, and Interventions

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Background: Short or poor-quality sleep can disrupt glucose metabolism, blood pressure regulation, appetite hormones, and inflammatory pathways, potentially accelerating weight gain, atherosclerosis, and insulin resistance. However, this multidimensional role of sleep in cardiometabolic disease prevention and management remains underappreciated. Existing literature has not fully synthesized how various aspects of sleep influence the spectrum of cardiometabolic conditions, nor how improving sleep might confer health benefits.

Objective: This narrative review examines epidemiological evidence linking sleep health to cardiometabolic outcomes, elucidates biological mechanisms underlying these associations, and discusses interventions aimed at optimizing sleep for cardiometabolic benefit.

Methods: We searched and reviewed recent studies and meta-analyses on sleep duration, sleep quality, insomnia, obstructive sleep apnea (OSA), and circadian disruption in relation to obesity, metabolic syndrome, diabetes, cardiovascular diseases, and mortality.

Findings: Abnormal sleep patterns are consistently associated with elevated risks of obesity, type 2 diabetes, hypertension, cardiovascular events, and all-cause mortality. Habitual short sleep predicts higher incidence of metabolic syndrome and type 2 diabetes, while chronic insomnia is linked to 45% greater odds of developing or dying from cardiovascular disease. Conversely, healthy sleep—typically 7–9 hours of regular, high-quality sleep per night—is associated with favorable cardiometabolic profiles and has been added to public health cardiovascular metrics. Mechanistically, inadequate sleep provokes insulin resistance, sympathetic overactivity, hormonal appetite changes, and systemic inflammation, which synergistically promote cardiometabolic disease.

Conclusion: Prioritizing healthy sleep habits and treating sleep disorders should become integral to lifestyle medicine for cardiometabolic health. Emerging intervention trials suggest that extending sleep or treating OSA can modestly improve weight control, glycemic regulation, and blood pressure.

Keywords: cardiometabolic diseases, interventions, mechanisms, sleep

Introduction

Cardiometabolic disorders encompass a range of interrelated chronic diseases including cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), metabolic syndrome (MetS), and related conditions like non-alcoholic fatty liver disease (NAFLD). Collectively, these disorders account for enormous morbidity and mortality worldwide. Abnormal sleep patterns (insufficient or excessive sleep, insomnia, OSA, and misaligned sleep timing) are consistently associated with elevated risks of obesity, T2DM, hypertension, cardiovascular events, and all-cause mortality.^{1,2} Habitual short sleep (<6 h/night) predicts higher incidence of metabolic syndrome and T2DM,³ while chronic insomnia is linked to 45% greater odds of developing or dying from cardiovascular disease.⁴ CVD remains the leading cause of death globally, responsible for an estimated 19.8 million deaths in 2022.⁵ T2DM afflicts over 500 million adults and, along with obesity, is rising in prevalence, contributing substantially to disability-adjusted life years (DALYs), healthcare costs, and reduced quality of life. MetS—the clustering of central obesity, hypertension, dyslipidemia, and hyperglycemia—affects roughly

one-quarter of adults and confers a twofold higher risk of heart disease, stroke, and diabetes. NAFLD, the hepatic manifestation of MetS, has become the most common cause of chronic liver disease and is associated with increased cardiovascular and liver-related mortality.^{6–8} Beyond their clinical impact, these cardiometabolic conditions carry immense financial and social burdens.

Importantly, cardiometabolic disorders are largely lifestyle-related and preventable. Classic modifiable risk factors include unhealthy diet, physical inactivity, smoking, and excess alcohol use. Public health efforts targeting these behaviors have yielded improvements; however, an often-overlooked lifestyle factor is sleep. Inadequate sleep has emerged as a significant and independent contributor to cardiometabolic risk. Sleep health is a multidimensional construct that includes sleep duration (quantity), sleep quality (depth and continuity of sleep, absence of insomnia or disturbances), sleep timing and regularity (alignment with circadian rhythms), and the absence of sleep disorders (such as obstructive sleep apnea). Daytime behaviors like habitual napping may also influence cardiometabolic outcomes. All these facets of sleep are potentially modifiable, making sleep an attractive target for prevention. Indeed, the American Heart Association now recognizes sleep duration as one of “Life’s Essential 8” cardiovascular health metrics, highlighting that 7–9 hours of consistent sleep per night is ideal for adults.¹ Still, beyond duration, other dimensions of sleep health are crucial but underappreciated in clinical practice.

Despite growing evidence linking poor sleep to cardiometabolic ills, a gap remains in translating this knowledge into integrated lifestyle guidance. Previous reviews have typically focused on single outcomes (eg sleep and obesity) or specific disorders (eg OSA), leaving a need for a comprehensive overview of how all aspects of sleep affect the spectrum of cardiometabolic diseases. Therefore, the objective of this narrative review is to synthesize epidemiological evidence on the associations between various sleep health components and cardiometabolic disorders, to examine underlying mechanisms, and to discuss interventions and clinical implications. We aim to provide clinicians and public health practitioners with an updated perspective on why and how optimizing sleep can help prevent and manage cardiometabolic disease. The sections below address each major cardiometabolic outcome in relation to sleep, outline biological pathways linking sleep to metabolic and cardiovascular dysfunction, and explore the impact of improving sleep through lifestyle changes or treatment of sleep disorders.

