

REVIEW

Sleep disorders and male sexual dysfunction: A meta-analysis of hormonal and erectile outcomes

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Summary

Background: Sleep disruptions, including insomnia and obstructive sleep apnea

(OSA), are strongly linked to male sexual dysfunction, affecting libido, erectile function, and hormonal balance. This meta-analysis aimed to evaluate the relationship between sleep disorders and male sexual function.

Methods: A systematic search of PubMed, Scopus, Web of Science, and ScienceDirect through September 2025 identified 9 peer-reviewed studies examining the impact of sleep disorders on male sexual health. The Newcastle-Ottawa Scale was used to assess the risk of bias. A meta-analysis was performed to evaluate outcomes, including the International Index of Erectile Function (IIEF), testosterone, follicle-stimulating hormone (FSH), and luteinizing hormone (LH) levels, with a subgroup analysis comparing OSA and shift work sleep disorder (SWSD). Statistical analyses were conducted using R version 4.2.2.

Results: Across seven studies (n = 913), the pooled mean IIEF score was approximately 16, indicating moderate erectile dysfunction, though heterogeneity was high (I² = 97.3%). Subgroup analysis revealed lower IIEF scores in OSA patients (≈14.4) compared to SWSD patients (≈17.5), suggesting more severe dysfunction in OSA. Hormonal analysis (n = 325) showed significantly lower testosterone levels in OSA (≈7-9 nmol/L) versus SWSD (≈20 nmol/L). FSH and LH were elevated in OSA (≈3.8-5.8 IU/L and 3.3-3.9 IU/L, respectively) compared to SWSD (≈2.2 IU/L and 1.8 IU/L). High heterogeneity indicated substantial variation in study design and populations.

Conclusions: Both OSA and SWSD impair sexual and hormonal function, but OSA causes greater erectile and endocrine disruption. Even mild OSA was associated with reduced erectile performance, with the severity worsening dysfunction. Despite consistent trends, high heterogeneity warrants cautious interpretation. Future research should employ standardized tools and uniform populations to clarify mechanisms linking sleep disorders to male sexual health.

KEY WORDS: Obstructive sleep apnea; Erectile dysfunction; Insomnia; Intermittent hypoxia; Men's sexual health; Sleep apnea.

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INTRODUCTION

Sleep has long been recognized as a fundamental pillar of overall health and well-being. Over the past several decades, extensive research has consistently demonstrated that insufficient or poor-quality sleep is strongly associated with adverse health outcomes and an increased disease burden (1).

As men progress into midlife and older age, they often encounter noticeable changes in sexual health, many of which can manifest as dysfunction. These changes are frequently linked to a decline in sex hormone levels, particularly testosterone. Reduced testosterone is associated with a variety of complications, including *erectile dysfunction* (ED), diminished sexual desire, loss of body and pubic hair, and impaired orgasmic or ejaculatory function, among others (2). Among these, ED is defined as the persistent inability to achieve and maintain an erection sufficient for satisfactory sexual performance (3). It is the most common and distressing condition, often prompting men to pursue medical care. The prevalence of ED is considerable, with studies showing that more than half (52%) of men between the ages of 40 and 70 experience the condition (4). While ED is commonly attributed to the natural process of aging, it may also develop as a consequence of comorbid medical conditions, side effects of treatments, or psychological factors such as anxiety, depression, and reduced self-esteem (5). Regardless of its underlying cause, ED can significantly diminish overall quality of life. The coexistence of *comorbid insomnia and sleep apnea* (COMISA) with ED represents a significant health concern, with each condition amplifying the impact of the other. Disruptions in sleep architecture and intermittent hypoxia characteristic of COMISA contribute to hormonal dysregulation, endothelial dysfunction, autonomic imbalance, and oxidative stress, all of which play central roles in the development and progression of ED. Together, these interconnected mechanisms highlight the strong physiological link between sleep disturbances and sexual health outcomes in men (6).

Adequate sleep, by contrast, is a cornerstone of health and well-being (7). In recent years, both medical profes-

sionals and the public have paid increasing attention to sleep medicine and the impact of sleep disorders. This growing interest stems in part from accumulating evidence that inadequate or disrupted sleep can negatively influence multiple aspects of human health and, if left untreated, may contribute to serious illnesses.

