

Research Article

Sleep disorders as contributors to cardiometabolic disease in adult populations

Kwamena Mensah-Amoah^{1*}, Efua Serwaa Osei-Tutu¹¹ University of Cape Coast (UCC) School of Medical Sciences, Ghana* Correspondence: mensah_7896@yahoo.com<https://doi.org/10.59652/56fsav07>

Abstract: Sleep disorders and disturbed sleep patterns are increasingly recognised as contributors to cardiometabolic disease in adults. The evidence is strongest for short sleep duration, obstructive sleep apnoea, insomnia with objective or perceived sleep loss, and circadian disruption related to shift work or irregular sleep timing. These exposures are associated with hypertension, type 2 diabetes, obesity, coronary heart disease, stroke, heart failure, atrial fibrillation, impaired glucose regulation, and increased cardiovascular risk. This literature review examines the contribution of insomnia, obstructive sleep apnoea, insufficient sleep, long sleep duration, circadian misalignment, and shift work to adult cardiometabolic disease. PubMed-indexed systematic reviews, meta-analyses, cohort studies, clinical practice guidelines, mechanistic studies, and randomized trials were reviewed. The epidemiological evidence shows consistent associations between short sleep duration and incident diabetes, hypertension, cardiovascular disease, coronary heart disease, and obesity. Long sleep duration is also associated with increased risk of several outcomes, although causality is less certain and reverse causation is more likely. Obstructive sleep apnoea is common, frequently underdiagnosed, and strongly enriched among patients with obesity, resistant hypertension, heart failure, atrial fibrillation, and type 2 diabetes. Continuous positive airway pressure improves sleepiness and quality of life and may reduce blood pressure most clearly in patients with uncontrolled or resistant hypertension, but its effect on major cardiovascular events is inconsistent in intention-to-treat trials. Circadian disruption adds a separate risk pathway through misalignment of sleep, feeding, endocrine rhythms, blood pressure regulation, and glucose tolerance. Clinical screening should therefore move beyond asking whether a patient sleeps enough. Adults with cardiometabolic disease should be assessed for sleep duration, insomnia symptoms, snoring, witnessed apnoea, daytime sleepiness, resistant hypertension, shift work, and irregular sleep timing. Sleep health should be treated as part of cardiometabolic risk assessment, not as a secondary lifestyle detail.

Keywords: Sleep disorders, insomnia, obstructive sleep apnoea, cardiometabolic disease, hypertension, type 2 diabetes, obesity, circadian disruption, shift work, clinical screening

Introduction

Sleep is often placed after diet, exercise, smoking, alcohol, and medication adherence in cardiometabolic care. That order is increasingly difficult to defend. Sleep duration, sleep continuity, sleep-disordered breathing, and circadian timing influence glucose metabolism, sympathetic activity, blood pressure, appetite regulation, weight gain, inflammation, endothelial function, and cardiovascular physiology. The adult patient with hypertension, obesity, type 2 diabetes, or atrial fibrillation may therefore need a sleep assessment as much as a lipid profile or renal function test.

The challenge is that sleep is not one exposure. Insomnia, obstructive sleep apnoea, short sleep, long sleep, poor sleep quality, restless sleep, night shift work, irregular sleep timing, social jet lag, and circadian misalignment are related but not interchangeable. A patient may sleep for eight hours but have severe obstructive sleep apnoea. Another may have chronic insomnia and sleep five hours. A third may work rotating nights and sleep at

Received: 28 January 2026

Accepted: 14 May 2026

Published: 28 May 2026

**Copyright:** © 2022 by the authors.

Submitted for open access publication under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

biologically adverse times despite adequate total sleep. Grouping all of these under “poor sleep” hides important differences in mechanism, diagnosis, and treatment.

A second problem is interpretation. Much of the cardiometabolic evidence comes from observational studies. These studies are large and useful, but they are exposed to residual confounding, reverse causation, self-reported sleep measurement, depression, chronic pain, medication effects, socioeconomic factors, occupational stress, and obesity. Mechanistic and intervention studies help, but they do not always reproduce the size of observational associations. This review therefore separates association from intervention evidence wherever possible.

The central argument is practical. Sleep disorders do not replace established cardiometabolic risk factors. They add to them. Their value in clinical medicine lies in identifying modifiable conditions that are common, under-recognised, and often present in the same patients already at high metabolic and cardiovascular risk.

Evidence handling and terminology

This review used PubMed-indexed literature on adult sleep duration, insomnia, obstructive sleep apnoea, sleep-disordered breathing, hypertension, type 2 diabetes, obesity, cardiovascular disease, circadian disruption, shift work, positive airway pressure, cognitive behavioural therapy for insomnia, and screening pathways. The source base included clinical practice guidelines, systematic reviews, meta-analyses, prospective cohort studies, randomized controlled trials, and human laboratory studies.

Sleep duration is described as short, normal, or long according to the definitions used by the original studies. Many epidemiological studies classify short sleep as less than six or seven hours and long sleep as more than eight or nine hours. The American Academy of Sleep Medicine and Sleep Research Society consensus recommends that adults sleep seven or more hours per night on a regular basis to promote optimal health (1). Insomnia is discussed as chronic insomnia disorder when symptoms persist for at least three months and produce daytime impairment, consistent with the modern sleep-disorder classification framework (2). Obstructive sleep apnoea is discussed in relation to apnoea-hypopnoea index, symptoms, cardiometabolic risk, and diagnostic testing guidance.

The review is not a formal meta-analysis. It gives priority to large meta-analyses and prospective evidence when estimating risk, and to randomized trials when considering treatment effects.