Sleep and Cardiometabolic Health: Epidemiological Evidence

A robust body of epidemiological research links abnormal sleep patterns with adverse cardiometabolic outcomes. Both insufficient and excessive sleep have been associated with increased risk for obesity, diabetes, and cardiovascular events, though specific relationships vary by outcome. In this section, we summarize associations between different components of sleep health—including sleep duration (short or long sleep), sleep quality (insomnia symptoms, sleep efficiency), sleep timing (circadian alignment or shift work), and sleep disorders (particularly OSA and chronic insomnia)—and major cardiometabolic disorders: obesity, metabolic syndrome, type 2 diabetes, NAFLD, hypertension, coronary heart disease, stroke, atrial fibrillation, heart failure, and mortality.

Obesity and Metabolic Syndrome

Short sleep duration is consistently associated with increased adiposity and risk of obesity. Cross-sectional studies show that habitually short sleepers (<6 hours per night) have higher body mass index (BMI) and waist circumference on average than those who sleep 7–8 hours.⁹ Prospective cohort studies indicate that chronic sleep curtailment contributes to gradual weight gain. In the large Nurses’ Health Study, women who slept ≤ 5 hours had a significantly higher risk of major weight gain over 16 years compared to those sleeping 7 hours.¹⁰ Mechanistically, even modest sleep deprivation can disrupt appetite regulation and increase caloric intake. These findings are supported by intervention trials: in a randomized trial, extending nightly sleep by 1.2 hours in short-sleeping adults led to a spontaneous 270 kcal/day decrease in intake and mild weight loss over two weeks.¹¹ Such evidence underscores that insufficient sleep is a contributing cause of obesity, and improving sleep may aid weight control.

Poor sleep quality and insomnia symptoms have similarly been linked to metabolic risk. Individuals who frequently experience non-restorative sleep or difficulty staying asleep are more prone to metabolic syndrome. In one meta-analysis, insomnia (as a clinical disorder) was associated with 1.5-fold higher odds of developing MetS.¹² Short sleep and insomnia

often co-occur, compounding their effects. A large Chinese cohort found that those with both insomnia and ≤ 6 hours sleep had a higher incidence of hypertension and MetS than those with neither condition.¹³ Regarding daytime napping, findings are mixed – long or frequent daytime naps in some studies correlate with higher MetS prevalence, especially in older adults, potentially due to underlying poor nighttime sleep or comorbidities.¹⁴ In contrast, short naps (<30 min) might be neutral or even beneficial for stress reduction; more research is needed to clarify this in relation to metabolic health.

Type 2 Diabetes Mellitus

Habitual short sleep is a well-established risk factor for type 2 diabetes. Meta-analyses of prospective studies have reported that people who sleep less than 6 hours per night have 30% higher risk of developing T2DM compared to 7–8-hour sleepers.⁷ Long sleep (>9 hours) has also been associated with elevated diabetes risk,¹⁵ though long sleep may reflect underlying illness or low socioeconomic status. A recent systematic review (2025) confirmed a U-shaped relationship between sleep duration and incident T2DM, with both short (<5–6 h) and long (>8–9 h) sleep linked to higher risk.¹⁶ Notably, one large US cohort found those sleeping under 6 hours were 2 times more likely to develop diabetes than those sleeping 7–8 hours.¹⁷ Poor sleep quality and OSA further exacerbate diabetes risk by inducing chronic intermittent hypoxia and sleep fragmentation, which promote insulin resistance. Over half of T2DM patients have coexisting OSA, and untreated OSA is associated with worse glycemic control (higher HbA1c). These epidemiological links are bolstered by experimental evidence that sleep restriction impairs glucose tolerance. A wealth of evidence implicates inadequate or disturbed sleep in the development of T2DM.

Non-Alcoholic Fatty Liver Disease (NAFLD)

Increasing attention has turned to the role of sleep in NAFLD, a condition closely tied to obesity and metabolic dysfunction. Several studies suggest that short sleep duration may elevate the risk of NAFLD. A meta-analysis by Wijarnpreecha et al⁷ found that individuals sleeping ≤ 6 hours had higher odds of NAFLD (pooled odds ratio 1.3) compared to normal sleepers. Similarly, a meta-analysis by Shen et al⁸ in 2016 reported a significant association between short sleep and fatty liver disease. However, not all analyses agree, and results have been somewhat conflicting.³ More recent prospective data strengthen the link: in a cohort of >86,000 Korean adults, those who developed incident NAFLD were more likely to have had decreases in sleep duration over time or persistently poor sleep quality, even after accounting for changes in BMI.³ This suggests that both short sleep and deterioration in sleep quality can contribute to NAFLD risk, partly via weight gain, insulin resistance, and heightened inflammation (as inadequate sleep activates stress pathways and cytokines that can promote liver fat accumulation). Given that NAFLD is now recognized as a multisystem disease linked with cardiometabolic disorders, these findings underscore the importance of good sleep in maintaining metabolic liver health.

Hypertension and Cardiovascular Disease

Epidemiological studies indicate that sleep duration and quality influence blood pressure and cardiovascular outcomes. Short sleep has been linked with higher risk of developing hypertension: habitually sleeping <6 hours was associated with increased odds of incident hypertension.¹⁸ Sleep loss contributes to sustained elevation of sympathetic tone and poorer nocturnal blood pressure dipping, which over time can lead to hypertension.

