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The Bidirectional Relationship Between Obstructive Sleep Apnea and Obesity – A Literature Review

Authors

Marek Dróżdż

F. Ceynowa Specialist Hospital in Wejherowo

A. Jagalskiego 10, 84-200 Wejherowo

https://orcid.org/0009-0007-1737-151X

marek.drozdz2007@gmail.com

Wiktoria Boral

F. Ceynowa Specialist Hospital in Wejherowo

A. Jagalskiego 10, 84-200 Wejherowo

bowik17@gmail.com

https://orcid.org/0009-0007-6047-2033

Alicja Czyszczoń

Podhale Specialist Hospital named after John Paul II in Nowy Targ

Szpitalna 14, 34-400 Nowy Targ, Poland

alicja.czyszczon216@gmail.com

https://orcid.org/0009-0009-1305-6971

Jan Puliński

F. Ceynowa Specialist Hospital in Wejherowo

https://orcid.org/0009-0007-6914-1978

jasiek.1999@wp.pl

Aleksandra Przelaskowska
F. Ceynowa Specialist Hospital in Wejherowo
https://orcid.org/0009-0006-1784-1203
olaprzelaskowska@gmail.com

Aneta Tkaczyk
Podhale Specialist Hospital named after John Paul II in Nowy Targ
Szpitalna 14, 34-400 Nowy Targ, Poland
aneta.tkaczyk1121@gmail.com
https://orcid.org/0009-0005-3085-663X

Wiktoria Marzec
F. Ceynowa Specialist Hospital in Wejherowo
A. Jagalskiego 10, 84-200 Wejherowo
marzecwiktoria1@gmail.com
https://orcid.org/0009-0006-6395-6263

Julia Linke
F. Ceynowa Specialist Hospital in Wejherowo
A. Jagalskiego 10, 84-200 Wejherowo
julialinke719@gmail.com
https://orcid.org/0009-0009-1122-3625

Bartosz Maczkowski
Independent Public Specialist Hospital for Lung Diseases named after Klara Jelska
Gładkie 1, 34-500 Zakopane, Poland
maczko91@gmail.com
https://orcid.org/0009-0008-1693-8446

Corresponding author: Marek Dróżdż marek.drozdz2007@gmail.com

Abstract

Introduction and purpose

Obstructive sleep apnea (OSA) is recognized as one of the most prevalent sleep-related breathing disorders worldwide, affecting up to one billion adults aged 30 to 69 years. This disorder is characterized by recurrent episodes of upper airway collapse during sleep, leading to partial (hypopnea) or complete (apnea) cessation of airflow despite ongoing respiratory effort. These events cause intermittent oxygen desaturation and fragmented sleep architecture, resulting in significant daytime symptoms and adverse health outcomes. The current review aims to elucidate the complex bidirectional relationship between obstructive sleep apnea and obesity, emphasizing the necessity of a comprehensive and integrated clinical approach for the diagnosis, treatment, and management of these intertwined conditions.

A brief description of the state of knowledge

A systematic literature review was performed using PubMed and MDPI databases, encompassing original research articles, systematic reviews, and meta-analyses focusing on obstructive sleep apnea, with particular attention to publications from the last twenty years. The evidence consistently supports a reciprocal association between obesity and obstructive sleep apnea. Additionally, the effect of continuous positive airway pressure (CPAP) therapy on body weight is unclear, with weight gain primarily reported in patients who have poor adherence to the treatment.

Summary

Due to the lack of conclusive data, future investigations should focus on delineating the hormonal and physiological mechanisms that underpin OSA–obesity interaction, optimizing CPAP treatment protocols, and developing targeted weight management strategies tailored for this high-risk patient population. Moreover, clinicians should strongly advocate for the implementation of concurrent behavioral interventions alongside CPAP therapy to mitigate the risk of therapy-associated weight gain and improve overall patient outcomes.

Key words: obstructive sleep apnea; CPAP; obesity.

Introduction

Obstructive sleep apnea (OSA) is one of the most common sleep disorders affecting approximately 1 billion people aged 30-65 years [1]. It is defined by recurrent episodes of complete or partial airway obstruction during sleep causing a decrease (hypopnea) or complete (apnea) stop in airflow despite continued respiratory effort. These events result in oxygen desaturation and repeated arousals from sleep [2].

