Healthsains
Printed ISSN: 2722-7782 | Electronic ISSN: 2722-5356
Volume 7, No. 5 May, 2026
p ISSN 2723-6927-e ISSN 2723-4339

Obstructive Sleep Apnea-Hypopnea Syndrome as a Risk Factor for Polycystic Ovary Syndrome: A Narrative Review of Prevalence, Pathophysiological Mechanisms, and Clinical Implications

Reza Deni Rohmansyah
RSIA Hidayah Ibu Kalianda, Indonesia
Email: rezarohmansyah@yahoo.com

Keywords
Obstructive Sleep Apnea-Hypopnea Syndrome, Polycystic Ovary Syndrome, insulin resistance, hyperandrogenism, infertility, Continuous Positive Airway Pressure

Abstract

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine and metabolic disorders affecting women of reproductive age worldwide. Recent evidence indicates that Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) is frequently associated with PCOS and may contribute to worsening metabolic, hormonal, and reproductive dysfunctions. This study aimed to examine the role of OSAHS as a risk factor for PCOS by reviewing its prevalence, pathophysiological mechanisms, and clinical implications. This research employed a narrative review approach using relevant scientific literature obtained from PubMed, ResearchGate, and HHS Public Access databases. Articles discussing OSAHS and PCOS published in indexed journals were systematically analyzed to identify prevalence patterns, biological interactions, and therapeutic implications. The findings demonstrated that the prevalence of OSAHS in women with PCOS ranged from 20% to 37%, with higher incidence reported among obese and adult women. Several studies also identified a bidirectional causal relationship between OSAHS and PCOS. Pathophysiologically, intermittent hypoxia, oxidative stress, insulin resistance, inflammation, sympathetic activation, and hypothalamic-pituitary-gonadal axis dysfunction were found to contribute to hyperandrogenism, infertility, and increased cardiovascular risk. In conclusion, OSAHS plays a significant role in aggravating PCOS manifestations, highlighting the importance of early screening and comprehensive management to improve metabolic and reproductive outcomes in affected women.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a complex metabolic syndrome with more than one etiology involving the interaction of genetic, lifestyle, and environmental factors. PCOS is the most common endocrine disorder found in women of reproductive age with a prevalence rate ranging from 8% to 13% (Liu et al., 2024). Clinical symptoms that can be found include disruptions in the menstrual cycle resulting in irregular menstrual cycles, excessive hair growth in areas of the body that have high levels of sensitivity to androgen hormones, and the discovery of enlarged ovaries (Kahal et al., 2020; Thannickal et al., 2020). In addition, it has also been found that sleep disorders, especially Obstructive Sleep Apnea (OSA), are very common in PCOS (Sam & Tasali, 2021).

Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) is a sleep disorder characterized by repeated episodes of upper airway occlusion during sleep, leading to cessation of breathing (Apnea) or decreased airflow (Hypopnea), decreased oxygen saturation, and deteriorated sleep quality (He et al., 2024). Risk factors for OSAHS include overweight, advanced age, central obesity, hyperandrogenism, and ethnicity.

Polycystic Ovary Syndrome (PCOS) remains a major global women's health issue because

it affects reproductive, metabolic, psychological, and long-term cardiometabolic outcomes. The World Health Organization reports that PCOS affects an estimated 10–13% of women globally, while up to 70% of affected women may remain undiagnosed, showing that the disorder is still under-recognized despite its high burden. PCOS is commonly characterized by hyperandrogenism, ovulatory dysfunction, insulin resistance, menstrual irregularity, infertility, and increased metabolic risk. Therefore, PCOS should not be viewed only as a gynecological disorder, but also as a chronic endocrine-metabolic condition requiring integrated clinical attention.

At the same time, obstructive sleep apnea-hypopnea syndrome (OSAHS) has become an increasingly important public health concern. OSAHS is characterized by repeated upper-airway obstruction during sleep, resulting in intermittent hypoxia, sleep fragmentation, sympathetic activation, and poor sleep quality. Global estimates indicate that approximately 936 million adults may have obstructive sleep apnea, with about 425 million experiencing moderate-to-severe disease. Although OSAHS has historically been more frequently diagnosed in men, growing evidence suggests that it is clinically relevant in women, especially among those with obesity, insulin resistance, endocrine disorders, and reproductive dysfunction.