The statistical signal from sleep duration

Short sleep duration has one of the largest evidence bases. In a systematic review and meta-analysis including 153 studies and more than 5.1 million participants, short sleep duration was associated with increased risk of all-cause mortality, diabetes mellitus, hypertension, cardiovascular disease, coronary heart disease, and obesity. The reported risk ratios were 1.12 for mortality, 1.37 for diabetes, 1.17 for hypertension, 1.16 for cardiovascular disease, 1.26 for coronary heart disease, and 1.38 for obesity (4). These are not trivial associations. They are also not proof that extending sleep alone will reduce all these outcomes by the same magnitude.

Long sleep duration shows a different pattern. In a systematic review and meta-analysis of 137 prospective cohort studies including more than 5.1 million participants, long sleep was associated with mortality, diabetes, cardiovascular disease, stroke, coronary heart disease, and obesity. The reported risk ratios were 1.39 for mortality, 1.26 for diabetes, 1.25 for cardiovascular disease, 1.46 for stroke, 1.24 for coronary heart disease, and 1.08 for obesity; long sleep was not significantly associated with incident hypertension in that analysis (5). The interpretation of long sleep is more difficult than short sleep. Long sleep may reflect underlying disease, inflammation, depression, unemployment, sedative use, low physical activity, sleep fragmentation, or undiagnosed sleep apnoea. It may be a marker more often than a cause.

The diabetes evidence is particularly consistent. A dose-response meta-analysis of prospective studies including 18,443 incident type 2 diabetes cases among 482,502 participants found a U-shaped association between sleep duration and type 2 diabetes risk. The lowest risk was observed at seven to eight hours per day. Compared with seven hours, each one-hour reduction below seven hours was associated with a pooled relative risk of 1.09, while each one-hour increment above seven hours was associated with a pooled relative risk of 1.14 (6). Another meta-analysis of sleep disturbances and incident diabetes also supported associations between both sleep quantity and sleep quality and diabetes risk (7).

Hypertension follows a similar but not identical pattern. A meta-analysis of prospective cohort studies found that short sleep duration increased hypertension incidence, with a relative risk of 1.21. Sleep continuity disturbance and early-morning awakening were also associated with hypertension incidence, while evidence for long sleep and difficulty falling asleep was weaker (8). This matters because insomnia symptoms are not all equal. Difficulty maintaining sleep may have a stronger cardiometabolic signal than difficulty falling asleep alone in some datasets.

Obesity is closely linked to short sleep. A meta-analysis of prospective adult studies found that short sleep duration was significantly associated with incident obesity, whereas long sleep duration was not consistently associated with obesity (9). Another dose-response meta-analysis reported that obesity risk increased by approximately 9% for each one-hour decrease in sleep duration compared with seven hours (10). Sleep loss may influence food intake, reward sensitivity, late-night eating, energy expenditure, physical activity, and hormonal appetite regulation. Still, obesity also causes sleep disruption and obstructive sleep apnoea, so directionality can run both ways.

Table 1

Selected risk estimates linking sleep patterns with cardiometabolic outcomes

Sleep exposure	Outcome	Statistical finding	Interpretation
Short sleep duration	Diabetes mellitus	RR 1.37 in a large meta-analysis	Strong and consistent observational association
Short sleep duration	Hypertension	RR 1.17 in broad outcomes meta-analysis and RR 1.21 in hypertension-specific meta-analysis	Supports sleep assessment in hypertension prevention and management
Short sleep duration	Obesity	RR 1.38 in broad outcomes meta-analysis	Direction is likely bidirectional because obesity can also worsen sleep
Short sleep duration	Coronary heart disease	RR 1.26 in broad outcomes meta-analysis	Observational signal is clinically relevant but not fully causal
Long sleep duration	Stroke	RR 1.46 in long-sleep meta-analysis	May reflect underlying illness, reverse causation, or sleep fragmentation
Long sleep duration	Hypertension	No significant association in one large meta-analysis	Long sleep is not equivalent to short sleep biologically or clinically
Sleep continuity disturbance	Hypertension incidence	RR 1.20 in prospective meta-analysis	Insomnia phenotype matters
Each hour below seven hours	Type 2 diabetes	RR 1.09 per one-hour shorter sleep	Dose-response pattern supports biological plausibility

Sleep exposure	Outcome	Statistical finding	Interpretation
Each hour above seven hours	Type 2 diabetes	RR 1.14 per one-hour longer sleep	Stronger concern for confounding and reverse causation

RR indicates relative risk.

Plausible mechanisms

Several mechanisms connect disordered sleep to cardiometabolic disease. The evidence is strongest when epidemiological findings are supported by human experimental studies.

Sleep restriction can impair insulin sensitivity. A randomized crossover study found that experimental sleep restriction reduced insulin signalling in human adipocytes, showing that sleep loss can affect peripheral metabolic tissue directly (12). A more recent randomized trial in women found that curtailing sleep to a level resembling real-world short sleep impaired insulin sensitivity over six weeks (11). Even a single night of partial sleep deprivation has been shown to induce insulin resistance in multiple metabolic pathways (13).

Blood pressure is also sensitive to sleep loss. Sleep normally permits nocturnal blood pressure dipping and lower sympathetic tone. Experimental restriction can increase ambulatory and nocturnal blood pressure. In healthy young adults, experimental sleep restriction increased ambulatory and sleep blood pressure in a randomized crossover design (14). In premenopausal women, mild sleep restriction increased 24-hour ambulatory blood pressure without clear mediation by psychological stress measures (15). These controlled studies make the hypertension association more credible.