Diagnosis

The basis for diagnosing OSA is polysomnography (PSG). Diagnosis of the disorder and evaluation of its severity involve quantifying the number of apneic and hypoventilation events per hour of sleep, typically expressed as the apnea-hypopnea index (AHI) or the respiratory disturbance index (RDI). The RDI additionally encompasses respiratory-related arousals that occur during sleep and remain unperceived by the patient. OSA is diagnosed based on the presence of clinical symptoms in conjunction with a RDI of ≥5 events per hour, or in the absence of symptoms, by an RDI of ≥15 events per hour [3]. The symptoms that are required to make a diagnosis are sleep-associated respiratory dysfunction include habitual snoring, audible gasping and observed episodes of breathing cessation, as well as excessive daytime sleepiness or persistent fatigue despite adequate sleep duration, which cannot be attributed to other underlying medical conditions [4].

Epidemiology

OSA is a prevalent disorder with a substantial global burden. Adam V Benjafield et al. in 2019 estimated that 936 million adults aged 30 to 69 years worldwide are affected by mild to severe OSA, while approximately 425 million individuals in the same age group are affected by moderate to severe forms of the condition. The highest prevalence of OSA was observed in China, followed by the United States, Brazil, and India [1]. Research conducted in Poland in 2021 indicates that the prevalence of OSA diagnosis varies by region, ranging from 390 to 1,328 cases per 100,000 inhabitants. The highest prevalence of OSA is observed in the Kujawsko-Pomorskie, Świętokrzyskie, and Mazowieckie regions.The data indicate a continuous annual increase in the number of PSG tests performed across all regions of Poland, which is accompanied by a corresponding rise in newly diagnosed cases of OSA [5].

Description of the state of knowledge

Risk Factors

Risk factors for OSA encompass obesity-recognized as the most significant contributor-along with anatomical abnormalities of the upper airway, male sex, menopausal status, advancing age, alcohol consumption and smoking [3, 6].

As body mass index (BMI) increases, the risk of OSA correspondingly rises, with the prevalence of an AHI ≥ 15 escalating from 3.6% in individuals of normal weight to 56% in men aged 50–70 years with a BMI ≥ 40 [4].

OSA occurs more frequently in men than in women, with an estimated male-to-female ratio of approximately 2:1 in the general population. A similar sex-based disparity is observed in the prevalence of snoring. This male predominance is even more pronounced in clinical populations. Several factors have been proposed to account for this difference, including hormonal influences on upper airway muscle tone and airway collapsibility, sex-specific patterns of adipose tissue distribution, as well as anatomical and functional differences in the pharyngeal region. Hormonal factors may significantly contribute to the pathophysiology of OSA, as evidenced by the increased prevalence of the condition in postmenopausal compared to premenopausal women [7].

The incidence of OSA demonstrates a positive correlation with advancing age. Current estimates indicate that moderate to severe sleep-disordered breathing, defined by an AHI of 15 or higher, affects approximately 10% of men aged 30 to 49 years and 17% of men aged 50 to 70 years. Among women, the prevalence is lower, with around 3% in the 30 to 49 age group and 9% in those aged 50 to 70 [6,8].

Elevated risk of OSA has been associated with higher levels of alcohol intake and a greater frequency of binge drinking episodes, indicating a potential link between alcohol use disorders and the development of OSA. Moreover, current smoking status, particularly among heavy smokers, has been identified as a contributing factor to a heightened risk of sleep-disordered breathing [9,10].

Causes - PALM model

The pathophysiology of OSA is complex and multifactorial. Additionally, the etiological mechanisms vary considerably among affected individuals, with numerous aspects remaining unclear or insufficiently understood [6]. With the accumulation of research evidence it is increasingly acknowledged that both anatomical and functional factors contribute to upper airway collapse. In light of the interplay between anatomical and non-anatomical mechanisms

in the pathogenesis of OSA, the PALM model has been proposed. This framework encompasses four key components: pharyngeal critical closing pressure (P), reduced respiratory arousal threshold (A), elevated loop gain (L), and the responsiveness of upper airway dilator muscles (M) [6, 11].

P- pharyngeal critical closing pressure

Anatomical abnormalities of the pharynx and craniofacial structures have been identified through upper airway imaging in individuals who snore, both with and without OSA. Patients diagnosed with OSA demonstrate a higher prevalence of upper airway structural anomalies compared to primary snorers. The morphology of the oropharynx and hypopharynx has been shown to vary significantly with BMI in both groups, becoming more spherical in individuals with higher BMI-primarily as a result of a reduction in the transverse airway dimension [12].