Both short and long sleep are associated with elevated risk of coronary heart disease (CHD) and stroke. A meta-analysis of prospective studies found that short sleepers had a 48% greater risk of developing or dying from CHD, and about a 15% higher risk of stroke, compared to those sleeping 7–8 hours.² Long sleep (>9–10 hours) was also linked to increased cardiovascular events, though long sleep may be a marker of underlying ill health (eg, chronic inflammation or undiagnosed disease). For atrial fibrillation (AF), emerging evidence connects sleep problems to arrhythmia risk. Chronic insomnia has been associated with incident AF, likely through prolonged autonomic arousal. Mendelian randomization (MR) analyses even imply a potential causal role of insomnia in AF development. OSA is a well-known precipitant of arrhythmias: untreated OSA markedly increases the likelihood of AF and other arrhythmias via surges in blood pressure and sympathetic activity during apneas.

Chronic insomnia has been linked to higher cardiovascular risk independent of sleep duration. A meta-analysis reported that insomnia disorder is associated with 45% increased risk of developing or dying from CVD.¹⁹ Insomnia's chronic

hyperarousal (elevated cortisol and heart rate) is thought to drive this risk. Additionally, circadian rhythm disruption (such as in shift workers) is associated with cardiometabolic harm. Epidemiological studies of shift work find increased rates of hypertension, heart disease, and diabetes in long-term night shift workers.²⁰ Circadian misalignment – being awake and eating at biologically inappropriate times – can worsen blood pressure control and lipid and glucose metabolism.

Heart Failure and Mortality

Sleep characteristics also relate to heart failure (HF) risk and overall mortality. Persistently short sleep has been linked to higher incidence of congestive heart failure, potentially mediated by blood pressure and metabolic effects. OSA in particular contributes to HF by intermittent hypoxia-induced myocardial stress and by causing hypertension. Patients with untreated moderate–severe OSA have greater risk of developing left ventricular dysfunction and HF over time.²¹ On the other hand, treatment of sleep apnea (eg, CPAP) in patients with existing heart failure can improve ejection fraction and survival, highlighting the interplay.

All-cause mortality is U-shaped in relation to habitual sleep duration in population studies.² Individuals at the extremes of sleep (≤ 5 hours or ≥ 10 hours) have higher mortality rates than those around 7 hours, even after adjusting for comorbidities. A US National Health and Nutrition Examination Survey analysis found that very short sleepers had significantly elevated cardiovascular mortality.²² While causality is hard to prove, these patterns underscore that very poor sleep health often accompanies or exacerbates life-threatening conditions.

Table 1 and Table 2 summarize representative epidemiological studies linking various sleep components to cardio-metabolic outcomes, including positive associations and notable null findings. These data collectively support the

Table 1 Epidemiological Evidence Linking Sleep Duration/Quality to Metabolic Outcomes

Study or Source (Year)	Population & Design	Sleep Measure	Outcome (Assessment)	Key Findings	Limitations
Nurses' Health Study (Patel et al, 2006) ¹⁰	68,000 adult women, 16-year prospective cohort	Self-reported sleep duration	Major weight gain (self-report)	Short sleep (≤ 5 h) associated with higher risk of significant weight gain vs 7 h (OR 1.32). Long sleep (≥ 9 h) also modestly increased risk.	Self-reported sleep and weight; primarily middle-aged women.
JAMA Intern Med RCT (Tasali et al, 2022) ¹¹	80 adults with overweight, habitual <6.5 h sleep, 2-week randomized trial	Objective sleep extension (1.2 h)	Caloric intake, weight change	Intervention group reduced intake by 270 kcal/day and lost 0.5 kg vs controls, demonstrating causal effect of extending sleep on lowering intake.	Short trial duration; longer-term effects unknown.
Meta-Analysis (Pan et al, 2012) ²³	29 cross-sec and 11 cohort studies worldwide	Insomnia and sleep quality indirectly (depression as exposure)	Metabolic syndrome (and vice versa)	Depression and MetS are bidirectionally linked: baseline MetS increases depression risk by 49%, and baseline depression increases MetS risk by 52%. Poor sleep (common in depression) likely mediates part of this relationship.	Depression and MetS assessed variably; not all studies accounted for sleep or medications.
Korean Cohort (Um et al, 2022) ³	86,530 adults, 4-year cohort	Change in sleep duration and quality	New-onset NAFLD (ultrasound, Fibrosis score)	Decrease in sleep duration or persistently poor sleep quality over time predicted higher NAFLD incidence. Association attenuated but still significant after BMI adjustment, suggesting partial mediation by weight gain.	Predominantly young, healthy workers; limited generalizability to older populations.

Abbreviations: MetS, metabolic syndrome; NAFLD, non-alcoholic fatty liver disease; OR, odds ratio.

