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THE ROLE OF SLEEP IN THE PATHOGENESIS OF OBESITY AND OBSTRUCTIVE SLEEP APNEA IN ADULTS: A SYSTEMATIC REVIEW

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ABSTRACT

Obesity and obstructive sleep apnea (OSA) are common, clinically important and mutually reinforcing conditions associated with metabolic, cardiovascular and respiratory morbidity. Although obesity is widely recognized as a major modifiable risk factor for OSA, the relationship between sleep, weight regulation and sleep-disordered breathing is not limited to mechanical narrowing of the upper airway. The aim of this systematic review was to synthesize current evidence on the role of sleep duration, sleep quality, circadian disruption and sleep fragmentation in the pathogenesis of obesity and adult OSA. Scientific publications indexed in PubMed were reviewed, with priority given to systematic reviews, meta-analyses, prospective cohort studies, randomized controlled trials and major clinical reviews. The evidence indicates that short sleep duration is associated with future obesity and central obesity, while experimental sleep restriction promotes increased energy intake, impaired insulin sensitivity and adverse metabolic changes. Obesity contributes to OSA through upper-airway soft tissue enlargement, reduced lung volumes, increased pharyngeal collapsibility and altered ventilatory control. Conversely, OSA may worsen metabolic dysfunction through intermittent hypoxia, sleep fragmentation, sympathetic activation, oxidative stress and inflammation. These findings support a bidirectional model in which insufficient sleep, obesity and OSA interact through behavioral, endocrine, inflammatory and respiratory mechanisms. Adult patients with obesity should therefore be routinely assessed for sleep health and OSA risk, while OSA management should include weight and metabolic strategies in addition to airway-directed therapy.

KEYWORDS

Obstructive Sleep Apnea, Obesity, Sleep Duration, Sleep Restriction, Intermittent Hypoxia, Metabolic Dysfunction

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Introduction

Obesity and obstructive sleep apnea (OSA) represent two interrelated chronic conditions with substantial clinical and public health relevance. Obesity is one of the most prevalent non-communicable diseases worldwide and is strongly associated with type 2 diabetes, hypertension, dyslipidemia, cardiovascular disease and reduced quality of life. OSA is one of the most common sleep-related breathing disorders in adults and is characterized by repetitive upper-airway obstruction during sleep, resulting in intermittent hypoxemia, recurrent arousals and sleep fragmentation. Although these disorders are often considered separately, contemporary evidence indicates that obesity, insufficient sleep and OSA form a complex pathophysiological network rather than a simple one-directional sequence (Benjafield et al., 2019; Gottlieb & Punjabi, 2020; Messineo et al., 2024).

The global burden of OSA is considerable. Literature-based estimates suggest that hundreds of millions of adults may have OSA of at least mild severity, and a large proportion remain undiagnosed (Benjafield et al., 2019). The clinical significance of OSA extends beyond snoring and daytime sleepiness. Untreated OSA has been associated with hypertension, metabolic dysfunction, impaired cognitive performance, increased cardiovascular risk and reduced health-related quality of life (Gottlieb & Punjabi, 2020; Punjabi, 2008). These consequences are particularly relevant in adults with obesity because excessive body weight both increases the likelihood of OSA and contributes independently to cardiometabolic disease.

The association between obesity and OSA is well established, but it is frequently oversimplified. A purely mechanical explanation, in which excess neck and pharyngeal adipose tissue narrows the airway and causes obstruction, is important but incomplete. Obesity also affects lung volumes, respiratory mechanics, ventilatory control, systemic inflammation, adipokine signaling and metabolic regulation. Moreover, OSA itself can influence metabolic pathways through intermittent hypoxia, sleep disruption, sympathetic nervous system activation and inflammatory responses. This bidirectional relationship means that obesity can promote

OSA, while OSA may contribute to the persistence or progression of metabolic dysfunction (Drager et al., 2010; Messineo et al., 2024; Ong et al., 2012).