The specific issue addressed in this research is the possible role of OSAHS as a risk factor that worsens PCOS. The uploaded manuscript explains that OSAHS in women with PCOS is not merely a comorbidity, but may contribute to a worsening cycle involving insulin resistance, oxidative stress, adipose tissue dysfunction, hypothalamic-pituitary-gonadal axis disturbance, hyperandrogenism, and reduced reproductive function. This relationship is clinically important because many women with PCOS already experience metabolic and reproductive complications, while sleep-related breathing disorders may remain undetected unless clinicians actively screen for them.

Several recent studies have strengthened the evidence linking OSAHS and PCOS. He et al. (2024), in an updated meta-analysis, found that 20.8% of women with PCOS had comorbid OSAHS, with worse metabolic indicators among those affected. Kahal et al. (2020) reported an even higher pooled prevalence, showing that 35.0% of women with PCOS had obstructive sleep apnea. Abdul Jafar et al. (2025) further found that 37.0% of individuals with PCOS had OSA, compared with 6.0% among individuals without PCOS, and that the risk increased with symptom severity.

Previous research has also shown that the relationship between both disorders may be bidirectional. Liu et al. (2024), through bidirectional Mendelian randomization, suggested a causal relationship between PCOS and increased OSAS risk, indicating that these conditions may reinforce each other through shared biological pathways. Sam and Tasali (2021) also emphasized that OSA may contribute to insulin resistance and glucose intolerance in women with PCOS, thereby increasing metabolic risk. These findings show that OSAHS may influence not only sleep quality but also the broader endocrine and metabolic phenotype of PCOS.

Despite this growing evidence, an important research gap remains. Many previous studies have focused mainly on prevalence, while fewer have comprehensively explained how OSAHS contributes to PCOS progression through integrated mechanisms involving hypoxia, inflammation, oxidative stress, insulin resistance, androgen excess, reproductive dysfunction, and cardiovascular risk. In addition, differences in diagnostic criteria, age group, ethnicity, obesity status, and OSAHS assessment methods create variation in reported prevalence. Therefore, a narrative review is needed to synthesize the available evidence and present a clearer conceptual

understanding of the relationship between OSAHS and PCOS.

The urgency of this research lies in the fact that OSAHS is a manageable condition, but it is often overlooked in women with PCOS. The 2023 International Evidence-based Guideline for PCOS emphasizes the need for improved assessment and management because PCOS care remains inconsistent and many women experience delayed diagnosis. If OSAHS contributes to worsening insulin resistance, hyperandrogenism, infertility, and cardiovascular risk, then failure to detect sleep apnea may limit the effectiveness of PCOS management. Early screening may therefore improve both metabolic and reproductive outcomes.

The novelty of this research is its focus on positioning OSAHS as an active risk factor in PCOS rather than only a coexisting sleep disorder. By integrating prevalence evidence, pathophysiological mechanisms, and clinical implications, this study offers a more comprehensive perspective on how sleep-disordered breathing may worsen PCOS manifestations. This approach is important because it connects sleep medicine, endocrinology, gynecology, and metabolic health within one explanatory framework.

The purpose of this research is to review and analyze the association between OSAHS and PCOS based on current evidence regarding prevalence, biological mechanisms, and clinical consequences. Specifically, this study aims to explain how intermittent hypoxia, sleep fragmentation, oxidative stress, inflammation, insulin resistance, decreased sex hormone-binding globulin, and hypothalamic-pituitary-gonadal axis dysfunction may contribute to hyperandrogenism, infertility, and cardiometabolic complications in women with PCOS.

This research is expected to contribute theoretically by strengthening the conceptual understanding of the bidirectional relationship between OSAHS and PCOS. Practically, the findings may support clinicians in considering routine OSAHS screening among women with PCOS, especially those with obesity, insulin resistance, severe metabolic symptoms, or sleep-related complaints. The benefit of this research is that it may encourage more comprehensive PCOS management by integrating sleep evaluation, early detection, and appropriate interventions such as lifestyle modification and continuous positive airway pressure therapy when indicated.