Table 2 Epidemiological Evidence Linking Sleep Disturbances

Study or Source (Year)	Population & Design	Sleep Factor	Outcome (Assessment)	Key Findings	Limitations
Meta-Analysis (Cappuccio et al, 2011) ²	15 studies (24 cohort samples) included 474,684 male and female participants (follow-up 6.9–25 years), and 16,067 events (4,169 for CHD, 3,478 for stroke, and 8,420 for total CVD)	Short (<6 h) and long (>9 h) sleep	Composite: CHD events and stroke (ICD codes)	Short sleep associated with higher risk of CHD (RR 1.48) and stroke (RR 1.15) vs 7–8 h. Long sleep (>9 h) also linked to CHD/stroke in some cohorts. Suggests a U-shaped relationship for heart disease.	Included older studies with varied covariate adjustment; long sleep effect possibly confounded by illness.
SAVE Randomized Trial (McEvoy et al, 2016) ²⁴	2,717 patients with OSA and CVD, median 3.7 yr follow-up	CPAP therapy vs usual care for OSA	Composite CV events (CV death, MI, stroke, heart failure)	CPAP did not significantly reduce major cardiovascular events compared to no CPAP (primary event rate 17.0% vs 15.4%, HR 1.10, $p=0.34$). CPAP improved sleepiness and mood but not CV outcomes in this secondary prevention trial.	CPAP adherence was moderate (3.3 h/night); patients had minimal daytime sleepiness (less severe OSA consequences).
NHANES Analysis (Gupta et al, 2021) ²²	14,000 US adults, 16-year follow-up	Self-reported sleep duration	Cardiovascular mortality (death certificates)	Both short sleepers (<6 h) and long sleepers (>9 h) had elevated CV mortality (adjusted HRs 1.7 and 1.5, respectively) compared to 7 h sleepers. Highlights U-shaped sleep–mortality link.	Sleep measured at baseline only; cause of long sleep not assessed.

Abbreviations: CHD, coronary heart disease; CVD, cardiovascular disease; CPAP, continuous positive airway pressure; HR, hazard ratio; RR, relative risk; MI, myocardial infarction.

concept that optimizing sleep is not merely for improving alertness or mood, but indeed a vital component of cardiometabolic health.

Sleep, Depression, and Cardiometabolic Health

It is important to note the interplay between sleep, mental health, and metabolic disorders. Depression and sleep disturbances often coexist in a bi-directional relationship. On one hand, chronic insomnia or poor sleep quality is a well-known risk factor for developing depression; individuals who sleep poorly are more likely to report depressive symptoms over time.²⁵ On the other hand, depression itself can lead to changes in sleep patterns (insomnia or, in some cases, hypersomnia) and is linked to unhealthy behaviors that worsen cardiometabolic risk (such as physical inactivity and overeating). Critically, depression and metabolic syndrome tend to occur together. A 2012 meta-analysis by Pan et al²³ demonstrated a bidirectional association: baseline metabolic syndrome was associated with a 49% increased risk of developing depression, and baseline depression was associated with a 52% higher risk of incident metabolic syndrome. The likely mediators of this connection are stress and inflammation – depression is characterized by hyperactivation of the hypothalamic-pituitary-adrenal (HPA) axis and elevated inflammatory cytokines, which can promote insulin resistance and visceral fat accumulation, hallmarks of metabolic syndrome. Additionally, certain antidepressant medications have metabolic side effects (weight gain, dyslipidemia). The overlap of depression with poor sleep may thus create a vicious cycle amplifying cardiometabolic dysfunction. From a clinical perspective, screening for depression in patients

with obesity, diabetes, or heart disease (and vice versa) is warranted, and treatments that improve sleep (such as cognitive-behavioral therapy for insomnia) may benefit mood and metabolic control simultaneously.

Obstructive Sleep Apnea and Depression

Obstructive sleep apnea (OSA), a common sleep disorder in people with metabolic risk factors, is also strongly associated with depression. OSA is characterized by repetitive episodes of upper airway collapse during sleep, leading to nocturnal hypoxia and fragmented sleep. Beyond its well-documented cardiometabolic impacts, OSA can significantly affect mental health. Epidemiologic studies indicate that patients with moderate-to-severe OSA have a higher prevalence of depressive symptoms compared to the general population.²⁶ The mechanisms linking OSA to depression likely involve the chronic sleep disruption and oxygen deprivation in OSA, which can alter neurotransmitter systems and brain regions involved in mood regulation. Daytime fatigue, cognitive impairment, and reduced quality of life from OSA may also precipitate or exacerbate depression. Encouragingly, treatment of OSA with CPAP (continuous positive airway pressure) has been shown to improve mood and depressive symptoms in many patients. A meta-analysis of randomized trials found that CPAP therapy was associated with a significant reduction in depression scale scores, especially in patients who had more severe baseline depressive symptoms.²⁷ In a large observational study, effective OSA treatment corresponded with a decrease in depression prevalence from 21% at baseline to 9% after 3 months of CPAP use.²⁸ Not all studies show a dramatic effect—some short-term trials reported minimal mood change with CPAP in less symptomatic patients²⁹—but overall, addressing OSA can markedly improve daytime well-being and mental health. These findings reinforce that clinicians managing OSA should also monitor patients' mood, and conversely, patients with depression (especially if atypical or treatment-resistant) might benefit from evaluation for sleep apnea.

Mechanisms Linking Sleep to Cardiometabolic Disease

Multiple, overlapping biological pathways help explain how inadequate or disrupted sleep can detrimentally affect cardiometabolic function. Glucose metabolism and insulin sensitivity are particularly sleep-sensitive. Even short-term sleep deprivation causes decreased insulin sensitivity and impaired glucose tolerance, creating a pre-diabetic state. Inadequate sleep – as well as OSA-related sleep fragmentation – promotes insulin resistance independent of other factors. Over time, this insulin-resistant state contributes to hyperglycemia and elevates the risk of type 2 diabetes.