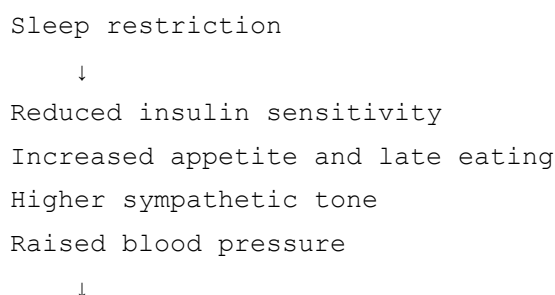
Circadian misalignment adds another pathway. In a controlled study, circadian misalignment increased markers of insulin resistance and inflammation independently of sleep loss (16). In a laboratory study of chronic shift workers, the internal circadian system and circadian misalignment affected glucose tolerance (17). These findings help explain why a person who sleeps during the day after night work may not receive the same metabolic protection as a person sleeping at night, even if sleep duration is similar.

Sleep-disordered breathing contributes through intermittent hypoxaemia, arousals, sympathetic activation, oxidative stress, intrathoracic pressure swings, endothelial dysfunction, inflammation, and changes in metabolic regulation. These mechanisms are especially relevant to resistant hypertension, atrial fibrillation, heart failure, and obesity.

Insomnia may operate through hyperarousal. Chronic insomnia is not simply lack of sleep. Many patients have heightened cognitive, autonomic, and physiological arousal. The result may include increased sympathetic activity, hypothalamic-pituitary-adrenal activation, inflammatory changes, reduced sleep depth, and behavioural changes such as reduced activity and irregular eating. Insomnia with short sleep duration may be a higher-risk phenotype than insomnia symptoms alone.

Figure 1

Major biological routes from sleep disturbance to cardiometabolic disease



Obesity diabetes hypertension cardiovascular risk

Obstructive sleep apnoea

↓

Intermittent hypoxaemia

Arousals and sleep fragmentation

Negative intrathoracic pressure swings

Sympathetic activation

↓

Resistant hypertension atrial fibrillation heart failure stroke risk

Insomnia

↓

Hyperarousal

Sleep fragmentation

Short sleep in high-risk phenotype

Stress-system activation

↓

Hypertension diabetes risk impaired quality of life

Circadian disruption

↓

Mismatch between sleep feeding work and internal clocks

Altered cortisol melatonin glucose tolerance and blood pressure rhythm

↓

Diabetes obesity hypertension cardiovascular disease risk

Insomnia and cardiometabolic disease

Insomnia is the most common sleep disorder in routine adult practice. A recent epidemiological review estimated that approximately 10% of adults have insomnia disorder, while another 20% experience occasional insomnia symptoms (3). The diagnosis requires more than dissatisfaction with sleep. Chronic insomnia disorder involves difficulty initiating sleep, difficulty maintaining sleep, early awakening, or non-restorative sleep despite adequate opportunity for sleep, with daytime impairment and persistence over time (2).

The cardiometabolic signal for insomnia is real but less uniform than for obstructive sleep apnoea or short sleep duration. Insomnia symptoms are heterogeneous. Difficulty falling asleep, difficulty maintaining sleep, early-morning awakening, non-restorative sleep, and short measured sleep may carry different risks. A meta-review combining observational meta-analyses and Mendelian randomization studies reported evidence linking insomnia with several cardiovascular diseases, including hypertension, coronary artery disease, atrial fibrillation, heart failure, and stroke outcomes (18). A meta-analysis of prospective studies found that sleep continuity disturbance and early-morning awakening increased hypertension incidence (8). A 2023 meta-analysis reported an association between insomnia and myocardial infarction incidence, especially with very short sleep duration (19).

Diabetes risk is also relevant. The older meta-analysis by Cappuccio et al. found that difficulty initiating sleep and difficulty maintaining sleep were associated with incident type 2 diabetes (7). Chronic insomnia may affect glycaemic regulation through sympathetic activation, altered cortisol rhythm, inflammation, poorer diet, late eating, inactivity, depression, and use of sedative medications in some patients.

Treatment evidence is more complex. Cognitive behavioural therapy for insomnia is the first-line treatment recommended by major guidelines (31,32). Its primary proven benefit is improvement in insomnia symptoms, sleep latency, sleep efficiency, wake after sleep onset, and daytime function. Whether it reduces cardiometabolic outcomes such as myocardial infarction, diabetes, or stroke remains less certain. A systematic review of cognitive behavioural therapy for insomnia and cardiometabolic biomarkers found the most consistent associations with improved HbA1c and C-reactive protein, but the evidence base was limited and not equivalent to hard-outcome prevention trials (33).

Clinically, insomnia should be identified because it is distressing, common, treatable, and associated with cardiometabolic risk. It should not be treated only with hypnotic medication, especially when chronic. Long-term sedative use can worsen falls, cognition, respiratory risk, and next-day function in vulnerable adults. A cardiometabolic clinic that detects insomnia should consider CBT-I referral, digital CBT-I, behavioural sleep intervention, and review of stimulants, alcohol, pain, mood symptoms, and medication timing.

Obstructive sleep apnoea as a cardiometabolic condition

Obstructive sleep apnoea is not simply snoring with tiredness. It is a disorder of repeated upper-airway obstruction during sleep, producing intermittent hypoxaemia, recurrent arousals, sleep fragmentation, and cardiovascular stress. Its cardiometabolic relevance is large because the same adult population at risk for hypertension, obesity, diabetes, and atrial fibrillation is also at high risk for OSA.

The burden is substantial. Benjafield et al. estimated that 936 million adults aged 30-69 years worldwide had mild to severe obstructive sleep apnoea, and 425 million had moderate to severe disease (20). A systematic review of general population studies found that at an apnoea-hypopnoea index of 15 or more events per hour, adult prevalence ranged from 6% to 17%, and reached as high as 49% in advanced age groups (21). These figures explain why OSA is encountered far beyond sleep clinics.