RESEARCH METHODS

This study employed a quantitative research design using a cross-sectional approach to examine the relationship between Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) and Polycystic Ovary Syndrome (PCOS) among women of reproductive age. The research population consisted of women diagnosed with PCOS based on the Rotterdam criteria who attended endocrine and gynecology clinics during the study period. The sample was determined using purposive sampling techniques with inclusion criteria including women aged 18–40 years diagnosed with PCOS and willing to participate in the study, while exclusion criteria included pregnancy, severe chronic disease, and previous sleep disorder treatment. The minimum sample size was calculated using a correlation analysis formula with a 95% confidence level and 5% margin of error to ensure adequate statistical power. This study focused on identifying the prevalence and clinical association of OSAHS symptoms, metabolic disturbances, and reproductive abnormalities in women with PCOS.

The research instruments included structured questionnaires, anthropometric measurement sheets, and clinical examination forms. OSAHS risk was assessed using the STOP-Bang Questionnaire and Epworth Sleepiness Scale, while PCOS clinical characteristics were obtained

from medical records and laboratory examinations. Anthropometric parameters such as body mass index (BMI), waist circumference, blood pressure, fasting glucose, and lipid profile were also collected. Instrument validity was evaluated using Pearson Product Moment correlation analysis, where item correlation coefficients greater than 0.30 were considered valid. Reliability testing was conducted using Cronbach's Alpha coefficient with a minimum acceptable value of 0.70 indicating reliable instruments. Data collection was carried out through direct interviews, questionnaire distribution, physical examination, and documentation review. The research procedure consisted of participant recruitment, informed consent, questionnaire administration, clinical assessment, data verification, and data coding before statistical analysis.

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 27 and Microsoft Excel for data tabulation and visualization. Descriptive statistical analysis was used to present respondent characteristics, prevalence rates, mean values, standard deviations, frequencies, and percentages. Inferential statistical analysis included the Chi-square test to assess associations between categorical variables, independent t-tests to compare mean differences between groups, and Pearson correlation analysis to evaluate the relationship between OSAHS severity and metabolic indicators in PCOS patients. Multiple linear regression analysis was further conducted to identify significant predictors of OSAHS occurrence in women with PCOS while controlling for confounding variables such as age and obesity. The results were interpreted using a significance level of $p < 0.05$ to determine statistical significance and support the study objectives.

RESULTS AND DISCUSSION

Table 1. Prevalence and Association of Obstructive Sleep Apnea-Hypopnea Syndrome in Women with Polycystic Ovary Syndrome Based on Previous Studies

Source	Reported prevalence	Method
Abdul Jafar et al. (2025)	Prevalence of combined OSAHS in PCOS women was 37.0% (95% CI: 17.0–57.0%)	*Meta-analysis*
He et al. (2024)	OSAHS morbidity in women with PCOS was 20.8% (95% CI: 14.7–27.6%) Caucasian (27.2%) compared to Asian (9.6%).	*Meta-analysis*
Kahal et al. (2020)	OSAHS morbidity in the group of women with PCOS was 20.8% (95% CI: 14.7–27.6%)	*Systematic review* dan *Meta-analysis*
Helvaci et al. (2017)	The combined prevalence of OSA in PCOS patients was 22% (95% CI: 8–40%), with a higher prevalence in adults (32%, 95% CI: 13–55%) compared to adolescents (8%, 95% CI: 0–30%)	*Meta-analysis*
Sam & Tasali (2021)	the group of women with PCOS had a higher incidence of OSAHS over a follow-up period of 2 to 8 years, with an adjusted hazard ratio of 2.6 (95% CI: 1.6–4.0)	*Narrative review*
Liu et al. (2024)	OSAS is causally associated with an increased risk of PCOS (OR: 1.463; 95% CI: 1.086–1.971; p=0.012) PCOS is also causally associated with an increased risk of OSAS (OR: 1,041; 95% CI: 1,012–1,072; p=0.006)	*Mendellian Randomization (MR)*

Source: Compiled from Abdul Jafar et al. (2025), He et al. (2024), Kahal et al. (2020), Helvaci et al. (2017), Sam & Tasali (2021), and Liu et al. (2024).