Neuroendocrine changes during sleep loss also drive cardiometabolic dysregulation. Notably, sleep curtailment disrupts appetite hormone balance. When people are sleep-deprived, the body produces more ghrelin (a stomach-derived hormone that stimulates hunger) and less leptin (an adipose-derived hormone that signals satiety). A single night of total sleep deprivation caused a 13% rise in ghrelin and a 7% drop in leptin the next morning.³⁰ These shifts corresponded with increased subjective appetite, especially for calorie-dense carbohydrates. Chronic short sleepers tend to have this hormone profile of “starvation in the midst of plenty”, which can lead to overeating. Furthermore, insufficient sleep may lower levels of peptide YY (a gut hormone promoting satiety) and elevate endocannabinoids that drive hedonic food intake. The net effect is an imbalance in hunger hormones that favors increased caloric intake and weight gain. This is one key mechanism linking short sleep to obesity. Conversely, improving sleep duration has been shown to normalize leptin and ghrelin levels, partially reversing this drive to eat.

Autonomic nervous system and blood pressure regulation are also critically tied to sleep. During normal restorative sleep, sympathetic nervous system activity declines and blood pressure exhibits a nocturnal “dipping” (nighttime BP 10% lower than daytime). Sleep deprivation interferes with these processes. Chronic insufficient sleep triggers elevated sympathetic output and stress hormone release, effectively putting the body in a persistent “fight-or-flight” state. Lack of sleep is associated with higher circulating catecholamines (adrenaline, noradrenaline) and elevated evening cortisol levels. Even in healthy individuals, acute sleep loss can raise resting heart rate and blood pressure by activating the sympathetic nervous system. Over time, this sympathetic overactivity contributes to hypertension. Experimental sleep restriction causes an increase in 24-hour blood pressure and reduces heart rate variability (a sign of sympathetic dominance).³¹ Chronic activation of stress pathways also promotes visceral fat accumulation and worsens insulin resistance, compounding metabolic risk. Additionally, disrupted or fragmented sleep (as in OSA or insomnia) impairs

normal nocturnal BP dipping; people who do not experience the normal dip are at higher risk of cardiac events. Thus, the loss of autonomic recuperation during sleep can lead to sustained hypertension and cardiovascular strain over time.

Another important pathway is inflammation and oxidative stress. Adequate sleep is anti-inflammatory, whereas sleep deficiency provokes a pro-inflammatory state. Research shows that even a modest reduction in sleep can increase inflammatory biomarkers. Adults restricted to 4 hours sleep per night for several nights had significantly higher C-reactive protein (CRP) and interleukin-6 levels than when sleeping 8 hours.³² Both acute total sleep loss and chronic short sleep are associated with elevated high-sensitivity CRP, a stable marker of systemic inflammation. Inflammatory cytokines like IL-6 and TNF- α also tend to rise with sleep disruption, which may contribute to endothelial dysfunction in blood vessels. Furthermore, sleep disorders involving intermittent hypoxia – notably OSA – generate oxidative stress. The cyclical drops and surges in blood oxygen during apneas trigger excess production of reactive oxygen species, promoting vascular inflammation and lipid peroxidation. Over time, this chronic low-grade inflammation from poor sleep can damage blood vessel walls, induce insulin resistance, and destabilize atherosclerotic plaques, thereby increasing cardiovascular risk.

Crucially, these pathways overlap and reinforce each other. Elevated cortisol from sleep loss not only raises blood pressure but also increases glucose levels and promotes abdominal fat deposition (worsening insulin resistance). Heightened hunger and caloric intake from hormonal changes lead to weight gain, which in turn can exacerbate insulin resistance and OSA severity (since obesity narrows the airway). OSA itself triggers many of the same mechanisms—intermittent hypoxia and arousals activate the sympathetic nervous system, raise BP, impair insulin sensitivity, and cause oxidative stress—often to a greater degree due to severe oxygen fluctuations. Thus, a person with chronic sleep deprivation and untreated OSA could experience compounded insults: persistent adrenergic stress, metabolic dysregulation, and inflammation, all feeding into a vicious cycle of cardiometabolic risk.

Incorporating Sleep into Lifestyle Interventions for Prevention

Given the strong connections outlined above, addressing sleep has become a vital component of lifestyle interventions aimed at preventing cardiometabolic disease. Public health guidelines are beginning to reflect this: along with diet, physical activity, and not smoking, healthy sleep is now promoted as fundamental for cardiometabolic health. For most adults, this means striving for a regular sleep duration of approximately 7–9 hours per night, with consistent bedtimes and wake times. Achieving this in practice often requires behavioral and environmental adjustments—collectively known as sleep hygiene. Key sleep hygiene recommendations include maintaining a cool, dark, quiet bedroom; avoiding heavy meals, caffeine, or alcohol close to bedtime; minimizing evening screen exposure; and managing stress. These measures can improve sleep quality and quantity, thereby favorably affecting cardiometabolic risk factors.

Epidemiologically, people who habitually obtain adequate sleep are less likely to develop obesity, diabetes, or hypertension. Individuals reporting 7–8 hours of nightly sleep tend to have lower rates of metabolic syndrome compared to those sleeping much less (or more). Improving or maintaining sufficient sleep could help stave off weight gain and metabolic deterioration. An illustrative example is the trial by Tasali et al¹¹ in which a simple sleep extension in short-sleeping adults led to reduced caloric intake and modest weight loss within a few weeks. This finding supports the integration of sleep counseling into weight management and diabetes prevention programs. Indeed, some multidomain lifestyle interventions have started to incorporate a sleep component (in addition to diet and exercise) to determine if attention to sleep boosts overall efficacy. Early results are promising – a systematic review of pediatric obesity prevention trials found that those which included a sleep education component saw more favorable BMI outcomes in children than those that did not.³³ While adult intervention data are still emerging, it stands to reason that helping patients improve their sleep might enhance other lifestyle changes (eg better energy for exercise, improved appetite regulation as leptin/ghrelin normalize, etc).