OSA prevalence is especially high in cardiovascular practice. The American Heart Association scientific statement noted that OSA prevalence is estimated at 40% to 80% among patients with hypertension, heart failure, coronary artery disease, pulmonary hypertension, atrial fibrillation, and stroke (24). Yet OSA remains under-recognised in many of these groups, partly because not all patients present with classic sleepiness. Some present with resistant hypertension, nocturia, morning headache, atrial fibrillation, poor concentration, or unrefreshing sleep.

The association with cardiovascular outcomes is supported by cohort data. A meta-analysis of prospective cohort studies reported that severe OSA significantly increased cardiovascular disease risk, stroke, and all-cause mortality; moderate OSA showed a positive association with cardiovascular disease, while mild OSA did not show the same strength of association (25). In an observational study of men, untreated severe obstructive sleep apnoea was associated with higher risk of fatal and non-fatal cardiovascular events, while CPAP-treated patients had lower event rates than untreated severe OSA patients (26). Observational results, however, can be influenced by adherence, health behaviour, and selection effects.

Randomized cardiovascular outcome trials are more cautious. The SAVE trial found that CPAP improved snoring, sleepiness, mood, and health-related quality of life, but did not significantly prevent major cardiovascular events in patients with moderate to severe OSA and established cardiovascular disease in the intention-to-treat analysis (27). This result is often misunderstood. It does not mean OSA is harmless or CPAP is useless. It means CPAP should not be promised as guaranteed secondary cardiovascular prevention in minimally sleepy or

variably adherent populations. Adherence, symptom burden, baseline risk, OSA phenotype, hypoxic burden, and blood pressure status likely matter.

OSA and hypertension

Hypertension is the most clinically actionable cardiometabolic outcome in OSA. OSA is common in resistant hypertension, and resistant hypertension is one of the strongest reasons to screen for OSA. Intermittent hypoxaemia and arousals increase sympathetic activation, reduce nocturnal dipping, and may sustain daytime blood pressure elevation.

CPAP lowers blood pressure modestly in many OSA populations, but the effect is not uniform. Meta-analyses show larger effects in patients with resistant hypertension and uncontrolled blood pressure than in minimally symptomatic or normotensive patients. A systematic review and meta-analysis of randomized trials in OSA with resistant hypertension reported that CPAP improved blood pressure in this group (28). A broader meta-analysis comparing OSA treatments found that expected reductions in blood pressure were modest overall and varied by phenotype (29). This is clinically useful: CPAP should be seen as one part of resistant hypertension care, not a replacement for antihypertensive treatment, weight loss, sodium reduction, and evaluation for other secondary causes.

Ambulatory blood pressure is more informative than clinic pressure in this setting. OSA may blunt nocturnal dipping or produce nocturnal hypertension. A patient with controlled clinic readings and high nocturnal blood pressure may still carry cardiovascular risk. Where available, ambulatory blood pressure monitoring can clarify the pattern.

OSA, diabetes, and obesity

The relationship between OSA and diabetes is bidirectional. Obesity increases OSA risk by narrowing the upper airway and increasing collapsibility. OSA may worsen insulin resistance through intermittent hypoxia, sleep fragmentation, sympathetic activation, and inflammation. Diabetes can also worsen sleep through nocturia, neuropathic pain, restless legs, and coexisting obesity.

CPAP effects on glycaemic control are inconsistent. In a systematic review and meta-analysis of patients with type 2 diabetes and OSA, CPAP did not clearly improve HbA1c (30). A randomized trial in patients with suboptimally controlled type 2 diabetes and OSA found improved glycaemic control after six months of CPAP (31). Differences in adherence, baseline HbA1c, OSA severity, sleepiness, duration of therapy, diabetes treatment, and weight change may explain some of the inconsistency.

Weight management is central. CPAP does not reliably produce weight loss by itself. It may improve sleepiness and energy, which can support physical activity, but weight reduction requires separate intervention. Anti-obesity pharmacotherapy, bariatric surgery, dietary strategies, and exercise can reduce OSA severity in selected patients through weight loss and metabolic improvement. The direction should be integrated: treat OSA, treat obesity, treat diabetes, and treat blood pressure together.

Table 2

Sleep disorder phenotypes and likely cardiometabolic consequences

Sleep phenotype	Typical clinical clues	Cardiometabolic links	Highest-yield screening setting
Short sleep duration	Less than six or seven hours, long work hours, caregiving, late device use	Obesity, diabetes, hypertension, coronary disease	Primary care, diabetes care, occupational health

Sleep phenotype	Typical clinical clues	Cardiometabolic links	Highest-yield screening setting
Chronic insomnia	Difficulty initiating or maintaining sleep with daytime impairment	Hypertension, diabetes risk, myocardial infarction signal, poor quality of life	Primary care, women's health, mental health, cardiology
Obstructive sleep apnoea	Snoring, witnessed apnoea, resistant hypertension, sleepiness, obesity, nocturia	Hypertension, atrial fibrillation, stroke, heart failure, diabetes	Hypertension clinic, cardiology, obesity medicine, diabetes clinic
Long sleep duration	More than nine hours, low activity, depression, chronic illness, sedating drugs	Mortality, diabetes, cardiovascular disease, stroke	Geriatrics, chronic disease clinics, mental health
Shift work and circadian disruption	Night shifts, rotating shifts, irregular meals, variable sleep timing	Diabetes, obesity, blood pressure elevation, cardiovascular risk	Occupational health, emergency services, healthcare workers
Social jet lag	Large weekday-weekend sleep timing difference	Adiposity, insulin resistance, metabolic syndrome markers	Young and middle-aged adults, students, shift workers
Sleep fragmentation	Frequent awakenings, pain, nocturia, OSA, restless legs	Hypertension, glucose dysregulation, fatigue, poor adherence	Chronic pain, urology, sleep clinics, cardiometabolic clinics

Circadian disruption and shift work

Circadian disruption is not the same as short sleep. It refers to misalignment between internal biological timing and external behaviour. A night-shift worker may sleep long enough after a shift and still experience metabolic strain because sleep, feeding, light exposure, physical activity, cortisol rhythm, melatonin rhythm, glucose tolerance, and blood pressure rhythm are not aligned.