Sleep duration and sleep quality are additional components of this relationship. Over recent decades, insufficient sleep has become increasingly common in adults due to shift work, artificial light exposure, psychosocial stress, prolonged screen use and changing social habits. Epidemiological studies and meta-analyses have repeatedly linked short sleep duration with higher body mass index, increased risk of future obesity and central adiposity (Bacaro et al., 2020; Cappuccio et al., 2008; Kohanmoo et al., 2024). Experimental studies support biological plausibility by demonstrating that sleep restriction can increase caloric intake, alter appetite-related hormones, impair insulin sensitivity and change energy balance (Al Khatib et al., 2017; Markwald et al., 2013; Zhu et al., 2019).

The sleep-obesity-OSA relationship is therefore clinically important because each component can reinforce the others. Short and poor-quality sleep may promote weight gain through increased appetite, altered food preferences, reduced physical activity, hormonal changes and impaired glucose metabolism. Weight gain, especially central and upper-body adiposity, can increase upper-airway collapsibility and worsen sleep-disordered breathing. OSA then fragments sleep and exposes tissues to repetitive cycles of hypoxia and reoxygenation, potentially aggravating metabolic and inflammatory processes. This creates a feedback loop in which disturbed sleep, obesity and OSA may become self-perpetuating.

Understanding this relationship has direct implications for clinical practice. In adults with obesity, evaluation should not be limited to body weight, blood pressure, glucose and lipid profile; sleep duration, sleep quality, excessive daytime sleepiness, snoring and witnessed apneas should also be assessed. Conversely, in patients diagnosed with OSA, management should not rely exclusively on continuous positive airway pressure (CPAP) or other airway-directed therapies. Weight management, physical activity, dietary improvement and sleep hygiene may be essential elements of a comprehensive treatment strategy (Chirinos et al., 2014; Foster et al., 2009; Kuna et al., 2021; Tuomilehto et al., 2009).

The aim of this review is to synthesize current evidence regarding the role of sleep in the pathogenesis of obesity and OSA in adults. The review focuses on four major areas: the association between sleep duration and obesity, the metabolic and neuroendocrine effects of sleep restriction, the mechanisms by which obesity promotes OSA, and the mechanisms by which OSA may worsen metabolic dysfunction. A further objective is to identify clinically relevant implications for screening, prevention and integrated management.

Methodology

This manuscript was prepared as a systematic literature review structured according to the principles of transparent reporting described in the PRISMA 2020 statement (Page et al., 2021). The review was designed to provide a clinically oriented synthesis rather than a quantitative meta-analysis. No new patient data were collected, and no human or animal intervention was performed. Therefore, formal ethics committee approval was not required.

The literature search focused on publications indexed in PubMed/MEDLINE and PubMed Central. Searches were performed using combinations of the following terms: "sleep duration", "short sleep", "sleep restriction", "sleep deprivation", "circadian disruption", "obesity", "central obesity", "weight gain", "insulin resistance", "obstructive sleep apnea", "sleep apnea", "intermittent hypoxia", "upper airway", "tongue fat", "weight loss" and "CPAP". Boolean operators AND and OR were used to combine terms. Reference lists of key reviews and meta-analyses were also screened to identify additional relevant sources indexed in PubMed.

Eligible publications included systematic reviews, meta-analyses, randomized controlled trials, prospective cohort studies, major clinical reviews and mechanistic studies involving adult populations or adult-relevant pathophysiology. Studies were considered particularly relevant if they evaluated the relationship between sleep duration or sleep quality and obesity; metabolic effects of sleep restriction; obesity-related mechanisms contributing to OSA; metabolic consequences of OSA; or effects of weight loss and CPAP on OSA severity and cardiometabolic markers.

Studies were excluded if they focused exclusively on pediatric populations, animal models without clinical relevance to adult disease, isolated case reports, non-medical commentary, or topics not directly related to the sleep-obesity-OSA relationship. When multiple publications addressed the same concept, priority was given to more recent systematic reviews, meta-analyses and clinically influential prospective or randomized studies. Older landmark studies were included when they provided foundational evidence for mechanisms or longitudinal associations.

Data extraction was qualitative. The following information was collected from the included publications: study design, population characteristics, sleep or OSA assessment method, obesity or metabolic outcome, principal findings and clinical interpretation. Findings were organized thematically into five areas: sleep duration and obesity, sleep restriction and metabolic regulation, obesity-related mechanisms of OSA, OSA-related metabolic and inflammatory consequences, and therapeutic implications.