From a cardiovascular prevention standpoint, addressing sleep could pay dividends as well. Short sleep has been linked with incident hypertension; thus, ensuring sufficient sleep might help normotensive individuals maintain healthy blood pressure. In those with pre-hypertension, extending sleep has shown to reduce blood pressure slightly.³⁴ Community-based efforts, such as educational campaigns about sleep or workplace policies to limit excessive overtime and night shifts, could contribute to long-term reductions in population blood pressure and CVD events. Another facet is

stress reduction: improving sleep often reduces daytime stress perception and sympathetic drive, which is beneficial for the heart and vascular system.

Incorporating sleep into lifestyle medicine also has a health equity consideration. Socioeconomic factors and modern lifestyles (shift work, multiple jobs, nighttime technology use, etc.) often curtail sleep in certain populations, potentially contributing to health disparities. Populations with lower socioeconomic status disproportionately experience short sleep duration and sleep disorders, which may partly mediate their higher cardiometabolic disease rates. Public health initiatives that facilitate adequate sleep – for example, implementing later school start times for adolescents, educating shift workers on circadian adaptation strategies, or simply broad campaigns raising awareness that “sleep matters” – are increasingly viewed as components of chronic disease prevention. Optimizing sleep should stand alongside nutrition and exercise as pillars of cardiometabolic health. Clinicians and health coaches are encouraged to assess patients’ sleep patterns and offer guidance on improving sleep, as even small increases in sleep duration or quality could translate into meaningful cardiometabolic benefits over time.

Sleep in the Management of Cardiometabolic Diseases

Beyond prevention, targeting sleep is also important in patients who already have cardiometabolic diseases. Integrating sleep management into treatment plans can improve outcomes in obesity, diabetes, and cardiovascular conditions. Below we highlight several areas where optimizing sleep or treating sleep disorders has shown benefits:

Weight Management and Obesity: Adequate sleep can augment weight loss efforts, whereas sleep deprivation may undermine them. Patients enrolled in weight loss programs who sleep better tend to lose more fat mass and adhere more closely to dietary goals. One mechanism is hormonal – with sufficient sleep, appetite hormones (leptin, ghrelin) are more balanced, reducing overeating impulses. In contrast, ongoing sleep deficiency causes persistent hunger and preferential cravings for high-carbohydrate, energy-dense foods. Therefore, clinicians managing overweight patients are advised to inquire about sleep habits. Simple interventions like setting a consistent bedtime or limiting screen time at night might meaningfully improve sleep duration and thus support weight reduction. Furthermore, in obese patients with confirmed OSA, weight loss itself is a first-line therapy that can significantly improve OSA severity. Even a 5–10% reduction in body weight can produce significant reductions in apnea–hypopnea index (AHI), improving sleep quality and oxygenation.³⁵ This creates a positive feedback loop: as OSA improves, the patient’s sleep becomes more restorative, which may further facilitate metabolic improvement and continued weight loss.

Obstructive Sleep Apnea (OSA) and CPAP Therapy: The mainstay treatment for moderate-to-severe OSA is continuous positive airway pressure (CPAP), which mechanically prevents airway collapse during sleep. Treating OSA is critical for patients’ cardiometabolic health as well as quality of life. Effective CPAP therapy can virtually eliminate apneic episodes, thereby restoring normal oxygenation and sleep continuity. This has several beneficial consequences: CPAP reduces excessive daytime sleepiness (improving alertness and ability to exercise), lowers nighttime blood pressure surges, and can improve insulin sensitivity. In patients with coexisting OSA and type 2 diabetes, CPAP use for 3 months modestly but significantly improved glycemic control (reducing HbA1c by 0.3% on average) and lowered insulin resistance.³⁶ CPAP has also been shown to reduce sympathetic overactivity at night, leading to small reductions in resting heart rate and better nocturnal blood pressure control. A meta-analysis reported that CPAP therapy in OSA patients with hypertension lowers 24-hour systolic BP by 2 mmHg on average, with larger drops in those who are younger or have more severe OSA.³⁷ While this blood pressure reduction is modest, it can be clinically meaningful when added to other therapies. Importantly, CPAP significantly improves snoring, mood, and overall daily functioning. Not all trials have shown hard outcome benefits: notably, the SAVE trial did not find a reduction in major cardiovascular events with CPAP in a population of mostly non-sleepy OSA patients with established CVD.²⁴ However, in patients who do have daytime symptoms or less advanced CVD, CPAP likely prevents progression of hypertension and heart rhythm disturbances. Thus, for patients with cardiometabolic disease and OSA, CPAP is recommended to improve symptoms and possibly reduce long-term risk (especially when adherence is good, ie >4 hours/night). Beyond CPAP, other therapies such as positional training (for positional OSA), oral mandibular advancement devices, or upper airway surgery can be considered in select cases; successful alleviation of OSA by any method can contribute to better blood pressure and metabolic control.