Shift work has been associated with diabetes risk in meta-analysis. Gan et al. reported that shift work was associated with increased risk of diabetes, with stronger effects in men and rotating shift workers (34). In the Nurses' Health Studies, longer duration of rotating night shift work was associated with increased risk of type 2 diabetes in women, partly mediated through body weight (35). Shift work has also been associated with obesity and overweight in meta-analyses (36). A systematic review and meta-analysis of shift work and blood pressure found that shift work was associated with blood pressure differences, particularly among permanent night workers and for systolic blood pressure (37).

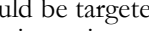
Social jet lag gives a related signal in non-shift workers. Wong et al. reported that misalignment of sleep timing was associated with metabolic risk factors that predispose to diabetes and atherosclerotic cardiovascular disease (38). Social jet lag is common when workdays force earlier wake times than the person's biological preference, followed by delayed sleep and waking on free days. The repeated shift in sleep timing resembles mild circadian travel without movement across time zones.

The clinical response to circadian disruption is difficult because many patients cannot simply stop shift work. Advice must therefore be realistic. Risk reduction may include more stable shift schedules, avoidance of rapidly rotating shifts where possible, planned light exposure, protected sleep opportunity, sleep environment control, consistent meal timing, reduced large meals at biological night, screening for hypertension and diabetes, and early recognition of OSA or insomnia. Occupational health has a major role.

Chart 1

Relative strength of evidence by sleep exposure and cardiometabolic outcome

Evidence signal based on current adult literature

Short sleep and type 2 diabetes support		Strong observational and mechanistic support
Short sleep and hypertension experimental support		Strong observational support with experimental support
Short sleep and obesity pathway likely		Strong association, bidirectional pathway likely
Long sleep and cardiovascular outcomes certain		Consistent association, causality less certain
Insomnia and hypertension phenotype		Moderate to strong, depends on insomnia phenotype
Insomnia and myocardial infarction		Growing evidence, heterogeneity remains
OSA and resistant hypertension treatment signal		Strong clinical association and treatment signal
OSA and major cardiovascular events prevention signal		Cohort signal stronger than RCT
OSA and glycaemic control		Mixed treatment evidence
Shift work and type 2 diabetes		Consistent observational evidence
Circadian misalignment and glucose control		Strong mechanistic support

This chart is a qualitative evidence map, not a pooled effect estimate.

Clinical screening

Sleep screening should be targeted. It is unrealistic to send every adult for polysomnography. It is also poor practice to ignore sleep in patients with cardiometabolic disease.

A practical screening history can be brief. Ask usual sleep duration on workdays and free days. Ask about difficulty falling asleep, difficulty staying asleep, early waking, and daytime impairment. Ask about snoring, witnessed apnoea, choking or gasping, nocturia, morning headache, daytime sleepiness, resistant hypertension, and atrial fibrillation. Ask about night shifts, rotating shifts, irregular meals, and large weekday-weekend differences in sleep timing. Ask about alcohol before sleep, sedatives, opioids, nasal obstruction, chronic pain, depression, restless legs, and menopausal symptoms where relevant.

For suspected OSA, the American Academy of Sleep Medicine diagnostic testing guideline recommends that diagnostic testing be performed in conjunction with a comprehensive sleep evaluation and follow-up. Polysomnography is the standard diagnostic test when concern for OSA exists, while home sleep apnoea testing can be used in uncomplicated adults at increased risk of moderate to severe OSA (22). The same guideline advises against diagnosing OSA using clinical tools, questionnaires, or prediction algorithms alone without objective testing (22).

Questionnaires still have value as triage tools. STOP-Bang, Berlin Questionnaire, Epworth Sleepiness Scale, and similar instruments can help identify risk or symptom burden, but they do not replace diagnostic testing. Sleepiness is useful but incomplete. Some high-risk cardiovascular patients with OSA are not very sleepy.

Table 3

Suggested sleep screening prompts in cardiometabolic clinics

Clinical setting	Minimum sleep questions	When to escalate
Hypertension clinic	Sleep duration, snoring, witnessed apnoea, nocturia, morning headache, resistant hypertension	Resistant hypertension, nocturnal hypertension, obesity, atrial fibrillation, high OSA questionnaire score
Diabetes clinic	Sleep duration, insomnia symptoms, OSA symptoms, shift work, nocturia	Poor glycaemic control, obesity, daytime sleepiness, recurrent early-morning hyperglycaemia
Obesity clinic	Snoring, apnoea, sleepiness, sleep timing, binge or night eating, insomnia	BMI elevation with symptoms, bariatric surgery assessment, severe fatigue, cardiopulmonary symptoms
Cardiology clinic	OSA symptoms, atrial fibrillation, heart failure symptoms, nocturnal dyspnoea, sleep duration	Atrial fibrillation recurrence, heart failure, pulmonary hypertension, resistant hypertension
Primary care	Sleep duration, insomnia, sleep quality, shift work, snoring	Persistent fatigue, cardiometabolic disease, sedative use, high occupational risk
Occupational health	Shift timing, sleep opportunity, sleepiness at work or while driving, irregular meals	Safety-sensitive work, night shifts, rotating shifts, hypertension, diabetes risk

Treatment and risk modification

The most important treatment principle is to match the intervention to the sleep phenotype. Sleep advice alone is insufficient for OSA. CPAP alone is insufficient for obesity. Hypnotic medication alone is insufficient for chronic insomnia. Telling a night-shift worker to “sleep better” without addressing schedule, light, and meal timing is clinically weak.