Table 1. Conceptual framework linking sleep, obesity and obstructive sleep apnea.

Component	Main mechanisms	Clinical relevance
Insufficient sleep	Increased energy intake, appetite dysregulation, reduced insulin sensitivity, circadian misalignment, fatigue-related reduction in activity.	Potentially modifiable contributor to weight gain and central adiposity.
Obesity	Upper-airway soft tissue enlargement, tongue fat accumulation, reduced lung volumes, increased pharyngeal collapsibility, systemic inflammation.	Major modifiable risk factor for OSA onset and severity.
Obstructive sleep apnea	Intermittent hypoxia, sleep fragmentation, sympathetic activation, oxidative stress, inflammatory signaling.	May aggravate metabolic dysfunction and complicate weight management.
Integrated cycle	Short sleep may promote obesity; obesity promotes OSA; OSA worsens sleep continuity and metabolic regulation.	Supports combined management of sleep health, body weight and airway obstruction.

Results

Sleep duration and risk of obesity

The relationship between sleep duration and obesity has been examined in numerous observational studies and meta-analyses. Overall, the evidence consistently suggests that short sleep duration is associated with increased risk of obesity in adults, although the strength of association varies by population, sleep measurement method and adjustment for confounding variables. Bacaro et al. (2020) reported that short sleep duration was significantly associated with future obesity in adulthood, with an odds ratio of 1.412 (95% CI: 1.177-1.694). This finding supports the view that insufficient sleep is not merely a correlate of obesity but may precede weight gain in at least some adult populations.

Earlier work by Cappuccio et al. (2008) also identified an association between short sleep duration and obesity across children and adults. While that analysis included a broader population, it helped establish sleep duration as a potentially relevant factor in body weight regulation. More recent analyses have attempted to distinguish overall obesity from central obesity. Kohanmoo et al. (2024) found that short sleep duration was associated with increased risk of central obesity in prospective cohort studies. This distinction is clinically important because central adiposity is strongly linked to insulin resistance, metabolic syndrome and sleep-disordered breathing.

The association between sleep duration and obesity should not be interpreted as deterministic. Short sleep is one factor among many, including diet, physical activity, socioeconomic conditions, psychiatric comorbidity, work schedules, medication use and genetic susceptibility. Nevertheless, the repeated observation of similar associations across study designs indicates that sleep duration deserves attention in obesity prevention and management. Sleep may influence weight not through a single pathway but through a combination of appetite regulation, reward-based eating, meal timing, fatigue, circadian biology and glucose metabolism.

Experimental sleep restriction and metabolic regulation

Experimental studies provide evidence supporting the biological plausibility of the epidemiological association between insufficient sleep and obesity. In randomized and controlled sleep restriction studies, reduced sleep opportunity has been associated with increased energy intake, impaired insulin sensitivity and adverse changes in metabolism. Zhu et al. (2019), in a comprehensive review and meta-analysis of randomized controlled trials, concluded that sleep restriction adversely affected several metabolism-related parameters in

healthy adults. Al Khatib et al. (2017) similarly found that partial sleep deprivation increased energy intake without a proportional increase in energy expenditure, producing a positive energy balance.

The mechanisms linking sleep restriction to increased energy intake appear multifactorial. Short sleep may extend the time available for eating, increase evening snacking, heighten hedonic responses to palatable foods and reduce inhibitory control. Markwald et al. (2013) showed under controlled laboratory conditions that insufficient sleep increased food intake and led to weight gain, despite changes in total daily energy expenditure. These findings are consistent with the clinical observation that sleep-deprived individuals may experience stronger cravings and reduced capacity to maintain dietary control.