Historically, sleep problems were not considered as potential contributors to ED. However, emerging research has demonstrated that sleep disturbances are common among individuals with ED, and the connection between sleep disruption, sleep disorders, and ED is now an area of expanding investigation (8). This meta-analysis aims to evaluate the impact of sleep disorders, including insomnia, OSA, on male sexual health, with particular focus on ED, libido, and other related outcomes, to clarify the strength and nature of these associations.

METHODS

This systematic review adheres to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (9). The Prospero registration number was given to the protocol of this systematic review (CRD420251154081).

Eligibility criteria

The study selection was guided by the PICOS framework (Table 1).

Case reports, reviews, posters, unpublished work, review articles, editorials, or pediatric or female-only population studies were excluded. Additionally, animal or in vitro studies lacking relevant sexual health outcomes were not considered. Regarding report characteristics, there were no limits on the year of dissemination; however, studies needed to be written in or translated in English.

Definition of key concepts

The IIEF is a validated multidimensional questionnaire assessing male sexual function across domains such as erectile function, orgasmic function, sexual desire, intercourse satisfaction, and overall satisfaction (10). Lower IIEF scores indicate greater erectile dysfunction severity. Testosterone levels (measured in nanomoles per liter, nmol/L) reflect androgenic hormonal activity, which is crucial for libido and erectile function (11). FSH and LH (measured in IU/L) are key gonadotropins regulating testicular function; FSH stimulates spermatogenesis, while LH promotes testosterone synthesis (12). Collectively, these biomarkers and functional scores provide compre-

hensive insight into male sexual health and endocrine balance in sleep-related disorders.

Search strategy for the identification of studies

A comprehensive search strategy was employed across major electronic databases, including *PubMed*, *Scopus*, *Web of Science*, and *ScienceDirect*, up to September 2025. The search strategy incorporated both *Medical Subject Headings* (MeSH) and free-text terms, such as “sleep disorders,” “insomnia,” “sleep apnea,” “obstructive sleep apnea,” “COMISA,” combined with “male sexual health,” “sexual dysfunction,” “erectile dysfunction,” “libido,” and “testosterone.” Reference lists of relevant articles were also manually screened to identify additional studies.

Screening process and data extraction

We utilized EndNote as a reference manager to screen the articles and remove duplicates. We applied the eligibility criteria to the screening criteria in the title and abstract. Based on the criteria, two reviewers independently screened the titles and abstracts of the articles to see if they were eligible for full-text review. Manuscripts chosen for further assessment were retrieved and read fully.

One reviewer then conducted the full-text review, and the second reviewer and the senior double-checked the full-text review to ensure its validity. We collected detailed information on the characteristics of study participants, including the type of sleep disorder, duration of disorder, comorbidities, and reported sexual outcomes. We also investigated the methodology, such as the country, study design, and sample size. Additionally, we examined information about the interventions, including a description of sleep disorders and their duration. The outcomes evaluated included erectile dysfunction, libido, testosterone levels, and overall sexual satisfaction (case vs. control group).

Data synthesis and statistical analysis

Statistical analyses were conducted using R software 4.2.2. Binary variables were analyzed using *risk ratios* (RR) with 95% *confidence intervals* (CI) and a significance level of $p = 0.05$, while continuous outcomes were analyzed using *standardized mean differences* (SMD) with 95% CI. We evaluated heterogeneity using a Forest plot and I-square (I^2) test and interpreted the I^2 as 0%-40%: might not be important; 30%-60%: moderate heterogeneity; 50%-90%: substantial heterogeneity; and 75%-100%: considerable heterogeneity (13).

Due to variability in reporting across studies, most data

Table 1.
PICOS framework.

Population (P)	Adult males (≥ 18 years) diagnosed with sleep disorders, including OSA, insomnia, and COMISA.
Intervention/Exposure (I)	Presence of sleep disorders diagnosed using polysomnography or validated clinical diagnostic criteria.
Comparison (C)	Healthy controls or subgroups stratified by disease severity where applicable.
Outcomes (O)	Primary outcomes included erectile dysfunction assessed using the International IIEF. Secondary outcomes included sexual desire, libido, testosterone levels, and other reproductive hormones.
Study design (S)	Observational studies (cross-sectional, case-control, cohort) and interventional studies or clinical trials. Case reports, reviews, editorials, and non-human studies were excluded.

