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REVIEW

The Intertwined Epidemics of Obesity and Obstructive Sleep Apnea: A Comprehensive Analysis

Sarang Patil

Department of Pulmonary, Critical Care and Sleep Medicine, Maharashtra University of Health Sciences, Nashik, Maharashtra, India

Correspondence: Sarang Patil (sarangspatil1@gmail.com)

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ABSTRACT

The escalating global prevalence of both obesity and obstructive sleep apnea (OSA) represents a major public health challenge due to their combined impact on individual health outcomes and healthcare infrastructure. This review provides an analytical evaluation of the complex, bidirectional relationship between these conditions. Evidence demonstrates that OSA prevalence rises markedly across progressive body mass index (BMI) categories, reaching up to 90% in individuals with Class III obesity. This association is driven by mechanical mechanisms—such as upper airway pharyngeal fat accumulation and reduced lung volumes—as well as systemic metabolic inflammation and cellular dysfunction. Conversely, OSA promotes weight gain and metabolic dysregulation through sleep fragmentation, excessive daytime somnolence, and disruption of appetite-regulating hormones (elevated ghrelin and leptin resistance). Interventions yielding weight loss, including structured lifestyle modifications, novel glucagon-like peptide-1 (GLP-1) receptor agonists, and bariatric surgery, demonstrate significant reductions in the apnea–hypopnea index (AHI) and cardiometabolic risk. Given the high co-occurrence and severe cardiovascular risk associated with untreated OSA, routine clinical screening and integrated multidisciplinary management strategies targeting both conditions are essential.

1 | Introduction: The Global Epidemics of Obesity and OSA

The global health landscape is increasingly defined by the dual epidemics of obesity and obstructive sleep apnea (OSA) [1]. Obesity, recognized as a progressive chronic disease characterized by excessive adiposity, has reached pandemic levels globally [2]. Concurrently, the incidence of OSA—a sleep disorder marked by recurrent partial or complete upper airway collapse during sleep—has escalated in parallel [3]. OSA causes intermittent nocturnal hypoxia, hypercapnia, cortical arousals, and sleep fragmentation, which collectively drive daytime somnolence, reduced functional status, and an elevated risk of hypertension, coronary artery disease, and stroke [4].

Despite its high prevalence, a large proportion of individuals living with OSA remain undiagnosed and untreated, representing a missed opportunity to mitigate long-term cardiovascular and metabolic comorbidities [5]. The frequent co-occurrence of obesity and OSA points to a shared pathophysiological framework. Understanding these mechanisms is vital for formulating screening protocols and multi-modal treatment strategies. This manuscript examines the prevalence of OSA across BMI classifications, details the underlying anatomical and cellular pathways, reviews weight loss

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modalities, and outlines the bidirectional forces that complicate long-term management.

2 | Defining Obesity and OSA

2.1 | Obesity: Classification by Body Mass Index (BMI)

Obesity is clinically classified using the BMI, calculated as weight in kilograms divided by height in meters squared (kg/m^2). Standard adult categories define overweight as a BMI of 25.0–29.9 kg/m^2, Class I obesity as 30.0–34.9 kg/m^2, Class II obesity as 35.0–39.9 kg/m^2, and Class III (severe) obesity as ≥ 40.0 kg/m^2 [2].

While BMI is an efficient screening tool in population studies, it does not differentiate between lean muscle mass and adipose tissue, nor does it capture regional fat distribution [6]. Central adiposity, often quantified by waist circumference or neck circumference, offers greater predictive value for upper airway collapse than BMI alone [7]. Men with a waist circumference exceeding 102 cm (40 inches) and women exceeding 88 cm (35 inches) display a higher incidence of cardiometabolic disease and airway compromise due to localized cervical and visceral fat accumulation [7].