The meta-analysis conducted by Abdul Jafar et al. (2025) strictly included only studies that used polysomnography (PSG) as a diagnostic tool. Of the eight cross-sectional studies that met the inclusion criteria, it was found that the prevalence of combined OSAHS in women with PCOS was 37.0% (95% CI: 17.0–57.0%), with the prevalence in adolescents at 29.0% and adults at 40.0%, compared to only 6.0% (95% CI: 1.0–10.0%) in the population without PCOS. Women with PCOS had a 9.52-fold higher risk of OSAHS than women without PCOS (OR: 9.52; 95% CI: 3.90–23.26; $p<0.001$), and this risk increased with symptom severity: AHI≥5 alone (OR: 3.90; 95% CI: 1.63–9.34), AHI≥5 with symptoms (OR: 17.95; 95% CI: 6.17–52.22), to AHI≥10 with symptoms (OR: 30.61; 95% CI: 7,99–117,25).

Another meta-analysis by He et al. (2024) sourced from 21 articles consisting of CNKI, EMBASE, PubMed, Web of Science, and Wanfang found OSAHS morbidity in the group of women with PCOS to be 20.8% (95% CI: 14.7–27.6%). The study also reported that the prevalence of OSAHS was higher in women with PCOS with obesity (27.5%) compared to no obesity (15.8%), in adults (21.5%) compared to adolescents (17.4%), and in Caucasian ethnicity (27.2%) compared to Asians (9.6%). In addition, most metabolic indicators—including fasting blood glucose, HOMA-IR, systolic and diastolic blood pressure, and lipid profiles—were significantly worse in women with PCOS and OSAHS than in those with only PCOS.

A systematic review by Kahal et al. (2020) and a meta-analysis involving 17 studies with a total of 648 participants all using polysomnography diagnostic tools found that the prevalence of combined OSAHS in women with PCOS was 35.0% (95% CI: 22.2–48.9%). This prevalence was not influenced by variations in the definition of PCOS between studies. Analysis of the subgroup showed that the prevalence of OSA was higher in adults than adolescents, as well as in obese women compared to non-obese. When compared to controls, women with PCOS had a 3.83-fold higher risk of OSA (OR: 3.83; 95% CI: 1.43–10.24).

Helvaci et al. (2017) in a meta-analysis involving 8 studies in adults and 5 studies in adolescents with a total of 404 PCOS patients and 611 control subjects found that the prevalence of combined OSA in PCOS patients was 22% (95% CI: 8–40%), with a higher prevalence in adults (32%, 95% CI: 13–55%) compared to adolescents (8%, 95% CI: 0–30%). The risk of OSA was significantly increased in adult PCOS patients with an OR of 9.74 (95% CI: 2.76–34.41), while in the adolescent group the increased risk was not significant (OR: 4.54; 95% CI: 0.56–36.43) (Abdul Jafar et al., 2025; Helvaci et al., 2017). A narrative review by Sam & Tasali (2021) also confirmed that the average prevalence of OSA from various clinical studies in women with PCOS was about 40%, although these clinical studies were limited by small sample sizes and the inclusion of mostly PCOS women who were overweight or obese.

A population-based study conducted by Sam & Tasali (2021) discussed a longitudinal study from the Taiwan National Health Insurance Research Database (n=4,595) that showed that the group of women with PCOS had a higher incidence of OSAHS during the follow-up period of 2 to 8 years, with an adjusted hazard ratio of 2.6 (95% CI: 1.6–4.0), after adjusting for obesity and other factors. A larger-scale population-based study from the United Kingdom, The Health Improvement Network (THIN), with 76,978 women with PCOS and 143,077 controls, reported an adjusted hazard ratio of 2.3 (95% CI: 1.9–2.7) for OSA risk in PCOS, even after adjusting for age and weight. In the UK cohort study, an increased risk of OSA in PCOS was particularly seen in the overweight or overweight group. obesity, but the results did not reach statistical significance in the normal weight group. These findings are sufficiently confirming that obesity factors cannot fully explain the increased risk factors for OSA in PCOS, and there are other

intrinsic mechanisms involved.