OSA: obstructive sleep apnea; COMISA: comorbid insomnia and sleep apnea; IIEF: International Index of Erectile Function.

were grouped according to OSA severity categories (mild, moderate, severe). Therefore, pooled analyses were performed within severity strata rather than a single aggregated OSA versus control comparison.

Assessment of Risk of Bias in included studies

The *Newcastle-Ottawa Criteria scale* (NOS) evaluates non-randomized studies in three domains: selection, comparability, and outcomes for cohort studies and exposure for case-control studies (14). Each item on the scale is scored from one to five stars, except for the comparability item. The maximum star rating for each study is nine, with studies scoring less than five points being identified as representing a high risk of bias.

Assessment of reporting biases

Publication bias was planned to be assessed using funnel plots and Egger's regression test if at least ten studies were available for a given outcome. However, when fewer than ten studies contributed data, these methods were not performed due to limited power and the risk of misleading results.

RESULTS

Studies selection

We executed the search methodologies outlined previously, resulting in the identification of a total of 611 citations, subsequently reduced to 476 following the removal of duplicates. Upon screening titles and abstracts, only 39

citations met the eligibility criteria for further consideration. Through full-text screening, this number was further refined to 9 articles (15-23), aligning with our inclusion and exclusion criteria. Figure 1 provides an in-depth depiction of the search strategy and screening process.

The demographic characteristics of the included studies

A total of nine studies, published between 2016 and 2025, were included in this systematic review and meta-analysis, representing diverse geographic regions, including Egypt, the USA, Korea, China, Spain, and Turkey. Study designs comprised retrospective cohorts ($n = 3$), prospective cohorts ($n = 2$), and cross-sectional studies ($n = 4$). Across all studies, populations included men with different types of sleep disorders such as obstructive sleep apnea (OSA), insomnia, circadian rhythm dysfunction, and shift work sleep disorder. Sample sizes varied substantially, ranging from small single-center cohorts of fewer than 50 patients to large database analyses encompassing over 4 million individuals. The mean age of participants generally fell within the 40-70-year range. Baseline characteristics of the included studies are summarized in Table 2.

Association between sleep disorders and comorbidities across multiple studies

Across the included studies, comorbidities were found to be strongly associated with OSAS and related conditions. Youssef (2025) (15) reported a significant relationship between diabetes mellitus and OSAS severity ($p < 0.001$), with the highest prevalence in mild OSAS patients, while cerebrovascular accidents were notably more frequent in

Figure 1.
PRISMA flow chart.