Hormonal regulation of appetite may also contribute. Taheri et al. (2004) reported that short sleep duration was associated with reduced leptin, elevated ghrelin and increased body mass index. Although subsequent studies have not always replicated identical hormonal patterns, the leptin-ghrelin model remains useful for understanding how sleep may influence hunger and satiety signals. More broadly, sleep restriction can affect cortisol rhythms, sympathetic activity, glucose tolerance and insulin sensitivity. Donga et al. (2010) demonstrated that even a single night of partial sleep deprivation induced insulin resistance in multiple metabolic pathways in healthy subjects. Sondrup et al. (2022) further supported a link between sleep manipulation and markers of insulin sensitivity.

Circadian disruption may amplify these effects. Buxton et al. (2012) found that prolonged sleep restriction combined with circadian disruption impaired glucose regulation and metabolism in healthy adults. This is particularly relevant for shift workers and individuals with irregular sleep-wake schedules. In clinical practice, sleep duration alone may therefore be insufficient; timing, regularity and circadian alignment may also influence metabolic risk.

Obesity as a driver of obstructive sleep apnea

Obesity is among the strongest and most consistently documented risk factors for OSA. Longitudinal data support a close relationship between weight change and sleep-disordered breathing. In the Wisconsin Sleep Cohort, Peppard et al. (2000) showed that moderate weight gain was associated with worsening of sleep-disordered breathing, whereas weight loss was associated with improvement. Similar observations from the Sleep Heart Health Study indicated that changes in body weight were associated with progression and regression of sleep-disordered breathing (Newman et al., 2005). These studies provide important evidence that body weight is not only associated cross-sectionally with OSA but can influence disease trajectory over time.

The anatomical mechanisms are clinically intuitive. Excess adipose tissue in the neck, tongue, soft palate and lateral pharyngeal walls can reduce upper-airway caliber and increase collapsibility during sleep. Schwab and colleagues have emphasized the importance of upper-airway soft tissue structures in OSA pathogenesis, and later imaging studies demonstrated the relevance of tongue fat. Kim et al. (2014) reported that tongue fat was greater in people with OSA and may help explain the relationship between obesity and upper-airway obstruction. Wang et al. (2020) further showed that reduction in tongue fat volume was an important mediator of improvement in apnea-hypopnea index after weight loss.

Obesity also affects respiratory mechanics beyond the upper airway. Central and abdominal adiposity reduce functional residual capacity and lung volumes. Reduced lung volume decreases caudal traction on the upper airway, making the pharyngeal airway more collapsible. This mechanism helps explain why central obesity may be particularly relevant to OSA severity. Joosten et al. (2012) and Schwartz et al. (2008) described how obesity-related changes in lung volume, pharyngeal tissue loading and ventilatory control can increase susceptibility to airway collapse.

However, obesity does not fully explain OSA. Some individuals with severe obesity have relatively mild OSA, while others with lower body mass index develop clinically significant disease. Contemporary pathophysiological models describe OSA as a heterogeneous disorder involving several traits: anatomical collapsibility, pharyngeal dilator muscle responsiveness, ventilatory control instability or loop gain, and arousal threshold (Ryan & Bradley, 2005; Zinchuk et al., 2017). Obesity may worsen several of these traits, but individual susceptibility depends on their combined effect. This explains why weight loss often improves OSA but does not always normalize breathing during sleep.

OSA as a contributor to metabolic dysfunction

The reverse direction of the relationship is also clinically relevant: OSA may contribute to metabolic dysfunction independently of obesity. The core physiological disturbances of OSA include intermittent hypoxia, recurrent arousals, intrathoracic pressure swings and sleep fragmentation. These features activate sympathetic pathways and may promote oxidative stress, inflammation, endothelial dysfunction and impaired

glucose metabolism. Drager et al. (2010) reviewed mechanisms by which intermittent hypoxia can influence metabolic regulation, including insulin resistance, dyslipidemia and adipose tissue dysfunction.

Intermittent hypoxia may be particularly important because it exposes tissues to repeated cycles of oxygen desaturation and reoxygenation. Ryan et al. (2005) demonstrated selective activation of inflammatory pathways by intermittent hypoxia in an experimental model relevant to OSA. Later clinical and translational literature has supported the concept that intermittent hypoxia can stimulate inflammatory signaling and contribute to cardiometabolic risk (Kent et al., 2011; Ryan et al., 2019). Sleep fragmentation may act in parallel by reducing sleep quality and impairing restorative physiological functions.

