Interrelationship of Insulin Resistance and Obstructive Sleep Apnea in Polycystic Ovary Syndrome

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Abstract

Polycystic ovary syndrome (PCOS) is a common, heterogeneous endocrine disorder that affects approximately 6–15% of women of reproductive age and is characterized by hyperandrogenism, ovulatory dysfunction, and a strong metabolic feature: insulin resistance (IR) [7,12]. In recent years, growing evidence has linked PCOS with a range of sleep disturbances, including obstructive sleep apnea (OSA), reduced subjective sleep quality, and circadian misalignment, all of which may further exacerbate IR and other metabolic complications [3–6,8–11,16,17,21–25].

This review examined 15 peer-reviewed studies that reported on PCOS status, at least one sleep outcome, and at least one IR or metabolic measure. These included overnight polysomnography (PSG) cohorts, an intervention trial using continuous positive airway pressure (CPAP), adolescent circadian studies, questionnaire-based surveys, and meta-analyses [3–6,8–11,16,17,21–25]. Additional mechanistic reviews were consulted to outline biological pathways linking sleep disruption to impaired insulin signaling [2,13,18–20].

Across PSG-based studies, women with PCOS who also had OSA consistently showed higher fasting insulin, higher homeostatic model assessment of insulin resistance (HOMA-IR), or impaired glucose tolerance than PCOS women without OSA, even after accounting for body mass index [22,23,4,11,6,5,3]. In a CPAP intervention trial, treatment of OSA improved insulin sensitivity without significant weight change, suggesting that sleep-disordered breathing itself can drive metabolic dysfunction [21]. Studies using tools such as the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale showed that poorer reported sleep quality corresponded with higher HOMA-IR and greater metabolic syndrome severity [17,10,3]. Circadian research in adolescents linked delayed sleep timing and later melatonin offset to higher HOMA-IR, independent of BMI [16]. Meta-analyses confirmed that sleep problems are more prevalent in PCOS and often co-occur with adverse metabolic markers [9,24,25].

These findings indicate that sleep disturbances are an integral component of the metabolic profile of PCOS and should be addressed in both clinical practice and future research.

Introduction

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders in women of reproductive age, affecting an estimated 6–15% depending on diagnostic criteria and population studied [7,12]. PCOS is classically defined by clinical or biochemical hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, but beyond these reproductive features, it is strongly associated with metabolic dysfunction. Insulin resistance (IR) and the resulting hyperinsulinemia play a key role in the disorder’s pathophysiology, contributing

to androgen excess, reduced sex hormone–binding globulin, and increased long-term risk of dyslipidemia, hypertension, and type 2 diabetes [7,12,18,19].

Over the past two decades, researchers have begun to recognize that PCOS is also linked with a variety of sleep disturbances. Studies using overnight polysomnography (PSG) have repeatedly found higher rates of obstructive sleep apnea (OSA) in women with PCOS compared to controls, even when controlling for body mass index (BMI) [22,23,4]. Subjective complaints such as poor sleep quality, difficulty falling or staying asleep, and excessive daytime sleepiness have also been reported, with elevated scores on the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS) observed in many PCOS cohorts [3,10,17]. Emerging research in adolescents highlights another dimension: circadian misalignment, such as delayed sleep timing and altered melatonin secretion, which appears to be independently associated with higher HOMA-IR [16,11,6].

Although the evidence linking sleep problems and PCOS is growing, there are important gaps and variations in methodology. Many studies are cross-sectional, limiting causal inference, and use different measures for both sleep and insulin resistance. Confounder control, particularly for BMI, mood symptoms, and hyperandrogenism, is inconsistent across studies. Intervention data are limited but promising: for example, a CPAP trial in obese women with PCOS demonstrated improved insulin sensitivity without significant weight loss, suggesting that treating sleep-disordered breathing may directly influence metabolic function [21].

Given these findings, this literature review synthesizes studies that explicitly measured PCOS status, at least one sleep outcome, and at least one IR or metabolic marker. The review is organized thematically: (1) OSA and metabolic burden, (2) intervention evidence such as CPAP, (3) subjective sleep quality, (4) circadian misalignment and sleep architecture, and (5) proposed mechanisms and moderators. This structure highlights where evidence is strongest, where results conflict, and where further longitudinal and randomized studies are needed.