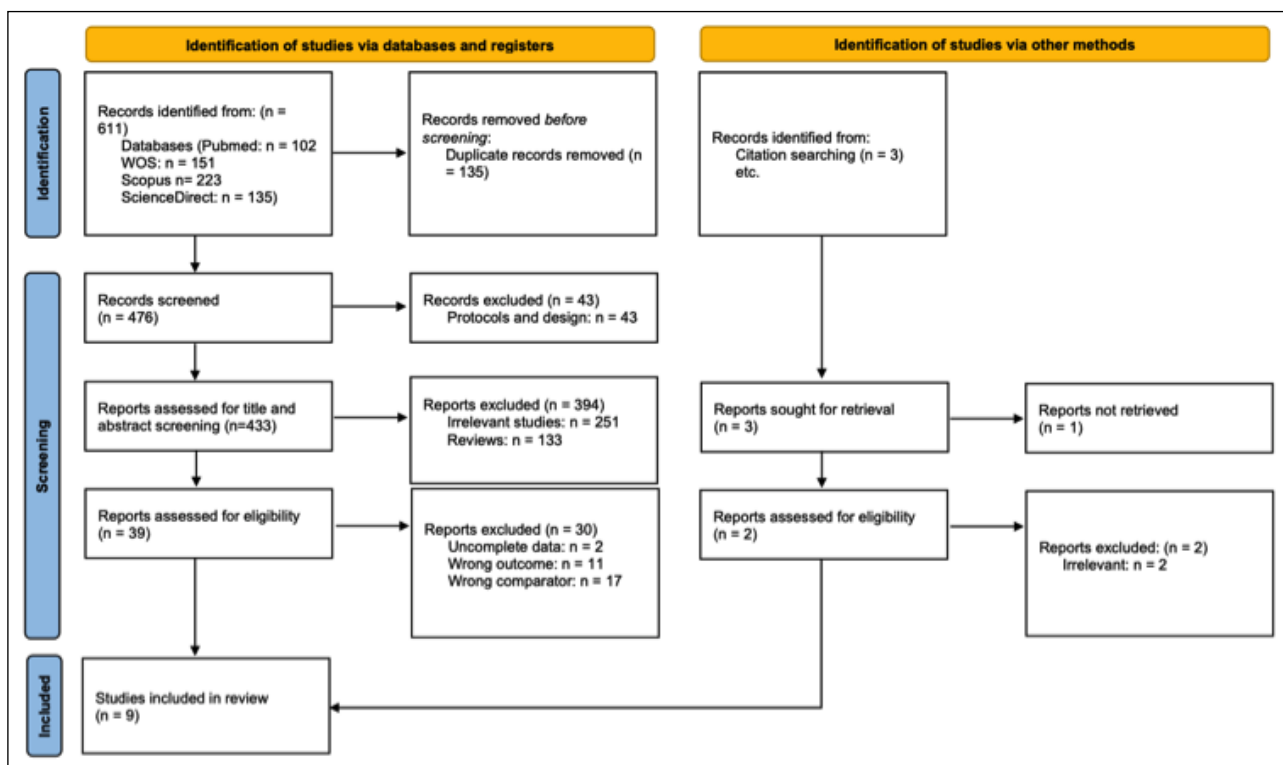


Table 2.
Baseline characteristics of the included studies.

Study ID	Country	Design	Population	Duration	Sample size		Age, mean (SD)		Outcome
					Sleep disorder	Control	Sleep disorder	Control	
Youssef 2025 (15)	Egypt	Cross-sectional	Obstructive sleep apnea	NR	130	50	46.5 (7.9)	45.14 (9.24)	Erectile dysfunction and testosterone level
Agrawal 2024 (16)	USA	Retrospective cohort	Sleep apnea, insomnia, and circadian rhythm dysfunction	5 years	4198743	4198743	40-70		Erectile dysfunction and testosterone level
Belladelli 2024 (17)	USA	Retrospective cohort	Insomnia	2007-2016	182534	182534	47.9 (17.1)		Erectile dysfunction
Kim 2024 (18)	Korea	Retrospective cohort	Obstructive sleep apnea	3 months	129	NA	50.17 (11.84)	NA	Erectile dysfunction
Zheng 2024 (19)	China	Cross-sectional	Shift work sleep disorder	6 months	148	251	18-60		Hormones associated with premature ejaculation
Rodriguez 2021 (20)	USA	Cross-sectional	Shift work sleep disorder	January 2014 - July 2017	46	156	38.7 (2.9)	37.8 (2.5)	Erectile dysfunction
Pascual 2018 (21)	Spain	Cross-sectional	Obstructive sleep apnea	April 1st 2013 and July 31th 2016	150	NA	51.8 (1.05)	NA	Erectile dysfunction
Li 2016 (22)	China	Prospective cohort	Obstructive sleep apnea	January 2013 to August 2015	153	60	46.5 (8.4)	47.7 (9.5)	Erectile dysfunction
Irer 2018 (23)	Turkey	Prospective cohort	Obstructive sleep apnea	October 2017 and March 2018	104	22	42.13 (6.7)	38.3 (7.6)	Sexual dysfunction and quality of life improvements

NA: not applicable; NR: not reported; SD: standard deviation.

severe OSAS ($p = 0.039$). Other comorbidities, such as hypertension, dyslipidemia, and ischemic heart disease, showed no significant differences among groups. Belladelli (2024) (17) highlighted high rates of hypertension (40.8%), hyperlipidemia (45.5%), and diabetes (14.7%) among patients, indicating the widespread burden of metabolic and cardiovascular comorbidities. Similarly, Zheng (2024) (19) found that shift workers had significantly higher rates of hypertension, diabetes, and hyperlipidemia compared with non-shift workers (all $p < 0.01$). Finally, Irer (2018) (23) demonstrated that the prevalence of comorbidities increased markedly with OSAS severity, from 9.1% in the normal group to 51.9% in the severe group ($p < 0.001$), reinforcing the strong link between OSAS severity and the presence of multiple health conditions (Table 3).