2.2 | OSA: Characteristics and Diagnosis

OSA is characterized by repeated transient collapse of the pharyngeal airway during sleep despite persistent respiratory effort [3]. These events are classified as apneas (complete cessation of airflow for ≥ 10 s) or hypopneas (partial airflow reduction accompanied by oxygen desaturation or arousal) [8]. Polysomnography (PSG) serves as the diagnostic gold standard, providing the apnea–hypopnea index (AHI)—the total number of apneas and hypopneas per hour of sleep [8]. Disease severity is stratified as outlined in Table 1.

3 | Prevalence of OSA in Populations With Overweight and Obesity

The prevalence of OSA is substantially higher in individuals with overweight or obesity compared to normal-weight controls [9]. While general population estimates suggest an OSA prevalence of 2%–4%, rates among individuals living with obesity range between 20% and 45%, reaching over 70% in clinical populations undergoing weight management [9, 10].

3.1 | Prevalence Across BMI Categories

Among adults classified as overweight (BMI 25.0–29.9 kg/m^2), mild OSA is present in approximately 26% of individuals [11]. Specific cohort investigations report moderate-to-severe OSA in 21% of overweight men and 9% of overweight women [12].

In Class I obesity (BMI 30.0–34.9 kg/m^2), moderate-to-severe OSA rises to 63% in men and 22% in women [12]. Among patients with type 2 diabetes and Class I obesity, the odds of presenting with OSA are significantly elevated (OR = 5.16; 95% CI, 3.10–8.58) [13].

For Class II obesity (BMI 35.0–39.9 kg/m^2), the odds ratio for OSA increases further (OR = 10.52; 95% CI, 6.20–17.85) among diabetic populations [13].

The highest burden is observed in Class III obesity (BMI ≥ 40.0 kg/m^2), where OSA prevalence reaches 84.6% to 90.0% [14]. In pregnant women with Class III obesity, prospective studies document an OSA prevalence between 40% and 43%, underscoring the compounding physiological strain of gestational and severe adiposity [15]. A summary of prevalence data across BMI categories is presented in Table 2.

3.2 | Demographic, Geographic, and Ethnic Factors

Demographic variables modulate the expression of OSA across weight categories. While OSA is historically more prevalent in men, postmenopausal status in women reduces this gap, aligning female risk closer to male equivalents due to loss of protective progesterone-mediated upper airway muscle tone [16]. Younger adults (under 65 years) diagnosed with OSA demonstrate a higher proportion of severe obesity compared to older adults, in whom structural tissue laxity plays a larger role independent of weight [17].

Geographic and ethnic variations further demonstrate that BMI cut-offs cannot be universally applied without adjustment [18]. For example, South Asian populations experience significant

TABLE 1 | OSA severity classification by apnea–hypopnea index (AHI).

OSA severity	AHI range (events/h)	Clinical description
Normal	< 5	Absence of clinically significant sleep-disordered breathing.
Mild	≥ 5 and < 15	Mildly increased frequency of apneas/hypopneas; minimal daytime fatigue.
Moderate	≥ 15 and ≤ 30	Moderately increased frequency; noticeable daytime somnolence and cardiovascular strain.
Severe	> 30	Markedly elevated respiratory disruptions; prominent hypoxemia, severe fatigue, high comorbidity risk.

Note: Categorization of obstructive sleep apnea (OSA) severity based on the apnea–hypopnea index (AHI, events/h) according to the American Academy of Sleep Medicine (AASM) standard adult criteria, detailing the corresponding clinical descriptions and impact on physiological strain. Based on standard American Academy of Sleep Medicine (AASM) adult diagnostic criteria [8].

TABLE 2 | BMI classifications and corresponding OSA prevalence.