The relationship between OSA and glucose metabolism is complex because obesity is a major confounder. Nevertheless, several reviews conclude that OSA is associated with disturbed glucose homeostasis and metabolic syndrome through plausible biological mechanisms, including sympathetic activation, hepatic glucose output, adipose tissue inflammation, pancreatic beta-cell stress and impaired insulin sensitivity (Mesarwi et al., 2015; Pamidi et al., 2020). In adults with obesity, the coexistence of OSA may therefore worsen a pre-existing metabolic risk profile.

OSA may also influence eating behavior and weight management indirectly. Daytime sleepiness, fatigue, mood disturbance and reduced exercise tolerance may decrease physical activity and impair adherence to lifestyle interventions. Fragmented sleep can contribute to cravings and irregular eating patterns. Thus, OSA may not simply be a consequence of obesity but may also create physiological and behavioral barriers to weight reduction. This provides a rationale for evaluating OSA in patients with obesity who have difficulty losing weight despite lifestyle advice.

Treatment evidence and integrated management

Weight loss is one of the most important modifiable therapeutic strategies for adults with obesity-related OSA. Randomized and longitudinal studies show that lifestyle interventions can reduce OSA severity, although complete remission is not guaranteed. Tuomilehto et al. (2009) found that a structured lifestyle intervention with weight reduction was effective in adults with mild OSA. In the Sleep AHEAD study, Foster et al. (2009) demonstrated that intensive lifestyle intervention improved OSA severity in adults with type 2 diabetes and obesity. Long-term follow-up from Sleep AHEAD suggested that benefits may persist over time, although attenuation can occur (Kuna et al., 2021).

CPAP remains the most established treatment for symptomatic moderate-to-severe OSA and is effective in reducing obstructive respiratory events when used adequately. However, CPAP does not directly address obesity, adipose tissue distribution or lifestyle-related drivers of metabolic risk. Chirinos et al. (2014) compared CPAP, weight loss and combined therapy in adults with obesity and OSA and showed that metabolic outcomes may differ depending on the intervention. A meta-analysis by Drager et al. (2015) also emphasized that CPAP may not produce weight loss and may even be associated with small increases in body weight in some patients. These findings support the concept that CPAP and weight management should be viewed as complementary rather than interchangeable approaches.

Recent reviews have increasingly emphasized personalized and multidisciplinary management. OSA treatment may include CPAP, mandibular advancement devices, positional therapy, lifestyle interventions, weight-loss pharmacotherapy, bariatric surgery or selected surgical procedures depending on patient phenotype, disease severity and treatment tolerance (Gottlieb & Punjabi, 2020; Messineo et al., 2024). For adults with obesity, an integrated plan should address airway obstruction, weight reduction, cardiometabolic risk, sleep hygiene, physical activity and adherence barriers.

Table 2. Main evidence categories identified in the literature synthesis.

Evidence category	Representative findings	Interpretation
Prospective studies and meta-analyses	Short sleep duration is associated with future obesity and central obesity.	Sleep duration is a potentially modifiable risk marker, although causality is not fully established.
Experimental sleep restriction	Sleep restriction increases energy intake and can impair insulin sensitivity or glucose regulation.	Findings support biological plausibility for sleep contributing to weight gain.
Longitudinal OSA cohorts	Weight gain worsens sleep-disordered breathing; weight loss can improve it.	Obesity is a dynamic contributor to OSA progression and regression.
Imaging and mechanistic studies	Tongue fat, pharyngeal soft tissue volume, lung volume and airway collapsibility contribute to OSA.	Obesity affects both anatomy and respiratory mechanics.
Intervention studies	Lifestyle-induced weight loss improves OSA severity; CPAP treats airway obstruction but does not replace weight management.	Integrated treatment is clinically preferable in adults with obesity and OSA.