Liu et al. (2024) conducted a Mendelian Randomization (MR) analysis and from the results obtained provided strong evidence of a two-way causal relationship between PCOS and OSAS. Using data from GWAS involving 3,609 cases of PCOS and 229,788 controls, as well as 38,998 OSAS subjects and 336,659 controls (all of European descent), the study found that OSAS was causally associated with an increased risk of PCOS (OR: 1,463; 95% CI: 1,086–1,971; p=0.012), and conversely PCOS was also causally associated with an increased risk of OSAS (OR: 1,041; 95% CI: 1,012–1,072; p=0.006). These two directions of causality remained significant after ejecting SNPs associated with obesity and BMI, suggesting that the causal relationship between PCOS and OSAS was not fully mediated by obesity.

Several major factors have been identified influencing the prevalence of OSAHS in PCOS. First, obesity was the dominant risk factor, with a consistently higher prevalence in the obesity group when compared to the non-obese group in all meta-analyses. Obesity can play a role in OSAHS through an increase in fat in the parapharynx which can narrow the upper airway, decreased thoracic complications, and changes in neural compensation mechanisms and control systems in the respiratory system. Second, age is also an important factor, where the adult group of women with PCOS has a higher prevalence of OSAHS than the group of adolescent women. This is directly proportional to the increased risk of OSAHS with age in the general population, and suggests that the onset of OSAHS is preceded by PCOS. Third, ethnicity factors may affect prevalence, with Asian risk being lower when compared to white groups, it is possible that this is due to possible differences in craniofacial anatomy, fat distribution, and different arousal thresholds between ethnicities (Zuo et al., 2016). Fourth, PCOS diagnostic criteria: although there are variations in the definition of PCOS used in various studies (NIH vs. Rotterdam vs. other criteria), Abdul Jafar et al. (2025) specifically say that the increased risk of OSAS in PCOS is independent of the diagnostic criteria used. Fifth, the severity of symptoms: the more severe the OSAHS symptoms suffered, the higher the risk in the PCOS population, as documented by Abdul Jafar et al. (2025) through an increase in OR from AHI≥5 alone to AHI≥10 with symptoms. Sixth, insulin resistance and hyperandrogenism as the main features of PCOS also play a role in the increased risk of OSAHS. Hyperandrogenism in PCOS can predispose OSAHS through effects on the neural control of the respiratory system and upper airway mechanics, while insulin resistance and hyperinsulinemia can exacerbate both conditions simultaneously through inflammatory mechanisms and oxidative stress.

Pathophysiological Mechanisms

OSAHS is characterized by the occurrence of recurrent episodes of airway obstruction that causes intermittent hypoxia (repeated cycles of oxygen decline and reoxygenation) and sleep fragmentation. these two characteristics are the main triggers of a series of pathophysiological responses that have a direct impact on the worsening of PCOS.

Intermittent hypoxia can lead to excessive formation of Reactive Oxygen Species (ROS), leading to systemic oxidative stress and cellular damage. In PCOS, oxidative stress increases independently with age and Body Mass Index, and plays a role in worsening insulin resistance, causing hyperandrogenism, and playing a role in infertility.

OSAHS can worsen insulin resistance through intermittent hypoxia and sleep fragmentation that trigger 4 main pathways, namely inflammatory oxidative stress, adipose tissue dysfunction, pancreatic beta cell disorder, and sympathetic activation (Arnaud et al., 2024;

Murphy et al., 2017).Oxidative and inflammatory stress pathways occur when intermittent hypoxia triggers the formation of excess ROS and the release of proinflammatory cytokines that interfere with insulin signaling. The mechanism of adipose tissue dysfunction occurs when hypoxia induces inflammation in fat visceral, interfering with the work of insulin in the liver, muscles, and adipose tissue. Intermittent hypoxia can also impair the function of insulin-producing beta cells, resulting in a decreased ability to compensate for insulin resistance. Sleep fragmentation and hypoxia can increase sympathetic nerves that can trigger lipolysis and the release of free fatty acids, which can worsen insulin resistance independently of obesity.