Type 2 Diabetes and Glycemic Control: Sleep optimization is emerging as a supportive component of diabetes management. Poor sleep raises blood glucose levels and impairs glycemic control in people with diabetes. Conversely, improving sleep quality or duration can modestly lower fasting glucose and HbA1c. A randomized trial in patients with T2DM found that those who received counseling on sleep hygiene (eg regular sleep schedule, bedroom environment) had slight improvements in morning glucose and reported better overall glycemic control than those who did not.³⁸ Treating coexisting sleep disorders is especially crucial in diabetes: OSA can worsen insulin resistance and make blood sugar harder to control. Several studies have examined CPAP in diabetics with OSA. A meta-analysis concluded that CPAP treatment leads to improved insulin sensitivity and a reduction in HbA1c by 0.2–0.3% in patients with T2DM and OSA.³⁶ Although CPAP is not a substitute for diet, exercise, or medications in diabetes management, it addresses an often-overlooked contributor to hyperglycemia. Clinicians should have a low threshold to screen diabetic patients for symptoms of OSA (snoring, daytime fatigue) and initiate treatment, as this can remove a barrier to achieving glycemic targets.

Hypertension and Cardiovascular Disease: For hypertensive patients—especially those with resistant hypertension or an abnormal nocturnal blood pressure pattern (“non-dippers”)—evaluating and improving sleep is important. Both short sleep and untreated OSA commonly contribute to difficulty in blood pressure control. CPAP therapy in OSA patients with hypertension has been shown to lower blood pressure by a few mmHg on average. While these reductions are modest, certain patients (younger individuals or those with severe OSA) experience larger BP drops with CPAP. Even a 2 mmHg reduction in systolic BP can translate into meaningful reductions in stroke risk at the population level. Moreover, CPAP use is linked to improvements in cardiac afterload and a reduction in nighttime arrhythmic triggers (since OSA-related surges in sympathetic activity can precipitate atrial fibrillation and ventricular arrhythmias). In patients with heart failure with preserved or reduced ejection fraction, treating coexisting sleep apnea (either OSA or central sleep apnea) is often recommended as part of comprehensive care, as it may improve oxygenation, cardiac output, and fatigue levels. Beyond CPAP, other treatments for sleep apnea or insomnia can also benefit CVD patients. Cognitive-behavioral therapy for insomnia (CBT-I) has been associated with better quality of life and possibly fewer angina episodes in those with coronary artery disease. Even in post-myocardial infarction or post-stroke patients, paying attention to sleep during recovery can influence rehabilitation outcomes – poor sleep may impede recovery via increased inflammation and blood pressure variability, whereas good sleep supports healing.

Metabolic Syndrome: Patients with the clustering of risk factors that constitute metabolic syndrome often have unrecognized sleep issues (like OSA or chronic short sleep). Addressing these can improve multiple components of MetS simultaneously. Treating OSA in an obese patient may modestly improve fasting blood glucose and triglyceride levels, while also facilitating weight loss by reducing fatigue. A meta-analysis indicated that CPAP therapy in OSA patients led to small but significant reductions in fasting glucose (0.15 mmol/L) and in total cholesterol and triglycerides, even though LDL and BMI changes were non-significant.³⁹ Moreover, ensuring adequate sleep duration can help with weight management, a cornerstone of MetS treatment. Thus, managing sleep disorders can be considered an adjunctive therapy for metabolic syndrome aimed at preventing progression to diabetes or cardiovascular disease.

The management of cardiometabolic diseases should take a multidisciplinary approach that includes sleep evaluation and intervention. Patients should be routinely asked about sleep patterns and symptoms (snoring, daytime sleepiness, insomnia), and if suggestive of a disorder, referred for appropriate testing (such as home sleep apnea testing or polysomnography for OSA). Treating conditions like OSA or chronic insomnia can improve patients’ daily functioning and, in some cases, their disease biomarkers. Even when no formal sleep disorder is present, providing guidance on optimizing sleep often enhances patients’ ability to adhere to other therapies—improving energy for exercise, dietary compliance, and timing of medications. The interplay is bidirectional: better diabetes control can reduce nocturia or neuropathic pain that might be fragmenting sleep; reciprocally, better sleep can yield lower daytime glucose and blood pressure readings. Thus, sleep and chronic disease management support each other, and improving one can beneficially influence the other.

Methodological Considerations in Sleep–Cardiometabolic Research

When interpreting the evidence linking sleep and cardiometabolic disorders, several methodological limitations should be acknowledged. First, much of the epidemiological data comes from observational studies, which are susceptible to recall bias and measurement error in sleep assessment. Many studies rely on self-reported sleep duration or quality, which may

be imprecise; people tend to misestimate their sleep, and questionnaires like “average nightly sleep” have inherent error. The use of objective measures (actigraphy or polysomnography) in large populations is still limited but growing. Second, residual confounding is a concern. Short sleep or poor sleep might be correlated with other unmeasured factors – high stress jobs, depression, or socioeconomic disadvantage – which themselves elevate cardiometabolic risk. Although studies adjust for many confounders (BMI, smoking, etc.), some factors like chronotype (individual preference for morning/evening activity) or detailed diet quality are not always accounted for. Chronotype variation can influence results; eg, short sleep in a “night owl” might have different health effects than in a “morning lark”, due to differences in circadian alignment that are hard to measure. Third, the cross-sectional associations can suffer from reverse causality – people with undiagnosed illness (eg, early heart failure or diabetes) might sleep longer or report fatigue, creating an appearance that long sleep causes disease when in fact disease caused changes in sleep. Longitudinal studies mitigate this but can still be confounded, subclinical disease leads to sleep changes years before diagnosis. Mendelian randomization (MR) studies (using genetic variants as proxies for sleep traits) have attempted to infer causality; interestingly, some MR analyses suggest that lifelong insomnia may causally increase heart disease risk, whereas genetic propensity for short sleep has shown mixed results regarding BMI and diabetes. These genetic approaches have their own assumptions and sometimes yield discrepancies with epidemiological findings (for example, one MR study found no causal effect of short sleep on obesity despite observational links, possibly due to pleiotropy or measurement differences). Additionally, many large studies to date have been in Western or high-income country populations; there is a paucity of data from low- and middle-income countries (LMICs), where patterns of sleep and comorbid conditions may differ (eg, more manual labor or infectious disease could influence sleep in ways not seen in affluent settings). Finally, the role of age must be considered – what is “optimal” sleep may vary across the lifespan, and the impact of napping or other behaviors likely differs between young and older adults (daytime napping in an elderly person might reflect poor night sleep or illness, whereas in a young adult it might be a cultural norm). Future research should strive to address these limitations by using objective multi-dimensional sleep measures, exploring diverse populations, and employing longitudinal or interventional designs (including more randomized trials of sleep interventions) to strengthen causal inference.