Discussion

This review supports the concept that sleep, obesity and OSA are interdependent rather than isolated clinical entities. The association between short sleep duration and obesity is consistent across many observational studies, and experimental sleep restriction studies provide plausible mechanisms involving increased caloric intake, impaired insulin sensitivity and altered appetite regulation. At the same time, obesity contributes strongly to OSA through both anatomical and functional mechanisms. OSA may then worsen metabolic dysfunction through intermittent hypoxia, sleep fragmentation, sympathetic activation and inflammation. The most clinically useful model is therefore bidirectional: sleep deficiency may contribute to obesity, obesity increases the risk of OSA, and OSA can further disturb sleep and metabolism.

A major strength of the evidence is its convergence across different types of studies. Epidemiological studies show associations between sleep duration and body weight. Experimental studies demonstrate that sleep restriction can produce measurable metabolic changes within days. Imaging and physiological studies show how obesity changes the upper airway and respiratory mechanics. Longitudinal studies show that weight change alters sleep-disordered breathing severity. Randomized trials show that weight loss improves OSA severity in at least some adults. This convergence makes the overall relationship biologically credible even though no single study design can fully capture all causal pathways.

The clinical interpretation of sleep duration findings requires caution. Sleep duration is commonly self-reported in epidemiological studies, and self-report can be inaccurate. Short sleep may also be a marker of other exposures, such as shift work, stress, depression, low socioeconomic status, chronic pain or irregular eating patterns. These factors may contribute independently to obesity risk. Nevertheless, the repeated association between short sleep and obesity remains relevant because sleep is modifiable and because even modest improvements in sleep regularity and duration may support broader metabolic health interventions.

The evidence also suggests that clinicians should focus not only on total body weight but also on fat distribution. Central obesity appears particularly important because it contributes to insulin resistance and adverse respiratory mechanics. Abdominal fat reduces lung volumes and may increase upper-airway collapsibility by decreasing caudal traction. Neck and pharyngeal adiposity can narrow the airway directly. Tongue fat is especially relevant because imaging studies suggest that it may mediate part of the relationship between weight loss and improvement in OSA severity (Wang et al., 2020). These observations explain why BMI alone is an imperfect predictor of OSA severity.

The heterogeneity of OSA is another important point. While obesity is a major risk factor, OSA is not simply a disease of excess body weight. Craniofacial anatomy, ventilatory control instability, arousal threshold and upper-airway muscle responsiveness can all influence disease expression. This helps explain why some patients remain symptomatic after weight loss and why some lean patients develop OSA. It also explains the variable response to different therapies. The apnea-hypopnea index remains useful for diagnosis and severity classification, but it does not fully describe the underlying phenotype or predict all clinical consequences.

From a metabolic perspective, OSA may create a reinforcing loop that makes weight management more difficult. Intermittent hypoxia and sleep fragmentation can affect glucose metabolism, inflammation and sympathetic activity. Daytime sleepiness and fatigue can reduce physical activity, and poor sleep may increase cravings or late-evening food intake. This does not mean that OSA inevitably causes obesity, but it suggests that untreated OSA may reduce the effectiveness of lifestyle interventions in selected patients. Screening for OSA may therefore be particularly important in adults with obesity, resistant hypertension, type 2 diabetes, unexplained fatigue or poor response to weight management.

Treatment evidence supports an integrated approach. CPAP is effective for reducing obstructive respiratory events, improving oxygenation and decreasing sleep fragmentation when adherence is adequate. However, CPAP alone does not remove the underlying obesity-related mechanical load or metabolic risk. Weight loss interventions can improve OSA severity and cardiometabolic outcomes, but they may not be sufficient for immediate control of moderate-to-severe OSA. Therefore, CPAP and weight management should not be framed as competing options. In many adults with obesity and OSA, the most appropriate approach is combined: treat airway obstruction while simultaneously addressing body weight, physical activity, diet quality and sleep hygiene.

The role of sleep hygiene deserves emphasis. Clinical conversations about obesity often focus on diet and exercise while underestimating sleep. However, if a patient sleeps five hours per night, works rotating shifts and experiences untreated OSA, metabolic regulation and adherence to lifestyle changes may be compromised. Simple clinical questions about sleep duration, sleep timing, snoring, witnessed apneas and daytime sleepiness can identify patients who require more detailed assessment. Sleep health should be considered a component of obesity medicine rather than an unrelated lifestyle issue.