Hyperandrogenism caused by OSAHS in PCOS can occur through the mechanism of SHBG Decreased Levels, insulin resistance due to sleep disturbances, and hypothalamic-pituitary-gonadal (HPG) axis dysfunction (Kent et al., 2015). SHBG is an androgen-binding protein found in the circulation, so only a small amount of testosterone is biologically active, a meta-analysis study by Abdul Jafar et al. (2025) found that significantly lower levels of SHBG were found in women who had OSAHS and PCOS compared to women who only had PCOS (Jafar et al., 2023). This study is directly proportional to the study conducted by He et al. (2024) which showed a significant decrease in SHBG levels in the PCOS group with OSAHS. Insulin resistance exacerbated by OSAHS can suppress the production of SHBG in the liver, resulting in increased levels of free testosterone which can exacerbate manifestations of hyperandrogenism such as hirsutism and acne. Sleep disturbances can trigger insulin resistance which then encourages the ovaries to produce more androgens. Recurrent episodes of apnea in OSAHS cause hypoxia which has been shown to interfere with the function of the HPG shaft which will result in an imbalance in hormone production gonadotropin (Azziz, 2018).

OSAHS causes recurrent oxygen deprivation conditions that directly affect brain performance, namely interfering with the normal function of the hormonal axis (Cojocaru et al., 2023). Research conducted by Beroukhim et al (2022) explains that in PCOS women, low progesterone levels are associated with increased respiratory distress during sleep.

OSAHS in PCOS significantly increases cardiovascular risk through the mechanisms of hypertension, endothelial dysfunction, atherosclerosis, and dyslipidemia triggered by sympathetic activation, oxidative stress, and chronic inflammation (Patel et al., 2018). Given that PCOS itself already increases cardiovascular risk, the presence of OSAHS adds to the risk burden cumulatively (Beroukhim et al., 2022). Therefore, the identification and management of OSAHS with CPAP in women with PCOS is an important strategy not only to improve sleep quality but also to reduce long-term cardiovascular morbidity and mortality in this high-risk population.

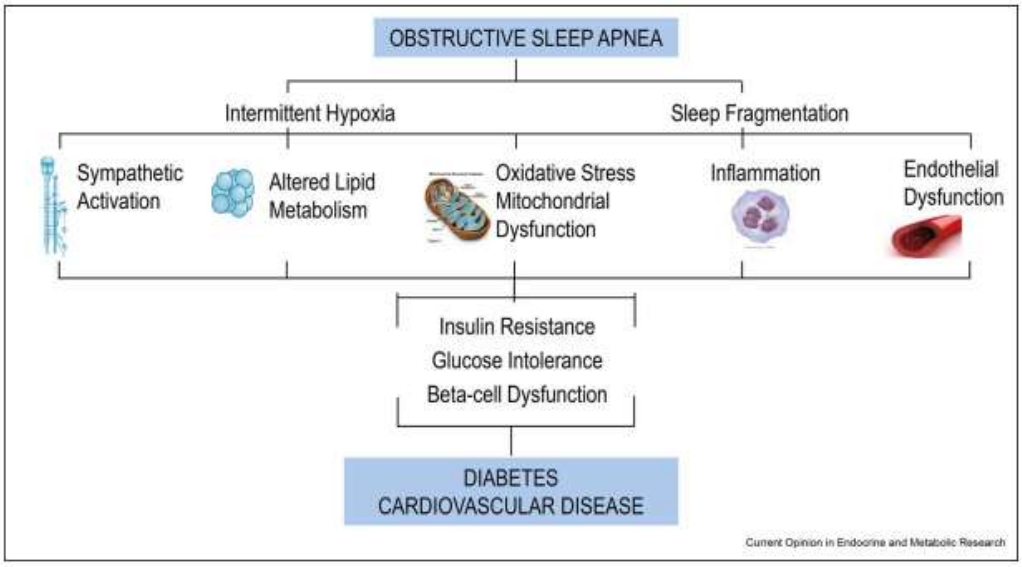

Figure 1. Potential mechanisms linking OSAHS to Metabolic.