BMI category	BMI range (kg/m^2)	Reported prevalence	Key demographic findings
Overweight	25.0–29.9	25.0%–26.0% overall	Moderate-to-severe OSA present in 21.0% of men and 9.0% of women [11, 12].
Obesity Class I	30.0–34.9	34.1%–63.0%	Moderate-to-severe OSA present in 63.0% of men and 22.0% of women; OR = 5.16 in type 2 diabetes [12, 13].
Obesity Class II	35.0–39.9	45.0%–65.0%	Substantial risk escalation; OR = 10.52 in individuals with type 2 diabetes [13].
Obesity Class III	≥ 40.0	84.6%–90.0%	Severe risk; documented in 40.0%–43.0% of pregnant women with Class III obesity [14, 15].

Note: Summary of body mass index (BMI) categories (overweight, obesity Classes I–III) with their respective reported OSA prevalence rates and key demographic findings from epidemiological meta-analyses and clinical cohort studies. Synthesized from population-based meta-analyses and cohort investigations [11–15].

upper airway obstruction at lower BMI thresholds due to craniofacial skeletal traits and higher visceral fat deposition compared to European cohorts [18]. In India, where urban obesity rates have reached 40.3%, regional studies in cities like Ahmedabad report an OSA prevalence of 69.1% among individuals with obesity [19]. Similarly, population screenings in Tehran, Iran, demonstrate elevated OSA risk in 38.6% of participants, directly correlating with waist-to-hip ratios [20].

4 | Correlation Between BMI and OSA Severity

Longitudinal and cross-sectional data confirm a linear, dose-dependent relationship between BMI increases and AHI severity [21]. A community-based cohort study demonstrated that a one standard deviation increase in BMI is associated with a fourfold increase in OSA risk [21]. Every single-unit increase in BMI yields an estimated 2%–4% rise in severe OSA risk [21].

Weight dynamics directly alter AHI measurements over time (Table 3). A 10% weight gain over a 4-year period predicts a 32% increase in AHI and a sixfold risk of developing moderate-to-severe OSA [22]. Conversely, a 10% reduction in total body weight predicts an approximate 26% decrease in AHI [22]. On a granular level, each 1-point reduction in BMI corresponds to a 6%–7% drop in AHI, while a 7-pound weight loss yields roughly a 7% AHI reduction [23].

However, BMI alone has limitations as an absolute metric. In severe obesity, neck circumference (≥ 43 cm in men, ≥ 40 cm in women) serves as a stronger isolated anatomical predictor of an AHI ≥ 15 events/hour than total body mass [24].

TABLE 3 | Quantifiable relationship between weight dynamics and AHI changes.

Weight change metric	Mean AHI impact	Clinical evidence and mechanism
+10% weight gain	+30% to +32% AHI increase	Longitudinal 4-year data; confers a sixfold increased risk of developing moderate-to-severe OSA [22].
−10% weight loss	−20% to −26% AHI reduction	Reduces cervical fat volume and increases longitudinal tracheal traction [22, 23].
−7 pounds weight loss	−7% AHI reduction	Predicts proportional reduction in mild-to-moderate OSA severity [23].
−1 kg/m^2 BMI reduction	−6% to −7% AHI reduction	Dose-dependent relationship demonstrated in lifestyle intervention cohorts [23].

Note: Overview of the quantitative effect of weight dynamics (weight gain, weight loss, BMI reductions) on apnea–hypopnea index (AHI) measurements and the primary clinical mechanisms driving airway stability changes. Data derived from long-term epidemiological studies and weight loss clinical trials [22, 23].

5 | Physiological and Cellular Mechanisms Linking Obesity to OSA

5.1 | Anatomical Upper Airway Compromise

Adipose deposition beneath the mandible, within the parapharyngeal space, and inside the tongue body directly narrows the lumen of the pharyngeal airway [25]. Volumetric magnetic resonance imaging demonstrates that tongue fat volume is significantly greater in individuals with obesity and OSA compared to weight-matched controls without OSA, directly impairing the mechanical dilator action of the genioglossus muscle during neuromuscular relaxation in sleep [25].