Cephalometric analysis reveals that adults with OSA, in comparison to healthy controls, typically exhibit a narrowed pharyngeal airway space, caudally displaced hyoid bone, and increased vertical facial dimensions [13]. Anatomical abnormalities associated with skeletal malformations, such as those observed in Pfeiffer syndrome (craniofacial synostosis), Pierre Robin syndrome (midface hypoplasia), as well as Crouzon and Apert syndromes, have also been implicated in the development of OSA [14].

Accumulating evidence supports the notion that pharyngeal collapsibility constitutes a critical determinant in the pathophysiology of upper airway obstruction. Notably, approximately 25% of patients with moderate to severe OSA undergoing continuous positive airway pressure (CPAP) therapy exhibit only mild pharyngeal collapsibility, suggesting their potential eligibility for alternative, more tolerable non-CPAP treatment modalities [6, 15, 16].

A- reduced respiratory arousal threshold

Respiratory stimuli such as hypoxia, hypercapnia, or increased breathing effort can trigger brief arousals from sleep. The intensity of stimulation required to induce such arousals is referred to as the respiratory arousal threshold, which varies significantly between individuals. Individuals with a low threshold tend to awaken more easily in response to mild airway obstruction and are prone to frequent sleep disruptions [17]. Upper airway vulnerability is a universal feature among individuals with OSA. Research has demonstrated that over 90% of patients with REM-specific OSA present with a low respiratory arousal threshold [18].

Arousal serves a dual role in the pathophysiology of OSA. On one hand, arousals at the termination of respiratory events act as a crucial protective response, facilitating the restoration of airway patency through neuromuscular activation and respiratory compensation, thereby resuming normal breathing and preventing asphyxia during sleep. On the other hand, a reduced

respiratory arousal threshold contributes to frequent microarousals, resulting in marked sleep fragmentation and impaired sleep quality in affected individuals [6].

L- elevated loop gain

Loop gain, a parameter within ventilatory control, quantifies the degree of respiratory instability and reflects the sensitivity and responsiveness of the ventilatory chemoreflex system. Mathematically, it can be described as the ratio of the ventilatory response (e.g., hyperpnea) to the initial disturbance (such as apnea or hypopnea).When the loop gain is below 1, a respiratory disturbance triggers a corrective response that is proportionate and allows ventilation to return to a stable pattern relatively quickly. In contrast, when the loop gain exceeds 1, the response to the disturbance is exaggerated, resulting in persistent oscillations in ventilation, characterized by cyclical waxing and waning. Elevated loop gain is considered a fundamental pathophysiological component contributing to the development and persistence of OSA [6, 19]. During wakefulness, ventilation is regulated by both metabolic chemoreflexes and a behavioral drive from suprapontine centers, which helps maintain breathing even in the presence of minimal chemical stimuli. This behavioral drive is lost during sleep, leaving ventilation dependent on chemoreflex control. In non-REM sleep, a drop in $PaCO_2$ below eupneic levels can suppress respiratory drive, causing central apnea until $PaCO_2$ rises again. The magnitude of ventilatory drive influences not only the activity of thoracic pump muscles but also that of the upper airway dilator muscles. Instability in chemoreflex-mediated ventilatory control may contribute to the pathogenesis of OSA, as the upper airway becomes more prone to collapse when $PaCO_2$ levels-and consequently, neural drive to the pharyngeal dilator muscles-are reduced [20].

M- the responsiveness of upper airway dilator muscles

Elevated activity of pharyngeal dilator muscles, observed in patients with OSA compared to matched controls, has been interpreted as a neuromuscular compensatory mechanism that counteracts anatomical vulnerabilities associated with OSA. During wakefulness, neuronal stimulation maintains activation of these dilator muscles, thereby preventing pharyngeal narrowing and collapse and preserving airway patency. However, at the onset of sleep, the reduction or loss of this upper airway dilator muscle activity diminishes the ability to sustain airway openness, increasing the risk of airway narrowing and collapse [6].

Symptoms

Clinical manifestations are essential for identifying patients with OSA. However, no single symptom is exclusively indicative of the disorder. Over time, significant progress has been made in recognizing symptoms and identifying risk factors to improve the diagnosis and

management of OSA. Although excessive daytime sleepiness is the most commonly reported symptom, it occurs in only 15% to 50% of individuals with OSA in the general population. Other frequent symptoms include loud snoring, gasping or choking during sleep, dry mouth upon awakening, insomnia, nighttime awakenings, and difficulties with concentration or attention [1, 21, 22].