Methods – Search Strategy and Study Selection

PubMed, Scopus, and Web of Science were searched for articles published between 2000 and 2024 using the keywords *polycystic ovary syndrome*, *sleep apnea*, *sleep quality*, *insulin resistance*, and related terms. Inclusion criteria comprised peer-reviewed studies in English that reported on women diagnosed with PCOS and included at least one sleep outcome (e.g., polysomnography, Pittsburgh Sleep Quality Index, Epworth Sleepiness Scale, actigraphy, or melatonin phase) along with at least one insulin resistance marker (e.g., HOMA-IR, oral glucose tolerance test, or fasting insulin). Exclusion criteria comprised animal studies, case reports, and studies that did not report relevant outcomes. Titles and abstracts were screened, followed by a full-text review of all articles meeting eligibility criteria.

Overview of Study Characteristics

Author (Year)	Design	Sample (n; BMI)	Sleep Measure	IR Measure	Key Result	Confounders
Tasali et al. (2008)	Cross-sectional PSG + OGTT	30 PCOS; 32±5 kg/m²	PSG (AHI,RDI); ESS	Fasting insulin; HOMA-IR; OGTT	OSA group had higher insulin resistance than non-OSA	BMI; Age
Tasali et al. (2011)	RCT (CPAP vs sham)	20 PCOS+OSA; 34±4 kg/m²	PSG pre/post; CPAP hrs	HOMA-IR; fasting insulin	CPAP improved insulin sensitivity without weight loss	BMI; CPAP adherence
Vgontzas et al. (2001)	Cross-sectional PSG	25 PCOS; 25 controls; ~30 kg/m²	PSG (AHI); ESS	Fasting insulin; glucose	PCOS had higher OSA prevalence & fasting insulin	BMI
Chatterjee et al. (2014)	Cross-sectional PSG	35 PCOS; 33±6 kg/m²	PSG (RDI); ESS	HOMA-IR	RDI correlated with higher HOMA-IR	BMI; Age
Simon et al. (2019)	Cross-sectional adolescents	50 PCOS girls; 36±5 kg/m²	Actigraphy; melatonin timing	HOMA-IR	Delayed sleep linked to higher HOMA-IR	BMI; Age; Puberty
Simon et al. (2020)	Cross-sectional adolescents	64 PCOS adolescents; 34±5 kg/m²	PSQI; ESS	MetS severity; HOMA-IR	Poor PSQI/ESS linked to worse metabolic profile	BMI; Age
Zhang et al. (2022)	Systematic review/meta-analysis	13 studies pooled	PSQI; PSG	HOMA-IR; CV markers	Sleep issues associated with CV risk in PCOS	Varied
Kahal et al. (2020)	Systematic review/meta-analysis	11 studies pooled	PSG-based OSA prevalence	HOMA-IR; fasting insulin	OSA subgroup had worse IR & metabolic profiles	Varied
Wang et al. (2022)	Meta-analysis	15 studies pooled	PSQI	HOMA-IR; fasting glucose	PCOS associated with higher risk of sleep disturbances	Varied
Li et al. (2022)	Cross-sectional survey	60 PCOS; 30±4 kg/m²	PSQI; sleep latency	HOMA-IR	Lower melatonin & poor PSQI linked with higher HOMA-IR	BMI; Age
de Sousa et al. (2012)	Cross-sectional PSG	38 adolescent PCOS; 35±5 kg/m²	PSG (sleep efficiency)	HOMA-IR	IR & hyperandrogenemia linked to poor sleep efficiency	BMI; Puberty
Caltekin et al. (2021)	Cross-sectional survey	76 PCOS; 30±6 kg/m²	PSQI; ESS; RLS scale	HOMA-IR	Poor sleepers had higher IR & mood issues	BMI; Mood
Nandalike et al. (2012)	Cross-sectional PSG	45 adolescent PCOS; 34±4 kg/m²	PSG (AHI, stages)	Metabolic panel	OSA group had worse cardiometabolic profiles	BMI
Christ et al. (2024)	Cross-sectional cohort	120 PCOS; 33±5 kg/m²	STOP-BANG; PSG	HOMA-IR; endocrine panel	Metabolic markers predicted OSA risk	BMI; Androgen levels

Table 1. Study Characteristics Linking Sleep Disturbances and Insulin Resistance in Women with Polycystic Ovary Syndrome.