5.2 | Respiratory Mechanics and Lung Volumes

Abdominal and thoracic fat accumulation reduces chest wall compliance and diaphragm mobility [26]. This leads to decreased functional residual capacity (FRC) and end-expiratory lung volume [26]. Lower lung volumes decrease the longitudinal traction force (tracheal tug) exerted on the mediastinum and pharynx during inspiration, reducing upper airway wall stiffness and promoting lumen collapse [26].

5.3 | Cellular Mechanisms and Neuroendocrine Dysregulation

At the cellular level, adipocyte hypertrophy triggers local hypoxia, immune cell infiltration, and secretion of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) [27]. This systemic inflammation induces neuromuscular dysfunction in upper airway dilator muscles [27].

Adipose-derived leptin normally acts on central receptors in the hypothalamus and brainstem to stimulate ventilation and suppress appetite [28]. In individuals with obesity, central leptin resistance impairs respiratory motor output during sleep, exacerbating hypoventilation [28].

5.4 | Emerging Incretin-Based Pharmacotherapies

The introduction of glucagon-like peptide-1 (GLP-1) receptor agonists and dual GLP-1/GIP receptor agonists (e.g., semaglutide, tirzepatide) has introduced a powerful pharmacological tool for managing obesity-induced OSA [29]. Beyond driving substantial weight loss (15% to 22%), these agents reduce visceral and upper airway fat volume, decrease systemic neuroinflammation, and improve metabolic insulin sensitivity [29]. Clinical trials demonstrate significant reductions in AHI alongside improvements in systolic blood pressure and cardiometabolic risk profiles in patients living with OSA [29].

6 | Impact of Weight Loss Interventions and Bariatric Surgery

6.1 | Dietary and Lifestyle Interventions

Structured weight loss programs combining hypocaloric diets, resistance exercise, and behavioral therapy serve as primary therapy for mild-to-moderate OSA [30]. A meta-analysis of randomized controlled trials (RCTs) by Carneiro-Barrera et al. (n = 1,420) confirmed that combined dietary and physical activity interventions significantly reduce AHI, sleepiness scores, and overall oxygen desaturation indices [30].

The long-term results of the sleep AHEAD trial (a 10-year follow-up of 264 adults with overweight/obesity and type 2 diabetes) demonstrated that an intensive lifestyle intervention produced persistent weight loss and sustained AHI reductions compared to standard support [31]. The INTERAPNEA trial further confirmed that an interdisciplinary lifestyle intervention achieved an AHI reduction of 51%–57% at 6-month follow-up, allowing 61.8% of participants to successfully discontinue continuous positive airway pressure (CPAP) therapy [32].

6.2 | Bariatric Surgery Outcomes

For patients with Class III obesity or Class II obesity with severe comorbidities, bariatric surgical procedures (such as Roux-en-Y gastric bypass and sleeve gastrectomy) provide high rate of disease modification [33]. Bariatric surgery yields OSA clinical remission (defined as an AHI < 5 events/hour) in 80%–85% of cases [33]. Postoperative reductions in visceral and pharyngeal adiposity restore lung FRC, improve tracheal tug, and dramatically reduce required therapeutic CPAP pressures [33].

6.3 | Limitations and the SAVE Trial Context

Despite significant weight loss, complete cure of OSA is not achieved in all patients, particularly those with permanent vascular remodeling, long-standing neuromuscular weakness, or underlying craniofacial structural traits [34]. Furthermore, while CPAP therapy resolves sleepiness and restores nighttime oxygenation, the randomized Sleep Apnea Cardiovascular Endpoints (SAVE) trial showed that CPAP monotherapy alone did not significantly lower secondary major adverse cardiovascular events (MACE) in patients with established cardiovascular disease [35]. This finding reinforces the necessity of combining mechanical airway support with comprehensive weight management and cardiovascular risk control [35].

7 | Expanded Bidirectional Relationship: OSA as a Driver of Obesity

The relationship between obesity and OSA is an escalating bidirectional loop, as summarized in Figure 1. While excess adiposity directly promotes pharyngeal collapse, the physiological stresses of OSA simultaneously generate hormonal and metabolic resistance to weight loss [36, 37].