Neurocognitive impairments, particularly those affecting attention, working memory, episodic memory, and executive functioning, are frequently observed in patients with OSA. These disturbances often overlap with depressive and anxiety-related symptoms, suggesting a multifactorial etiology [23].

Headache is another commonly reported complaint among individuals with OSA and insomnia; however, morning headaches are more specific to OSA and have been shown to correlate positively with disease severity, supporting the involvement of distinct pathophysiological mechanisms [24]. Additional clinical manifestations may include:

Restless legs syndrome (RLS) - a chronic sensorimotor neurological condition - which occurs more frequently in patients with OSA (7–36%) compared to the general population, indicating a frequent comorbidity.

Sleep bruxism, defined as repetitive jaw-muscle activity such as clenching or grinding of the teeth, which is observed in 26–54% of OSA patients, in contrast to 13% in the general adult population [25].

Nocturia, which affects approximately 75.8% of individuals diagnosed with OSA, regardless of sex. Treatment with continuous positive airway pressure (CPAP) has been shown to significantly reduce the frequency of nocturia episodes [26].

Bilateral relation between OSA and Obesity

Obesity represents the primary risk factor for the development of OSA. Cross-sectional research has established a robust association between elevated BMI and the risk of OSA. It has been reported that approximately 40% of adults with obesity exhibit severe OSA, and over 70% of patients diagnosed with OSA in sleep clinic populations are obese. Furthermore, a population-based prospective study demonstrated that a 10% increase in body weight corresponds to a sixfold higher risk of OSA progression, whereas a 10% reduction in weight is associated with a 26% improvement in OSA severity [27, 28]. On the other hand, OSA disrupts sleep continuity, causing persistent sleep deprivation and excessive daytime sleepiness. These effects promote decreased physical activity, alongside increased appetite, which together contribute to weight gain [4, 29].

Obesity as the main cause of OSA

Abdominal obesity, characterized by increased visceral fat, is associated with a higher release of inflammatory cytokines compared to peripheral subcutaneous fat accumulation. This pro-inflammatory state may contribute to metabolic abnormalities and increase neck adiposity and upper airway fat deposition, even among individuals with normal overall body weight. Epidemiological evidence highlights that neck circumference is a more reliable predictor of OSA risk than waist circumference, underscoring the critical role of regional fat accumulation in the pathogenesis of the disease [30, 31]. The accumulation of fatty tissue in the neck and pharyngeal regions leads to anatomical narrowing of the upper airway, which increases the likelihood of airway obstruction during sleep. This localized fat deposition is independently correlated with the severity of OSA, as reflected by AHI. Obesity also contributes to OSA through its impact on respiratory mechanics, particularly by reducing lung volumes. Increased abdominal and thoracic adiposity elevates pressure on the diaphragm and chest wall, leading to a decline in end-expiratory lung volume [32, 33]. This reduction in lung volume impairs the caudal traction exerted on the upper airway during inspiration, which normally helps maintain airway patency. The resulting loss of tracheal "tug" promotes narrowing of the upper airway, particularly in the hypotonic state observed during sleep. Moreover, the recumbent posture during sleep further exacerbates this effect, intensifying the impact of abdominal fat mass on diaphragm positioning and upper airway stability [34]. Collectively, these mechanical alterations increase the susceptibility to pharyngeal collapse, thereby contributing to the pathophysiology of OSA and promoting recurrent episodes of airflow obstruction during sleep [32]. Notably, weight reduction in patients with sleep apnea improves upper airway function and attenuates disease severity, which is attributed to both anatomical changes and shifts in protective and pathogenic adipokine profiles [30].

OSA as a contributor to weight gain

OSA can contribute to sleep fragmentation, excessive daytime sleepiness, and overall poor sleep quality. These disturbances may enhance sympathetic nervous system activity and promote insulin resistance, both of which are factors that can aggravate obesity. Additionally, OSA has been linked to alterations in appetite-regulating hormones such as leptin, ghrelin, and orexin, leading to increased hunger, elevated caloric intake, and subsequent weight gain, thereby further reinforcing the cycle of obesity [33].