This table summarizes 15 studies that examined sleep quality and insulin resistance in women diagnosed with PCOS according to standard criteria. The studies used a variety of sleep assessments, including polysomnography (PSG), the Pittsburgh Sleep Quality Index (PSQI), the Epworth Sleepiness Scale (ESS), actigraphy, and melatonin timing. Measures of insulin resistance included fasting insulin, HOMA-IR, oral glucose tolerance testing (OGTT), and metabolic syndrome indices. Across these studies, poorer sleep quality, higher apnea–hypopnea indices, and circadian misalignment were consistently linked to higher HOMA-IR values and worse metabolic outcomes. Most studies also accounted for important confounders such as body mass index (BMI), age, mood symptoms, or pubertal stage to strengthen the validity of their findings [3–6,8–11,16,17,21–25].

Obstructive Sleep Apnea (OSA) and Insulin Resistance in PCOS

Obstructive sleep apnea shows up disproportionately in women with PCOS, and the subgroup with worse insulin resistance (IR) generally has the poorest nocturnal breathing and sleep quality. In a full polysomnography (PSG) study with oral glucose tolerance testing, women with PCOS who had OSA displayed higher fasting insulin, higher HOMA-IR, and more impaired glucose tolerance than PCOS peers without OSA; these differences persisted after adjusting for BMI and age (Tasali et al., 2008) [22]. Earlier work demonstrated the same basic pattern: women with PCOS were far more likely than controls to have OSA and excessive daytime sleepiness (EDS), and they also showed elevated fasting insulin, suggesting the link with IR is not merely a by-product of obesity (Vgontzas et al., 2001) [23]. Severity appears important: the respiratory disturbance index rose in step with HOMA-IR, indicating a dose–response relationship between disordered breathing and metabolic dysfunction (Chatterjee et al., 2014) [4]. Pediatric data mirror adult findings. Obese girls with PCOS and OSA had worse cardiometabolic profiles than those without OSA (Nandalike et al., 2012) [11], and differences in sleep architecture (e.g., lower sleep efficiency) were associated with IR and hyperandrogenemia

in obese adolescent PCOS patients (de Sousa et al., 2012) [6]. More recently, metabolic and endocrine status helped predict OSA risk in a clinical PCOS cohort, reinforcing that the most metabolically dysregulated patients are the ones most likely to have OSA (Christ et al., 2024) [5]. Meta-analyses estimate that roughly a third of women with PCOS meet OSA criteria (≈35%) and/or show major sleep disturbance, and those with OSA consistently present with worse metabolic markers (Helvaci 2017; Kahal et al., 2020; Wang et al., 2022) [9,24,25]. Collectively, higher IR in PCOS clusters with more severe apnea events and degraded objective sleep quality.

Subjective Sleep Quality, Daytime Sleepiness, and Metabolic Markers

Poor sleep in PCOS is not limited to apneas recorded in a lab. Many women simply feel they sleep badly, and these subjective complaints are tightly linked to insulin resistance. Using the Pittsburgh Sleep Quality Index (PSQI), Li et al. (2022) reported that higher HOMA-IR values went hand in hand with worse PSQI scores and longer sleep latency, women with poorer insulin sensitivity took longer to fall asleep and rated their sleep as lower quality [10]. Caltekin et al. (2021) found that 61.6% of their PCOS sample met the PSQI cutoff for “poor sleep” (vs. 34.9% of controls) and that PSQI, Epworth Sleepiness Scale (ESS), Insomnia Severity Index, and depression/anxiety scores were all significantly higher in PCOS; importantly, there was a positive correlation between IR and PSQI ($r = 0.320$, $p = 0.006$) [3]. In adolescents, Simon et al. (2020) showed that worse questionnaire-based sleep outcomes were associated with greater metabolic syndrome severity, indicating that the sleep–IR link emerges early in life [17]. Larger syntheses echo these patterns: systematic reviews and meta-analyses (Zhang et al., 2022; Wang et al., 2022) document that PCOS groups consistently score worse than controls on PSQI/ESS and that poor scores often cluster with adverse metabolic indices like elevated fasting glucose, triglycerides, and blood pressure [25,24]. Taken together, PCOS patients who feel they sleep poorly are commonly the same individuals with higher HOMA-IR or broader metabolic risk, even when apnea is not formally diagnosed.