7.1 | Sleep Fragmentation and Neuroendocrine Appetite Dysregulation

Intermittent nocturnal hypoxia and recurrent cortical arousals break sleep architecture, suppressing slow-wave and REM sleep phases [36]. Sleep disruption alters the neuroendocrine appetite axis, elevating plasma levels of the orexigenic peptide ghrelin while inducing peripheral leptin resistance [37]. This neurohormonal state increases cravings for energy-dense, carbohydrate-rich foods, driving excess daily caloric intake [37].

7.2 | Excessive Daytime Somnolence and Energy Balance

Fragmented sleep causes chronic daytime somnolence and fatigue, which systematically decreases voluntary physical activity and nonexercise activity thermogenesis (NEAT) [36].

Furthermore, clinical RCT data assessing weight loss interventions in populations with untreated OSA show that persistent sleep apnea hinders weight loss success [38]. Untreated hypoxemic stress activates the sympathetic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis, elevating nocturnal cortisol levels and blunting lipid oxidation during active caloric restriction [38]. Thus, untreated OSA

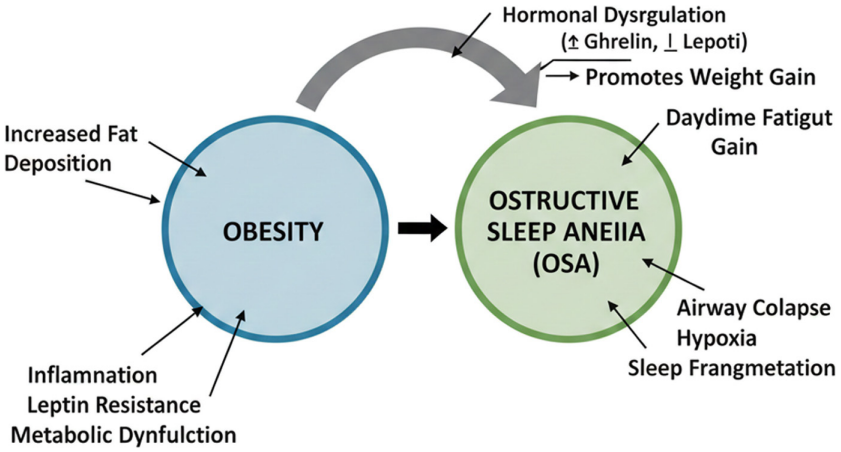

FIGURE 1 | The bidirectional vicious cycle between obesity and obstructive sleep apnea. Conceptual schematic illustrating the bidirectional interplay between obesity and obstructive sleep apnea (OSA). Obesity induces structural upper airway compression, pharyngeal fat accumulation, reduced FRC, and systemic inflammation. Conversely, OSA causes nocturnal intermittent hypoxia and sleep fragmentation, which trigger neuroendocrine dysregulation (elevated ghrelin and central leptin resistance) and excessive daytime somnolence, thereby suppressing energy expenditure and promoting further weight gain.

serves as an active physiological driver of weight gain and weight loss resistance [38].

8 | Conclusion

Obesity and OSA represent mutually reinforcing epidemics. Increasing BMI correlates directly with higher OSA prevalence and AHI severity, driven by mechanical airway narrowing, reduced thoracic compliance, and systemic adipose-mediated inflammation. Conversely, untreated OSA creates a neuroendocrine environment that suppresses physical activity, increases hyperphagia, and impairs metabolic weight loss efforts.

Managing these intertwined conditions requires an integrated model of care combining structured lifestyle modification, incretin-based pharmacotherapies, CPAP therapy, and bariatric surgery when indicated. Routine clinical screening for OSA in all individuals presenting with overweight or obesity is vital to interrupt this damaging cycle and optimize long-term cardiometabolic health.

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Conflicts of Interest

The author declares no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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