Clinical Implications

These findings make it clear that there is significant clinical significance. Abdul Jafar et al. (2025) highlight that because OSAHS is a manageable condition, the recognition of OSAHS symptoms in individuals with PCOS can provide a substantial clinical advantage.6 The 2023 International PCOS Guidelines, supported by a meta-analysis by Abdul Jafar et al. (2025), recommend that women with PCOS undergo screening for OSAHS symptoms using a tested questionnaire followed by a thorough sleep apnea anamnesis, to direct OSA treatment to those who are most likely to benefit.6 He et al. (2024) explicitly suggest that clinicians give priority to the detection and treatment of OSAHS in women with PCOS.5

A clinical trial conducted by Shandong First Medical University, showed that there was a significant reduction of around 38% in serum testosterone, insulin and triglyceride levels in the group that received Continuous Positive Airway Pressure (CPAP) intervention therapy for 8 weeks compared to the group that only received lifestyle interventions (Heze Hospital Affiliated of Shandong First Medical University, 2026). These results are directly proportional to the research conducted by Abdul Jafar et al. (2025) which concluded that OSAHS As a treatable condition, identification and administration can provide significant clinical benefits, namely improvements in insulin resistance, decreased androgen levels, improved lipid profiles, and decreased sympathetic nerve activity.

CONCLUSION

Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) is an important risk factor in Polycystic Ovary Syndrome (PCOS) that is not only present as a comorbidity, but also plays a role in worsening metabolic, hormonal, and reproductive disorders. Various studies have shown that the prevalence of OSAHS in women with PCOS tends to be higher than in the population without PCOS, with an increasing risk in the adult group, obesity, and more severe symptoms. The relationship between OSAHS and PCOS is also bidirectional, so that they can reinforce each other in a complex pathophysiological circle. Pathophysiologically, OSAHS exacerbates PCOS through intermittent hypoxia, oxidative stress, inflammation, sympathetic nerve activation, insulin resistance, decreased SHBG, hypothalamic-pituitary-gonadal axis disorders, and

decreased reproductive function. This condition has an impact on increased hyperandrogenism, infertility, as well as cardiovascular risk in women with PCOS. Therefore, the presence of OSAHS in PCOS needs to be understood as a relevant clinical condition and requires special attention in medical practice. Thus, routine OSAHS screening in women with PCOS is very important, especially in patients with obesity, sleep disorders, insulin resistance, or severe metabolic symptoms. Early detection and proper management, including the use of Continuous Positive Airway Pressure (CPAP), have the potential to improve sleep quality, metabolic profile, androgen levels, and reproductive output. This review confirms that clinical approaches to PCOS should not only focus on gynecological and endocrine aspects, but also integrate the evaluation of sleep disorders as part of comprehensive management.

REFERENCES

Liu, P., Zhang, Q., Ding, H., & Zou, H. (2024). Association of obstructive sleep apnea syndrome with polycystic ovary syndrome through bidirectional Mendelian randomization. *Frontiers in Medicine, 11*, Article 1429783. https://doi.org/10.3389/fmed.2024.1429783

Thannickal, A., Brutocao, C., Alsawas, M., Morrow, A., Zaiem, F., Murad, M. H., & Chattha, A. J. (2020). Eating, sleeping and sexual function disorders in women with polycystic ovary syndrome (PCOS): A systematic review and meta-analysis. *Clinical Endocrinology, 92*(4), 338–349. https://doi.org/10.1111/cen.14153

Kahal, H., Kyrou, I., Uthman, O. A., Brown, A., Johnson, S., Wall, P. D. H., Metcalfe, A., Parr, D. G., Tahrani, A. A., & Randeva, H. S. (2020). The prevalence of obstructive sleep apnoea in women with polycystic ovary syndrome: A systematic review and meta-analysis. *Sleep and Breathing, 24*(1), 339–350. https://doi.org/10.1007/s11325-019-01835-1

Sam, S., & Tasali, E. (2021). Role of obstructive sleep apnea in metabolic risk in PCOS. *Current Opinion in Endocrine and Metabolic Research, 17*, 46–51. https://doi.org/10.1016/j.coemr.2021.01.002