Sleep disorders and their impact on ef and hormonal levels

The studies in Table 4 collectively highlight the association between different sleep disorders and varying degrees of ED, as measured by the IIEF.

For patients with OSAS, scores generally fall in the mild-to-moderate erectile dysfunction range. Li (2016) (22) reported a mean IIEF-5 score of 15.2, while Kim (2024) (18) showed a nearly identical score of 14.97, both suggesting moderate impairment. Pascual (2018) (21) and Irer (2018) (23), who used the IIEF-15 scale, reported mean scores of 15.2 and 17.0, respectively, again consistent with ED, though variability was large in Pascual's study. The lowest score was observed in Youssef (2025) (15), with an IIEF-5 mean of 10.3, indicating more severe dysfunction compared to other OSAS cohorts.

In contrast, patients with SWSD tend to have better pre-

served EF. Zheng (2024) (19) reported a mean IIEF-5 score of 17.3, which indicates only mild dysfunction. Rodriguez (2020) (20) found a mean score of 21.6 using the full IIEF, which is close to normal erectile function, although the wide standard deviation suggests some patients still experienced significant issues.

Overall, the evidence suggests that OSAS is more consistently linked to reduced erectile function, with scores clustering in the moderate dysfunction range and sometimes worse. By comparison, SWSD appears to have a milder impact, with many patients maintaining near-normal function. This contrast reinforces the idea that OSAS, through mechanisms like intermittent hypoxia, sleep fragmentation, and associated comorbidities, exerts a stronger negative effect on sexual health than circadian disruption alone. In patients with OSAS, testosterone levels appear consistently lower, often within the borderline or mildly reduced range. Li (2016) (22) reported a mean of 5.4 nmol/L. Youssef (2025) (15) found a mean of 9.15 nmol/L, still at the lower end of the normal range, whereas Pascual (2018) (21) reported a somewhat higher mean of 11.5 nmol/L, which falls within the normal range but may still reflect impaired testosterone compared to healthy populations. These findings suggest that OSAS is frequently associated with reduced testosterone.

In contrast, patients with SWSD had much higher average testosterone levels. Rodriguez (2020) (20) reported a mean of 20.45 nmol/L, which is within the upper normal range. Still, the very large standard deviation (19.50) indicates wide variability, as some patients had very high levels, while others had much lower levels (Table 5).

Although inconsistent, the findings in OSAS generally suggest altered gonadal function. Li (2016) (22) reported a rel-

Table 3.

Association Between Sleep Disorders and Comorbidities Across Multiple Studies.

Study ID	Comorbidities
Youssef 2025 (15)	Diabetes Mellitus: Strongly associated with OSAS severity ($p < 0.001$). 100% of patients with mild OSAS had diabetes, compared with 24.4% in moderate, 35.5% in severe OSAS, and 20% in controls. It indicates a significant clustering of diabetes in OSA patients, particularly in the mild group. Hypertension: No significant difference among groups ($p = 0.924$). Prevalence remained similar: ~18-22% across all OSAS and control groups. Dyslipidemia: No statistically significant difference ($p = 0.183$). Slightly higher in severe OSAS (32.3%) compared with other groups (~17-18%). Ischemic Heart Disease: Not significantly different among groups ($p = 0.142$). Slight increase in severe OSAS (19.4%) compared with controls (8%). Cerebrovascular Accidents: Significant association with OSAS severity ($p = 0.039$). Cerebrovascular accidents more common in severe OSAS (24.2%) compared with controls (6%).
Belladelli 2024 (17)	Diabetes, n (%): 26,903 (14.74) Obesity, n (%): 18,148 (9.94) Hypertension, n (%): 74,439 (40.78) Coronary Artery Disease, n (%): 19,617 (10.75) Hyperlipidemia, n (%): 83,062 (45.50) Sleep Apnea, n (%): 9,484 (5.20) Anxiety, n (%): 23,485 (12.87)
Zheng 2024 (19)	Hypertension: Present in 16.54% of shift workers, significantly higher than 11.07% in non-shift workers ($p = 0.007$). Diabetes: Markedly higher among shift workers (15.04%) compared to non-shift workers (4.64%) with a highly significant difference ($p < 0.001$). Hyperlipidemia: Reported in 12.03% of shift workers versus 7.14% of non-shift workers ($p = 0.004$).
Irer 2018 (23)	The prevalence of comorbidities increased progressively with the severity of OSAS. Only 9.1% of individuals in the normal group had comorbidities, compared to 14.8% in the mild, 20.0% in the moderate, and 51.9% in the severe OSAS group. This shows a strong association between OSAS severity and the presence of comorbid conditions ($p < 0.001$), indicating that patients with more severe OSAS are significantly more likely to have additional health problems.