Ghrelin is currently the only identified gut-derived peptide with orexigenic properties. Its characteristic rise before meals and decline following food intake has supported its

classification as a key "hunger hormone" implicated in the initiation of feeding. Beyond its role in the short-term regulation of appetite, ghrelin also contributes to the long-term control of energy balance by promoting energy storage through the reduction of fat utilization [35].
The association between OSA and circulating ghrelin concentrations, as well as the impact of CPAP therapy on these levels, remains a subject of ongoing debate within the scientific community. Önder Öztürk et al. in their research from 2022 and Ümmiye Biçer et al. in their paper from 2021 indicate an increased level of ghrelin in individuals with OSA [36, 37]. On the other hand, the findings of the meta-analysis suggest that, overall, there was no statistically significant difference in serum or plasma ghrelin levels between adults with OSA and healthy controls, nor between pre- and post-CPAP therapy in OSA patients. However, after the exclusion of outlier studies, ghrelin levels were found to be significantly elevated in individuals with OSA compared to controls. Trial Sequential Analysis (TSA) indicated that two of the subgroup analyses were limited by small sample sizes. Factors such as the type of blood sample collected, sample size, study quality, participants' mean age, and AHI influenced outcomes in the case–control studies, while mean AHI prior to CPAP initiation was a relevant variable in before–after studies. Consequently, these results warrant further confirmation through larger, high-quality investigations [38].

Leptin primarily functions as a satiety hormone, reducing the drive to eat. Beyond appetite regulation, leptin and its receptor play crucial roles in modulating energy expenditure, maintaining glucose homeostasis, influencing inflammatory pathways, and regulating immune system activity [29]. Leptin dysregulation is frequently observed in individuals with OSA, especially among those with coexisting obesity, and is thought to play a critical role in energy homeostasis, adipose tissue distribution, and body weight regulation [4]. While circulating leptin levels are generally elevated in obese individuals, evidence suggests that OSA independently contributes to a significant further increase in leptin concentrations. The meta-analysis demonstrated that plasma and serum leptin concentrations were significantly elevated in individuals with OSA. compared to healthy controls across almost all subgroups [29]. This elevation was further supported by data showing mean leptin levels of 22.57 ± 4.23 ng/mL in OSA patients versus 5.71 ± 3.02 ng/mL in controls, indicating a marked difference between the two groups [39]. Notably, meta-regression analysis revealed that factors such as age, BMI, disease severity, assay methodology, study design, type of PSG, and ethnicity did not independently influence leptin levels. Additionally, a positive correlation was observed between serum/plasma leptin levels and AHI, suggesting a direct association between leptin

concentration and OSA severity [29]. Chronic hyperleptinemia may lead to the development of leptin resistance-an attenuated physiological response to leptin's appetite-suppressing and metabolic actions. This resistance can impair the hormone's ability to regulate body weight and energy expenditure, potentially contributing to systemic low-grade inflammation and metabolic disturbances, thereby aggravating the underlying mechanisms of OSA [40].

Emerging evidence from studies on metabolic syndrome further supports this association, with elevated leptin concentrations commonly reported in patients with OSA. These findings suggest a state of relative leptin resistance in this population and highlight the interplay between OSA and key features of metabolic syndrome, such as insulin resistance. Additionally, genetic studies have identified polymorphisms in the leptin receptor gene that may influence the risk and severity of OSA, reinforcing the role of disrupted adipokine signaling pathways in the pathogenesis of sleep-disordered breathing [39].

Sleep fragmentation and chronic sleep insufficiency caused by OSA often result in excessive daytime sleepiness (EDS), diminished energy levels, and persistent fatigue, which in turn reduces physical activity levels and increases appetite, ultimately contributing to further weight gain [4, 41]. Studies indicate an inverse correlation between physical activity (PA) and OSA severity, suggesting that individuals with more severe OSA exhibit lower PA levels, thereby increasing their risk for obesity and cardiovascular comorbidities. Both EDS and obesity, prevalent among OSA patients, are significant modulators of physical activity patterns [42]. This reciprocal interaction between OSA and obesity creates a self-perpetuating feedback loop, wherein each condition exacerbates the severity of the other [41].

CPAP Therapy and Weight Change in Obstructive Sleep Apnea

Research on the impact of CPAP therapy on body weight in patients with OSA has yielded mixed results. In most CPAP-treated individuals, weight does not change significantly, though the rate of weight gain appears to be slower compared to that observed in the general age-matched population. Notably, approximately 10% of patients continue to experience progressive weight gain despite high adherence to CPAP, placing them at elevated risk for OSA-related multimorbidity in the long term. Contrastingly, other findings suggest that CPAP treatment may actually contribute to an increase in BMI and weight, highlighting the need for adjunctive weight management strategies in OSA care [43, 44, 45].

However, CPAP treatment of OSA can prolong weight loss in patients after bariatric surgery. In a cohort of bariatric surgery patients diagnosed with OSA, the majority experienced significant weight regain over time. Notably, adherence to CPAP therapy was associated with

more favorable long-term weight loss outcomes. These observations suggest that untreated OSA may negatively impact the sustainability of weight reduction following bariatric surgery, highlighting the critical role of OSA management in supporting long-term postoperative success [46].