For chronic insomnia, CBT-I is first-line. The American College of Physicians recommends CBT-I as the initial treatment for chronic insomnia disorder in adults (32). The American Academy of Sleep Medicine guideline strongly recommends multicomponent CBT-I for chronic insomnia disorder (31). Pharmacological therapy may have a role when clinically indicated, but long-term reliance on hypnotics does not address the behavioural and cognitive drivers of chronic insomnia.

For OSA, positive airway pressure is the standard treatment for many adults, with oral appliances, positional therapy, weight reduction, upper-airway surgery, and other approaches used in selected cases. The AASM positive airway pressure guideline supports PAP therapy for adults with OSA, particularly when excessive sleepiness or impaired sleep-related quality of life is present (23). Cardiometabolic expectations should be specific. CPAP is likely to improve sleepiness and quality of life in symptomatic patients. Blood pressure reductions are generally modest, larger in resistant hypertension or uncontrolled blood pressure. Prevention of major cardiovascular events is not consistently shown in intention-to-treat RCTs, especially when adherence is low.

For short sleep, sleep extension may be a reasonable intervention in habitual short sleepers, but evidence for hard cardiometabolic outcomes remains developing. Clinicians should still treat insufficient sleep as a risk behaviour, particularly in patients with hypertension, diabetes, obesity, or occupational fatigue. Practical steps include fixed wake time, protected sleep opportunity, reduced late caffeine, reduction of alcohol before bed, light management, digital curfew where relevant, and management of pain, nocturia, reflux, or mood symptoms.

For circadian disruption, the target is timing. Stable schedules, morning or shift-specific light exposure, limiting bright light before planned sleep, dark sleep environments, regular

meal timing, and avoiding large meals during biological night may reduce metabolic strain. In many workplaces, however, individual advice is not enough. Shift design and occupational policy matter.

Discussion

The sleep-cardiometabolic literature contains two messages that must be held together. The first is that sleep disorders are strongly associated with cardiometabolic disease. The second is that association does not always translate into large outcome reductions when a single sleep treatment is tested in randomized trials. This tension is not a weakness of the field. It is the reality of complex chronic disease.

The strongest epidemiological signal is seen with short sleep duration, OSA, and circadian disruption. Short sleep is associated with diabetes, hypertension, obesity, coronary disease, and mortality in large cohorts and meta-analyses (4,6,8-10). Mechanistic studies support biological plausibility by showing effects on insulin sensitivity and blood pressure (11-15). OSA is highly prevalent in cardiometabolic populations and strongly linked to resistant hypertension, atrial fibrillation, heart failure, stroke, and cardiovascular disease (20-25). Circadian disruption has human experimental support for impaired glucose tolerance and insulin resistance, and observational support from shift-work studies (16,17,34-38).

The weaker area is prevention of hard cardiovascular outcomes after sleep treatment. CPAP improves symptoms and blood pressure in selected OSA groups, but major cardiovascular event reduction has not been consistently demonstrated in intention-to-treat randomized trials (27). Several explanations are plausible. Adherence is often limited. Trials may enroll patients with established disease, in whom vascular risk is already advanced. OSA phenotypes differ: hypoxic burden, arousal frequency, sleepiness, insomnia overlap, obesity, and blood pressure status may influence treatment effect. Mean apnoea-hypopnoea index alone may not capture cardiovascular risk.

Insomnia also illustrates heterogeneity. Insomnia symptoms are common, but cardiometabolic risk may be greatest when insomnia is persistent, accompanied by short sleep duration, or associated with physiological hyperarousal. Treating insomnia improves sleep and quality of life; whether it prevents diabetes or cardiovascular events remains a separate question requiring stronger trials.

Clinically, the correct response is not to overpromise. It is to integrate sleep into risk assessment with the same seriousness given to diet, exercise, blood pressure, and lipids. A patient with resistant hypertension should be assessed for OSA. A patient with type 2 diabetes and insomnia should have sleep addressed as part of glycaemic care. A patient with obesity should be screened for OSA before bariatric surgery and during weight management. A night-shift worker with prediabetes deserves practical circadian and occupational advice, not only dietary advice.

Future research should move away from broad sleep categories toward phenotyping. In OSA, hypoxic burden, sleepiness, blood pressure pattern, arousal burden, insomnia overlap, and adherence may predict cardiometabolic response better than apnoea-hypopnoea index alone. In insomnia, objective sleep duration and physiological hyperarousal may identify higher-risk groups. In shift work, schedule type, duration of exposure, meal timing, chronotype, light exposure, and recovery sleep need better measurement. More trials should test combined interventions: CBT-I plus metabolic risk management, CPAP plus weight-loss therapy, circadian scheduling plus diabetes prevention, and workplace-level shift redesign.

Conclusion

Sleep disorders and disturbed sleep timing are clinically important contributors to cardiometabolic disease in adults. The evidence is strongest for short sleep duration, obstructive sleep apnoea, and circadian disruption, with additional evidence linking chronic insomnia to hypertension, diabetes risk, and cardiovascular outcomes. These relationships are biologically plausible and supported by large cohort studies, meta-analyses, and selected human experimental data.