Future Directions and Clinical Implications

Recognizing sleep as a fundamental component of cardiometabolic health carries several important implications. Clinically, healthcare providers should routinely incorporate sleep assessments into patient evaluations, especially for those with obesity, diabetes, or cardiovascular conditions. Simple screening questions about habitual sleep duration, sleep quality, snoring, or use of sleep aids can uncover problems that, if addressed, may substantially aid in disease control. Validated tools like the STOP-Bang questionnaire (for OSA risk) or the Pittsburgh Sleep Quality Index can be easily employed in primary care or specialty clinics. Integrating sleep specialists or sleep health educators into multidisciplinary teams for diabetes or hypertension could facilitate comprehensive risk factor management. A patient with difficult-to-control hypertension might benefit from a sleep study to identify undiagnosed OSA. As evidence grows, we may see sleep guidelines incorporated into standard care pathways—for example, recommending CBT-I for hypertensive patients with insomnia, or mandating OSA screening in all patients with type 2 diabetes.

In terms of public health policy, there is an urgent need for broader initiatives that promote healthy sleep on a population level. This includes educational campaigns analogous to those for diet and exercise, emphasizing that sufficient sleep is not a luxury but a health necessity. Schools can play a role (as seen in some regions adopting later start times for adolescents to align with their circadian rhythms), and workplaces can implement policies to reduce excessive overtime and allow adequate rest (such as limiting consecutive night shifts or offering flexible scheduling to accommodate chronotypes). Urban planning and community interventions can also contribute—efforts to reduce noise and light pollution at night (eg, through noise ordinances or shielded street lighting) can improve sleep quality for residents. Governments and insurers might consider covering behavioral sleep programs, knowing the downstream savings if better sleep leads to fewer chronic disease cases. Another policy aspect is addressing social determinants of sleep: communities with high rates of shift work or multiple jobholding (often lower-income groups) might need targeted interventions (like fatigue management training or improved access to sleep disorder treatment) to mitigate the added cardiometabolic risks they face.

From a research perspective, several avenues remain open. More large-scale trials are warranted to determine the extent to which improving sleep can directly prevent cardiovascular events or diabetes onset. For example, ongoing studies are examining whether adding a structured sleep extension program to standard weight-loss counseling yields greater long-term weight reduction than usual care alone. Thus far, evidence suggests improving sleep tends to move intermediate risk factors in the right direction (lowering blood pressure, improving insulin sensitivity, etc.), but demonstrating reductions in “hard” outcomes (heart attacks, strokes) will solidify sleep’s role in guidelines. The mixed results of trials like SAVE indicate that we may need to tailor interventions—perhaps treating only those OSA patients who are sufficiently symptomatic or at a certain risk threshold to see a cardiovascular benefit, rather than a one-size-fits-all approach. Precision medicine approaches to sleep could emerge, where genetic or biomarker profiles help identify who is most vulnerable to the effects of poor sleep and who would gain the most from intensive intervention.

Another emerging area is chronotherapy – aligning not just sleep but also timing of meals, medications, and physical activity with the body’s circadian rhythms to enhance efficacy and minimize metabolic disturbance. For instance, eating more calories earlier in the day (when we are biologically better at handling glucose) might improve weight loss and glycemic control, independent of calorie amount. Integrating such chronobiological strategies with sleep optimization could yield additional benefits for patients with metabolic syndrome or diabetes. Finally, as technology advances, wearable devices and digital apps provide new opportunities for monitoring and improving sleep at scale. These tools could enable personalized feedback loops (eg, an app that detects if you had a short sleep and coaches you on napping or adjusting bedtime) and facilitate pragmatic trials of sleep interventions in real-world settings.

In conclusion, the accumulated evidence makes it increasingly clear that sleep health is integral to metabolic and cardiovascular health. Just as clinicians routinely advice on diet and exercise, advising on sleep should become standard when managing patients at risk for or living with cardiometabolic diseases. Addressing sleep issues – whether through lifestyle modification, behavioral therapy, or medical treatment of sleep disorders – can improve patients’ quality of life and potentially their disease outcomes. The challenge ahead lies in raising awareness (among both the public and healthcare professionals) and overcoming barriers such as the underdiagnoses of sleep disorders and the perception that feeling tired is simply part of modern life. By embracing sleep as a vital sign and therapeutic target, we can take a more holistic and effective approach to reducing the burden of cardiometabolic disease in the population.

Disclosure

The author reports no conflicts of interest in this work.

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