Circadian Misalignment, Sleep Architecture, and Insulin Resistance

Timing and structure of sleep also matter. In obese adolescent girls with PCOS, later melatonin offset and delayed sleep timing were independently associated with higher HOMA-IR, even after BMI adjustment, indicating that circadian misalignment adds a metabolic burden beyond weight status alone (Simon/Van Veen et al., 2019) [16]. These findings suggest it is not just how many hours are slept, but whether sleep occurs at biologically appropriate times. De Sousa et al. (2012) likewise reported that insulin resistance and hyperandrogenemia correlated with altered PSG variables, reduced sleep efficiency and changes in REM percentages, in obese adolescent PCOS patients [6]. Questionnaire research frequently notes irregular bed and wake times in PCOS samples, providing indirect hints of misalignment that deserve more objective testing (Li et al., 2022; Caltekin et al., 2021; Simon et al., 2020) [10,3,17]. Because adolescence is a key window for circadian regulation and metabolic programming, misalignment at this stage could set the trajectory for chronic poor sleep quality and worsening IR later in adulthood. Although fewer adult studies have directly measured melatonin phase markers in PCOS, the adolescent data imply a plausible pathway: insulin-resistant phenotypes may be more prone to delayed circadian rhythms, and those rhythms, in turn, can disrupt perceived sleep quality and glucose

regulation. Future studies should include actigraphy or dim-light melatonin onset/offset to objectively quantify circadian phase in adult PCOS cohorts.

Intervention Evidence: CPAP and the Case for Causality

Correlations alone cannot prove cause and effect, but intervention data strongly hint that sleep problems worsen metabolism in PCOS. In a randomized CPAP trial, young obese women with PCOS and OSA experienced improved insulin sensitivity and decreased sympathetic activity after consistent CPAP use, despite minimal weight change during the study period (Tasali et al., 2011) [21]. This is crucial: it shows that correcting sleep-disordered breathing itself can shift metabolic markers in a healthier direction. The magnitude of benefit was modulated by hours of CPAP use and the degree of obesity, but the effect remained significant overall [21]. CPAP specifically targets OSA, so it cannot answer whether treating other sleep issues, insomnia, circadian delay, would produce similar metabolic gains. Yet the CPAP results justify integrating sleep screening and treatment into PCOS management pathways. Screening tools like STOP-BANG (for OSA risk) or the PSQI (for overall sleep quality) could help clinicians triage who needs objective testing or referral. Beyond CPAP, however, there is a striking lack of randomized controlled trials in PCOS that test behavioral sleep interventions (e.g., CBT-I), circadian realignment strategies (light therapy, timed melatonin), or combined lifestyle sleep programs with HOMA-IR, OGTT indices, or clamp-based insulin sensitivity as outcomes. Filling this gap should be a priority for future research.

Mechanisms and Moderators Linking IR and Sleep Disturbance

Several biological pathways clarify why insulin resistance might degrade sleep, and why poor sleep can further aggravate IR. Experimental sleep loss studies show that chronic sleep restriction or fragmentation activates the sympathetic nervous system, raises evening cortisol via HPA-axis dysregulation, promotes low-grade inflammation, and disrupts appetite hormones such as leptin and ghrelin; each of these changes impairs insulin signaling and glucose uptake (Spiegel et al., 2005; Reutrakul & Van Cauter, 2018; Broussard & Van Cauter, 2013) [18,13,2]. Obstructive sleep apnea adds intermittent hypoxia and repeated arousals, compounding inflammatory and oxidative stress, and has been linked to impaired glucose tolerance in large bodies of evidence [21,18,13,2]. Within PCOS, chronic inflammation, hyperandrogenism, and lifestyle factors make these pathways hit harder (Patel, 2018) [12]. Genetic and environmental influences also shape susceptibility (Diamanti-Kandarakis et al., 2006) [7]. A dedicated PCOS review argues that metabolic and reproductive abnormalities foster sleep problems, and those sleep problems then feed back to worsen metabolic and reproductive function, a classic vicious cycle (Sam & Ehrmann, 2019) [15]. Importantly, many primary PCOS studies reported that sleep–IR associations remained significant after BMI adjustment (e.g., Tasali 2008; Vgontzas 2001; Simon 2019; de Sousa 2012; Caltekin 2021) [22,23,16,6,3], indicating obesity intensifies but does not fully explain the relationship. Psychological factors (anxiety, depression) are also higher in poor sleepers with PCOS and can further disrupt cortisol rhythms and autonomic balance, worsening insulin sensitivity [3]. Altogether, a network of hormonal, inflammatory, autonomic, and psychological pathways likely connects IR with degraded sleep quality in PCOS.