OSAS: Obstructive sleep apnea syndrome; SD: standard deviation.

Study	Scale	Population	Number of participants	Mean (SD)
Li 2016 (22)	IIEF-5	Obstructive sleep apnea	72	15.2 (3.9)
Pascual 2018 (21)	IIEF-15	Obstructive sleep apnea	77	15.2 (12.1)
Zheng 2024 (19)	IIEF-5	Shift work sleep disorder	399	17.3 (4.5)
Rodríguez 2020 (20)	IIEF	Shift work sleep disorder	46	21.6 (7.7)
Kim 2024 (18)	IIEF	Obstructive sleep apnea	63	14.97 (4.81)
Youssef 2025 (15)	IIEF-5	Obstructive sleep apnea	130	10.3 (5.2)
Irer 2018 (23)	IIEF-15	Obstructive sleep apnea	126	17.0 (4.9)

Table 4.

The IIEF scores of the patients.

Study	Population	Number of participants	Mean (SD)
Li 2016 (22)	Obstructive sleep apnea	72	5.4 (1.9)
Pascual 2018 (21)	Obstructive sleep apnea	77	11.5 (4.0)
Youssef 2025 (15)	Obstructive sleep apnea	130	9.15 (4.65)
Rodríguez 2020 (20)	Shift work sleep disorder	46	20.45 (19.50)

Table 5.

Testosterone (nmol/L) level of patients.

Study	Population	Number of participants	Mean (SD)
Li 2016 (22)	Obstructive sleep apnea	72	9.1 (2.0)
Pascual 2018 (21)	Obstructive sleep apnea	77	5.9 (3.3)
Youssef 2025 (15)	Obstructive sleep apnea	130	2.49 (1.26)
Rodríguez 2020 (20)	Shift work sleep disorder	46	2.24 (2.83)

Table 6.

FSH (IU/L) level of patients.

atively high mean FSH of 9.1 IU/L. Pascual (2018) (21) found a lower mean of 5.9 IU/L, still within the normal range but lower than Li's findings. Youssef (2025) (15), however, reported a much lower mean value of 2.49 IU/L. For SWSD, Rodríguez (2020) (20) observed an average FSH

of 2.24 IU/L, which is similarly low and close to the level reported by Youssef (2025) (15) in OSAS patients. The large standard deviation (2.83) indicates considerable variability, with some individuals showing normal levels and others having markedly reduced values (Table 6).

Study	Population	Number of participants	Mean (SD)
Li 2016 (22)	Obstructive sleep apnea	72	5.5 (2.2)
Pascual 2018 (21)	Obstructive sleep apnea	77	3.6 (1.7)
Youssef 2025 (15)	Obstructive sleep apnea	130	2.62 (1.52)
Rodriguez 2020 (20)	Shift work sleep disorder	46	1.82 (2.37)

Table 7.
LH (IU/L) level of patients.

Table 8.
Quality assessment of the included studies using NOS.