A meta-analysis by Baixin Chen et. al. from 2021 indicates that BMI significantly increased in patients with OSA following CPAP therapy compared to control interventions. This weight gain was often accompanied by increases in waist and neck circumferences. Subgroup analyses and meta-regression revealed that BMI increase was primarily observed in patients with poor CPAP adherence (≤5 hours/night), while those with better adherence (>5 hours/night) did not exhibit this effect. Additionally, BMI rose significantly in patients without cardiovascular disease (CVD) and in those with baseline dysglycemia, whereas patients with CVD experienced a decrease in BMI. These results suggest that the impact of CPAP on weight is influenced by adherence level and baseline metabolic or cardiovascular status [47].

Several hypotheses have been proposed to elucidate the mechanisms behind weight gain observed after the commencement of CPAP therapy. At first, leptin has been found to decrease following CPAP therapy, as shown in two meta-analyses. While reduced leptin levels may suggest increased appetite and potential weight gain, they could also indicate improved leptin sensitivity, particularly in patients with high CPAP adherence (>5 h/night), which may help regulate appetite and stabilize body weight. Conversely, poor adherence (≤5 h/night) might not confer this benefit, potentially contributing to increased caloric intake and weight gain [47]. Additionally, studies using indirect calorimetry have demonstrated that CPAP therapy led to a significant reduction in basal metabolic rate by approximately 5%, thereby contributing to a positive energy balance, likely due to reduced respiratory effort during sleep. This decline in energy expenditure, in the absence of increased physical activity, may promote positive energy balance and weight gain. However, variables related to energy intake-particularly dietary behaviors-exerted a greater influence on body weight changes than those associated with energy expenditure [47, 48]. Another explanation is that individuals with poor adherence to CPAP therapy may compensate by demonstrating greater motivation to modify lifestyle behaviors, such as improving dietary habits or increasing physical activity. In contrast, patients who adhere successfully to CPAP and experience symptomatic relief may perceive less need for additional behavioral changes, thereby exhibiting lower motivation to engage in supportive lifestyle interventions [43]. Moreover, short-term randomized trials suggest that CPAP use may lead to fluid retention, as indicated by increased extracellular water and body weight. Further well-

designed, large-scale RCTs are warranted to elucidate the multifactorial mechanisms underlying post-CPAP weight changes [47].

Summary

Based on the literature review presented in the article, obesity is identified as one of the most common indirect causes of OSA. However, OSA itself may also contribute to weight gain or hinder weight loss through mechanisms described in the article. This bidirectional relationship can lead to a vicious cycle in which each condition exacerbates the other. Therefore, clinicians should aim to detect or rule out OSA as early as possible in patients with obesity, to enable the implementation of a more effective and integrated therapeutic approach to both disorders.

The impact of CPAP therapy on body weight remains inconclusive, as highlighted in the article. Weight gain has been observed particularly among patients with poor adherence to CPAP therapy. Although the underlying mechanisms are not yet fully understood, proposed explanations in the literature include hormonal alterations, significant reductions in basal metabolic rate, diminished need for behavioral changes, reduced motivation to engage in lifestyle interventions, and fluid retention associated with CPAP use. Given the lack of definitive evidence, future studies should focus on elucidating the hormonal and physiological mechanisms underlying OSA–obesity axis, optimizing CPAP-based interventions, and developing targeted weight management strategies for this high-risk population.

Moreover, it is essential that physicians introducing CPAP therapy in patients underscore the critical importance of simultaneous behavioral interventions, which play a key role in mitigating the risk of post-treatment weight gain.

Disclosure

Author's contribution

Conceptualization: Marek Dróżdż;

Methodology: Aneta Tkaczyk, Magdalena Mandyna;

Software: Bartosz Maczkowski;

Check: Aleksandra Przelaskowska;

Formal analysis: Alicja Czyszczoń;

Investigation: Marek Dróżdż, Jan Puliński;

Resources: Aneta Tkaczyk, Bartosz Maczkowski;

Data curation: Szymon Mikler;

Writing-rough preparation: Wiktoria Boral, Marek Dróżdż;

Writing–review and editing: Wiktoria Boral;
Visualization: Szymon Mikler;
Supervision: Alicja Czyszczoń;
Project administration: Aleksandra Przelaskowska, Jan Puliński.
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