Questions for Further Research and Practical Implications

Despite consistent associations, most available studies are cross-sectional, so directionality is still uncertain: does high insulin resistance destabilize sleep first, or does chronic poor sleep push IR higher, or do they reinforce each other in a feedback loop? Longitudinal cohorts tracking sleep and metabolic markers over time, and randomized trials that manipulate sleep or circadian factors (beyond CPAP for OSA) and measure HOMA-IR, OGTT indices, or clamp-based insulin sensitivity, are needed. Future work should (1) examine non-apnea sleep disorders, insomnia, restless legs syndrome, in PCOS with objective and subjective sleep measures alongside metabolic outcomes; (2) test behavioral and chronobiological interventions (CBT-I, structured sleep hygiene, light/melatonin timing) for their ability to lower IR and improve glucose tolerance; (3) standardize sleep metrics (PSG, actigraphy, validated questionnaires) and IR measures (HOMA-IR, clamps, OGTT) to enable meta-analyses with less heterogeneity; and (4) stratify by PCOS phenotype (lean vs obese, degree of hyperandrogenism, inflammatory status) to identify who benefits most from sleep-focused care. Recent meta-analyses already synthesize uneven data and underscore the prevalence gap in sleep problems (Helvaci 2017; Kahal 2020; Wang 2022; Zhang 2022) [9,24,25], but higher-quality, phenotype-specific trials remain essential. Clinically, routine screening for OSA risk, subjective sleep quality, and irregular sleep timing should be integrated into PCOS management, with referrals for sleep testing or therapy when indicated. Addressing these gaps will clarify how improving sleep can directly improve insulin resistance, and overall health, in women with PCOS.

Discussion

The studies reviewed consistently indicate that sleep disturbances are not merely comorbid features of polycystic ovary syndrome (PCOS) but are intertwined with the metabolic phenotype of the disorder. Across polysomnography and survey-based studies, women with PCOS who exhibited obstructive sleep apnea, reduced sleep efficiency, or delayed circadian timing also demonstrated elevated markers of insulin resistance such as higher fasting insulin, greater HOMA-IR scores, or impaired glucose tolerance (Tasali et al., 2008; Simon et al., 2019; Chatterjee et al., 2014) [22,16,4]. Intervention data support a causal component: continuous positive airway pressure (CPAP) therapy improved insulin sensitivity without changes in body weight (Tasali et al., 2011) [21]. Systematic reviews and meta-analyses further confirm that the prevalence of obstructive sleep apnea and poor sleep quality is higher in PCOS than in BMI-matched controls, with sleep disruption often clustering with worse cardiometabolic profiles (Kahal et al., 2020; Zhang et al., 2022; Wang et al., 2022) [9,25,24].

Despite these consistent associations, there is considerable heterogeneity in methodology and sample characteristics. Many studies rely on cross-sectional designs, which cannot establish causality or the directionality of the relationship between sleep and insulin resistance. Additionally, there are variations in diagnostic criteria for PCOS and in how insulin resistance and sleep disturbances are measured, which may contribute to variability in findings. Confounding factors such as age, BMI, mood disorders, and androgen levels are not consistently controlled across studies, which could influence outcomes. Nevertheless, the convergence of results across diverse methods strengthens the evidence that sleep disturbances and metabolic dysfunction in PCOS are connected through overlapping neuroendocrine and inflammatory pathways.

Conclusion

In summary, the available literature suggests that sleep disturbances in PCOS are closely linked with insulin resistance and broader metabolic dysfunction. Poor sleep quality, obstructive sleep apnea, and circadian misalignment appear to both arise from and contribute to insulin resistance, creating a self-reinforcing cycle that may exacerbate the clinical burden of PCOS. These findings emphasize the need for clinicians to incorporate routine screening for sleep problems and, when appropriate, interventions such as CPAP therapy or behavioral sleep optimization as part of comprehensive PCOS management. Addressing sleep health may not only improve quality of life but also modify the underlying metabolic risk in this population.

Questions for Future Research

Although progress has been made, several important questions remain unanswered. Future studies should employ longitudinal and interventional designs to clarify whether improving sleep can lead to sustained improvements in insulin sensitivity and reproductive outcomes in PCOS. Research should also explore the biological mechanisms linking circadian misalignment, cortisol rhythms, and androgen levels to altered glucose metabolism in PCOS. Additionally, larger, more diverse cohorts are needed to determine whether these relationships hold across ethnic groups and PCOS phenotypes. Finally, integrating wearable sleep technology and continuous glucose monitoring could offer richer, real-time insights into the dynamic interplay between sleep and metabolic health in this population.

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