Short sleep duration is associated with increased risk of diabetes, hypertension, obesity, cardiovascular disease, coronary heart disease, and mortality. Long sleep duration is also associated with adverse outcomes, but its interpretation is more complex because it may reflect underlying illness, low activity, depression, medication effects, or undiagnosed sleep disorders. Obstructive sleep apnoea is common in adults with obesity, resistant hypertension, heart failure, atrial fibrillation, stroke, and type 2 diabetes. It should be actively sought in these groups, especially when symptoms or resistant blood pressure are present. Circadian disruption from shift work and irregular sleep timing should be recognised as a metabolic exposure, not only a work-schedule inconvenience.

Clinical screening can be simple and should be targeted. Adults with cardiometabolic disease should be asked about sleep duration, insomnia symptoms, snoring, witnessed apnoea, daytime sleepiness, nocturia, resistant hypertension, shift work, and irregular sleep timing. Suspected obstructive sleep apnoea requires objective diagnostic testing. Chronic insomnia should be treated with cognitive behavioural therapy for insomnia as first-line care where available.

Sleep treatment should be honest about expected benefits. CPAP improves symptoms and quality of life and can lower blood pressure, especially in resistant hypertension, but cardiovascular event reduction is not guaranteed. CBT-I improves insomnia, but hard cardiometabolic prevention data remain limited. Circadian and shift-work interventions are promising but need more rigorous trials.

Sleep health belongs inside adult cardiometabolic care. It is not a minor lifestyle add-on. For many patients, identifying and treating sleep disorders may improve symptoms, blood pressure control, glycaemic regulation, weight management, quality of life, and long-term risk stratification.

References

1. Watson NF, Badr MS, Belenky G, Bliwise DL, Buxton OM, Buysse D, et al. Recommended Amount of Sleep for a Healthy Adult A Joint Consensus Statement of the American Academy of Sleep Medicine and Sleep Research Society. *Sleep*. 2015;38(6):843-844. PMID: 26039963.
2. Sateia MJ. International classification of sleep disorders third edition Highlights and modifications. *Chest*. 2014;146(5):1387-1394. PMID: 25367475.
3. Morin CM, Jarrin DC. Epidemiology of insomnia Prevalence, course, risk factors, and public health burden. *Sleep Med Clin*. 2022;17(2):173-191. PMID: 35659072.
4. Itani O, Jike M, Watanabe N, Kaneita Y. Short sleep duration and health outcomes A systematic review meta-analysis and meta-regression. *Sleep Med*. 2017;32:246-256. PMID: 27743803.
5. Jike M, Itani O, Watanabe N, Buysse DJ, Kaneita Y. Long sleep duration and health outcomes A systematic review meta-analysis and meta-regression. *Sleep Med Rev*. 2018;39:25-36. PMID: 28890167.
6. Shan Z, Ma H, Xie M, Yan P, Guo Y, Bao W, et al. Sleep duration and risk of type 2 diabetes A meta-analysis of prospective studies. *Diabetes Care*. 2015;38(3):529-537. PMID: 25715415.
7. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes A systematic review and meta-analysis. *Diabetes Care*. 2010;33(2):414-420. PMID: 19910503.
8. Meng L, Zheng Y, Hui R. The relationship of sleep duration and insomnia to risk of hypertension incidence A meta-analysis of prospective cohort studies. *Hypertens Res*. 2013;36(11):985-995. PMID: 24005775.
9. Wu Y, Zhai L, Zhang D. Sleep duration and obesity among adults A meta-analysis of prospective studies. *Sleep Med*. 2014;15(12):1456-1462. PMID: 25450058.
10. Zhou Q, Zhang M, Hu D. Dose-response association between sleep duration and obesity risk A systematic review and meta-analysis of prospective cohort studies. *Sleep Breath*. 2019;23(4):1035-1045. PMID: 30941582.
11. Zuraikat FM, Wood RA, Barragán R, St-Onge MP. Chronic insufficient sleep in women impairs insulin sensitivity independent of adiposity changes. *Diabetes Care*. 2024;47(1):117-125. PMID: 37955852.