He, J., Ruan, X., & Li, J. (2024). Polycystic ovary syndrome in obstructive sleep apnea-hypopnea syndrome: An updated meta-analysis. *Frontiers in Endocrinology, 15*, Article 1418933. https://doi.org/10.3389/fendo.2024.1418933

Abdul Jafar, N. K., Al Balushi, A., Subramanian, A., Lee, S. I., Bennett, C. J., Moran, L. J., Mousa, A., Tay, C. T., Teede, H. J., & Mansfield, D. R. (2025). Obstructive sleep apnea syndrome in polycystic ovary syndrome: A systematic review and meta-analysis. *Frontiers in Endocrinology, 16*, Article 1532519. https://doi.org/10.3389/fendo.2025.1532519

Helvaci, N., Karabulut, E., Demir, A. U., & Yildiz, B. O. (2017). Polycystic ovary syndrome and the risk of obstructive sleep apnea: A meta-analysis and review of the literature. *Endocrine Connections, 6*(7), 437–445. https://doi.org/10.1530/EC-17-0129

Zuo, T., Zhu, M., & Xu, W. (2016). Roles of oxidative stress in polycystic ovary syndrome and cancers. *Oxidative Medicine and Cellular Longevity, 2016*, Article 8589318. https://doi.org/10.1155/2016/8589318

Murphy, A. M., Thomas, A., Crinion, S. J., Kent, B. D., Tambuwala, M. M., Fabre, A., Pepin, J. L., Roche, H. M., Arnaud, C., & Ryan, S. (2017). Intermittent hypoxia in obstructive sleep apnoea mediates insulin resistance through adipose tissue inflammation. *European Respiratory Journal, 49*(4), Article 1601731. https://doi.org/10.1183/13993003.01731-2016

Arnaud, C., Kaba, M., Gonthier, B., Pepin, J. L., & Borel, A. L. (2024). Intermittent hypoxia

increases lipid insulin resistance in healthy humans: A randomized crossover trial. *Journal of Sleep Research, 34*(2), Article e14243. https://doi.org/10.1111/jsr.14243

Kent, B. D., McNicholas, W. T., & Ryan, S. (2015). Insulin resistance, glucose intolerance and diabetes mellitus in obstructive sleep apnoea. *Journal of Thoracic Disease, 7*(8), 1343–1357. https://doi.org/10.3978/j.issn.2072-1439.2015.08.11

Jafar, N. K. A., Bennett, C. J., Moran, L. J., & Mansfield, D. R. (2023). Beyond counting sheep: Exploring the link between polycystic ovary syndrome and sleep health. *Seminars in Reproductive Medicine, 41*(1–02), 45–58. https://doi.org/10.1055/s-0043-1777724

Azziz, R. (2018). Polycystic ovary syndrome. *Obstetrics & Gynecology, 132*(2), 321–336. https://doi.org/10.1097/AOG.0000000000002698

Cojocaru, C., Cojocaru, E., Pohaci-Antonesei, L.-S., Pohaci-Antonesei, C.-A., & Dumitrache-Rujinski, S. (2023). Sleep apnea syndrome associated with gonadal hormone imbalance (Review). *Biomedical Reports, 19*(6), Article 101. https://doi.org/10.3892/br.2023.1683

Beroukhim, G., Esencan, E., & Seifer, D. B. (2022). Impact of sleep patterns upon female neuroendocrinology and reproductive outcomes: A comprehensive review. *Reproductive Biology and Endocrinology, 20*(1), Article 16. https://doi.org/10.1186/s12958-022-00889-3

Patel, S. V., Gill, H., Shahi, D., Rajabalan, A., Patel, P., Sonani, R., ... & Patel, P. (2018). High risk for obstructive sleep apnea hypopnea syndrome predicts new onset atrial fibrillation after cardiac surgery: A retrospective analysis. *Sleep and Breathing, 22*(4), 1117–1124. https://doi.org/10.1007/s11325-018-1645-3

Heze Hospital Affiliated of Shandong First Medical University. (2026). Clinical characteristics and pregnancy outcomes in polycystic ovary syndrome complicated by obstructive sleep apnea: A correlative study. *Frontiers in Medicine, 13*, Article 1714294. https://doi.org/10.3389/fmed.2026.1714294