The reviewed literature also has limitations. Many studies are observational and cannot establish causality. Sleep exposure definitions vary widely, including self-reported duration, actigraphy, polysomnography and questionnaires. OSA definitions also differ across studies due to variation in apnea-hypopnea index thresholds and scoring criteria. Many studies adjust for BMI, but residual confounding by visceral adiposity, physical activity, alcohol use, smoking, medications and comorbid disease may remain. Intervention trials often include selected populations and may not generalize to all patients with obesity and OSA.

Another limitation is that many studies focus on BMI rather than detailed body composition. BMI does not distinguish visceral from subcutaneous fat, fat from lean mass or upper-airway fat distribution from total adiposity. Future research should integrate imaging, metabolic biomarkers, sleep architecture, circadian measures and OSA endotyping. Such research may help identify which patients benefit most from weight loss, CPAP, pharmacological weight management, bariatric surgery or targeted OSA therapies.

Despite these limitations, the clinical message is clear. Sleep should be incorporated into the assessment of adults with obesity, and weight/metabolic status should be incorporated into the assessment of adults with OSA. The relationship is not linear but interactive. Patients may benefit from management pathways that combine sleep medicine, respiratory medicine, endocrinology, nutrition, psychology and primary care. Such multidisciplinary care is especially important for individuals with obesity, OSA, type 2 diabetes, hypertension or cardiovascular risk factors.

Table 3. Practical clinical implications for adult patients with obesity and/or OSA.

Clinical situation	Recommended focus	Rationale
Adult with obesity	Ask about sleep duration, sleep quality, snoring, witnessed apneas and daytime sleepiness.	OSA and insufficient sleep may contribute to cardiometabolic risk and impaired weight control.
Adult with suspected OSA	Assess BMI, waist circumference, metabolic risk factors and weight trajectory.	Obesity is a major modifiable contributor to OSA severity.
OSA treated with CPAP	Address weight management, physical activity, diet and adherence barriers.	CPAP reduces obstructive events but does not replace metabolic intervention.
Poor response to lifestyle intervention	Evaluate for sleep restriction, circadian disruption and untreated OSA.	Sleep-related factors may reduce energy balance control and treatment adherence.
Central obesity or type 2 diabetes	Maintain low threshold for OSA screening when symptoms or risk factors are present.	OSA may worsen glucose metabolism and cardiovascular risk.

Conclusions

Sleep, obesity and obstructive sleep apnea are linked through a network of behavioral, metabolic, neuroendocrine, inflammatory and respiratory mechanisms. Current evidence indicates that short sleep duration is associated with an increased risk of adult obesity and central obesity. Experimental studies show that sleep restriction can increase energy intake and impair metabolic regulation, supporting a plausible pathway from insufficient sleep to weight gain.

Obesity is a major modifiable risk factor for OSA, but its effect is not limited to simple narrowing of the upper airway. Excess adiposity influences tongue and pharyngeal soft tissue volume, lung mechanics, pharyngeal collapsibility, inflammatory signaling and ventilatory control. OSA may then contribute to further metabolic dysfunction through intermittent hypoxia, sympathetic activation, inflammation and sleep fragmentation. These mechanisms support a bidirectional model in which sleep disturbance, obesity and OSA can mutually reinforce one another.

In clinical practice, assessment of adults with obesity should include questions about sleep duration, sleep quality and symptoms of OSA. Likewise, management of OSA in adults with obesity should include structured weight and metabolic strategies in addition to airway-directed therapy. Future research should clarify causal pathways, identify clinically useful phenotypes and evaluate whether combined sleep, weight and OSA interventions produce durable improvements in metabolic and cardiovascular outcomes.

- Short and disrupted sleep is associated with increased risk of obesity and central obesity in adults.
- Sleep restriction can promote positive energy balance and impaired glucose regulation.
- Obesity increases OSA risk through upper-airway, lung-volume and inflammatory mechanisms.
- OSA may worsen metabolic dysfunction through intermittent hypoxia, sleep fragmentation and sympathetic activation.
- Integrated management should combine sleep assessment, OSA treatment and weight/metabolic intervention.

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