12. Broussard JL, Ehrmann DA, Van Cauter E, Tasali E, Brady MJ. Impaired insulin signaling in human adipocytes after experimental sleep restriction A randomized crossover study. *Ann Intern Med.* 2012;157(8):549-557. PMID: 23070488.
13. Donga E, van Dijk M, van Dijk JG, Biermasz NR, Lammers GJ, van Kralingen KW, et al. A single night of partial sleep deprivation induces insulin resistance in multiple metabolic pathways in healthy subjects. *J Clin Endocrinol Metab.* 2010;95(6):2963-2968. PMID: 20371664.
14. Covassin N, Bukartyk J, Singh P, Calvin AD, St Louis EK, Somers VK. Effects of experimental sleep restriction on ambulatory and sleep blood pressure in healthy young adults A randomized crossover study. *Hypertension.* 2021;78(3):859-870. PMID: 34247512.
15. St-Onge MP, Campbell A, Aggarwal B, Taylor JL, Spruill TM, RoyChoudhury A, et al. Mild sleep restriction increases 24-hour ambulatory blood pressure in premenopausal women with no indication of mediation by psychological effects. *Am Heart J.* 2020;223:12-22. PMID: 32135337.
16. Leproult R, Holmback U, Van Cauter E. Circadian misalignment augments markers of insulin resistance and inflammation independently of sleep loss. *Diabetes.* 2014;63(6):1860-1869. PMID: 24458353.
17. Morris CJ, Purvis TE, Hu K, Scheer FAJL. Effects of the internal circadian system and circadian misalignment on glucose tolerance in chronic shift workers. *J Clin Endocrinol Metab.* 2016;101(3):1066-1074. PMID: 26771705.
18. Zhang X, Wu S, Chen X, Li Y. Associations between insomnia and cardiovascular diseases A systematic meta-review and Mendelian randomization study. *Neurosci Biobehav Rev.* 2024;163:105765. PMID: 39167428.
19. Dean YE, Shebl MA, Rouzan SS, Rouzan SS, Alrubaian AM, Alkhater AA, et al. Association between insomnia and the incidence of myocardial infarction A systematic review and meta-analysis. *Clin Cardiol.* 2023;46(4):376-385. PMID: 36841256.
20. Benjafield AV, Ayas NT, Eastwood PR, Heinzer R, Ip MSM, Morrell MJ, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea A literature-based analysis. *Lancet Respir Med.* 2019;7(8):687-698. PMID: 31300334.
21. Senaratna CV, Perret JL, Lodge CJ, Lowe AJ, Campbell BE, Matheson MC, et al. Prevalence of obstructive sleep apnea in the general population A systematic review. *Sleep Med Rev.* 2017;34:70-81. PMID: 27568340.
22. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K, et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea An American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med.* 2017;13(3):479-504. PMID: 28162150.
23. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure An American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med.* 2019;15(2):335-343. PMID: 30736887.
24. Yeghiazarians Y, Jneid H, Tietjens JR, Redline S, Brown DL, El-Sherif N, et al. Obstructive sleep apnea and cardiovascular disease A scientific statement from the American Heart Association. *Circulation.* 2021;144(3):e56-e67. PMID: 34148375.
25. Wang X, Ouyang Y, Wang Z, Zhao G, Liu L, Bi Y. Obstructive sleep apnea and risk of cardiovascular disease and all-cause mortality A meta-analysis of prospective cohort studies. *Int J Cardiol.* 2013;169(3):207-214. PMID: 24161531.
26. Marin JM, Carrizo SJ, Vicente E, Agusti AGN. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure An observational study. *Lancet.* 2005;365(9464):1046-1053. PMID: 15781100.
27. McEvoy RD, Antic NA, Heeley E, Luo Y, Ou Q, Zhang X, et al. CPAP for prevention of cardiovascular events in obstructive sleep apnea. *N Engl J Med.* 2016;375(10):919-931. PMID: 27571048.
28. Labarca G, Dreyse J, Drake L, Jorquera J, Barbe F. Efficacy of continuous positive airway pressure in patients with obstructive sleep apnea and resistant hypertension A systematic review and meta-analysis. *Sleep Med Rev.* 2021;58:101446. PMID: 33607443.
29. Pengo MF, Soranna D, Giontella A, Perger E, Mattaliano P, Schwarz EI, et al. Obstructive sleep apnoea treatment and blood pressure Which phenotypes predict a response A systematic review and meta-analysis. *Eur Respir J.* 2020;55(5):1901945. PMID: 32079643.
30. Labarca G, Reyes T, Jorquera J, Dreyse J, Drake L. CPAP in patients with obstructive sleep apnea and type 2 diabetes mellitus Systematic review and meta-analysis. *Clin Respir J.* 2018;12(8):2361-2368. PMID: 30073792.

31. Martínez-Cerón E, Barquiel B, Bezos AM, Casitas R, Galera R, García-Benito C, et al. Effect of continuous positive airway pressure on glycemic control in patients with obstructive sleep apnea and type 2 diabetes A randomized clinical trial. *Am J Respir Crit Care Med*. 2016;194(4):476-485. PMID: 26910598.
32. Edinger JD, Arnedt JT, Bertisch SM, Carney CE, Harrington JJ, Lichstein KL, et al. Behavioral and psychological treatments for chronic insomnia disorder in adults An American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med*. 2021;17(2):255-262. PMID: 33164742.
33. Qaseem A, Kansagara D, Forcica MA, Cooke M, Denberg TD, Clinical Guidelines Committee of the American College of Physicians. Management of chronic insomnia disorder in adults A clinical practice guideline from the American College of Physicians. *Ann Intern Med*. 2016;165(2):125-133. PMID: 27136449.
34. Savin KL, Patel SR, Clark JM, Gary-Webb TL, Seixas AA, Taylor HA, et al. The effect of cognitive behavioral therapy for insomnia on cardiometabolic health biomarkers A systematic review. *Sleep Med Rev*. 2023;68:101746. PMID: 36476211.
35. Gan Y, Yang C, Tong X, Sun H, Cong Y, Yin X, et al. Shift work and diabetes mellitus A meta-analysis of observational studies. *Occup Environ Med*. 2015;72(1):72-78. PMID: 25030030.
36. Pan A, Schernhammer ES, Sun Q, Hu FB. Rotating night shift work and risk of type 2 diabetes Two prospective cohort studies in women. *PLoS Med*. 2011;8(12):e1001141. PMID: 22162955.
37. Sun M, Feng W, Wang F, Li P, Li Z, Li M, et al. Meta-analysis on shift work and risks of specific obesity types. *Obes Rev*. 2018;19(1):28-40. PMID: 28975706.
38. Madeira SG, Fernandes C, Paiva T, Santos Moreira C, Caldeira D. The impact of different types of shift work on blood pressure and hypertension A systematic review and meta-analysis. *Int J Environ Res Public Health*. 2021;18(13):6738. PMID: 34201492.
39. Wong PM, Hasler BP, Kamarck TW, Muldoon MF, Manuck SB. Social jetlag chronotype and cardiometabolic risk. *J Clin Endocrinol Metab*. 2015;100(12):4612-4620. PMID: 26580236.