Study ID	Selection (max 4)	Comparability (max 2)	Outcome (max 3)	Total (max 9)
Youssef 2025 (15)	☆☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆☆
Agrawal 2024 (16)	☆☆☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆☆
Belladelli 2024 (17)	☆☆☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆☆
Kim 2024 (18)	☆☆☆	-	☆☆☆	☆☆☆☆☆
Zheng 2024 (19)	☆☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆☆
Rodriguez 2021 (20)	☆☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆☆
Pascual 2018 (21)	☆☆☆	-	☆☆☆	☆☆☆☆☆
Li 2016 (22)	☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆
İrer 2018 (23)	☆☆☆☆	☆☆	☆☆☆	☆☆☆☆☆☆☆☆

☆: NOS assess studies based on three domains: Selection (maximum 4 stars), Comparability (maximum 2 stars), and Outcome (maximum 3 stars). A maximum of nine stars indicates the highest methodological quality. Studies with 7-9 stars were considered high quality, 4-6 stars moderate quality, and 0-3 stars low quality.

Among OSAS patients, LH levels are generally reduced or borderline. Li (2016) reported the highest mean at 5.5 IU/L, which is within the normal range but could reflect increased pituitary drive in response to impaired testosterone production. Pascual (2018) (21) observed a lower mean of 3.6 IU/L, while Youssef (2025) (15) found an even lower mean of 2.62 IU/L, suggesting suppressed gonadotropin release in many patients. This progressive decline across studies indicates that OSAS is often associated with blunted LH secretion.

For SWSD, Rodriguez (2020) (20) reported a mean LH of 1.82 IU/L, which is lower than most OSAS reports, though the very wide standard deviation (2.37) suggests substantial variability across individuals. This may reflect circadian misalignment affecting gonadotropin rhythms, with some patients experiencing significant suppression (Table 7).

Risk of bias assessment

Quality evaluation using the NOS indicated that seven of the included studies achieved high-quality scores (≥ 7 points), while the remaining two (18, 21) were of moderate quality (Table 8).

The IIEF scores

The meta-analysis of seven studies, including 913 patients, demonstrated a pooled mean IIEF score of 16.0 (95% CI: 15.7-16.3) under the common effect model and 15.9 (95% CI: 12.8-19.0) under the random effects model. The very high heterogeneity ($Q = 225.32$, $df = 6$, $p < 0.0001$; $I^2 = 97.3\%$, 95% CI: 96.0-98.2%; $\tau^2 = 10.67$, 95% CI: 4.10-54.93; $\tau = 3.27$, 95% CI: 2.02-7.41) indicates that almost all of the observed variability is due to real differences between studies rather than chance. The pooled results suggest that patients with OSA or SWSD

generally have moderately reduced IIEF scores, reflecting impaired erectile function. However, the substantial heterogeneity, likely arising from the use of different IIEF scales (IIEF-5, IIEF-15, or full IIEF), different populations studied (OSA vs. SWSD), and varying study designs and sample sizes, limits the interpretability of the overall pooled estimate. Thus, while the meta-analysis highlights a consistent trend toward sexual dysfunction in these populations, the findings should be interpreted with caution, and subgroup analyses stratified by scale or condition may provide more precise insights (Figure 2).

Subgrouping of the IIEF scores

The meta-analysis of IIEF scores across seven studies with 913 participants revealed notable differences in erectile function between OSA and SWSD populations.

Subgroup analysis highlighted differences between populations. In the common-effect model, OSA patients (five studies) had a pooled mean IIEF of 14.38 (95% CI: 13.92-14.84), whereas SWSD patients (two studies) had a higher pooled mean of 17.46 (95% CI: 17.03-17.90). Heterogeneity remained high within both subgroups (OSA $I^2 = 96.7\%$, SWSD $I^2 = 92.8\%$). The test for subgroup differences was highly significant ($Q = 90.98$, $p < 0.0001$), confirming that SWSD patients tend to have higher IIEF scores than those with OSA.

Under the random-effects model, OSA patients had a mean of 14.50 (95% CI: 11.33-17.68), and SWSD patients had a mean of 19.31 (95% CI: -7.95 to 46.56), with wide confidence intervals reflecting substantial uncertainty and variability. The test for subgroup differences remained significant ($Q = 3.90$, $p = 0.0483$).

Overall, these findings indicate that erectile function, as measured by IIEF scores, is lower in OSA patients compared to SWSD patients (Figure 3).

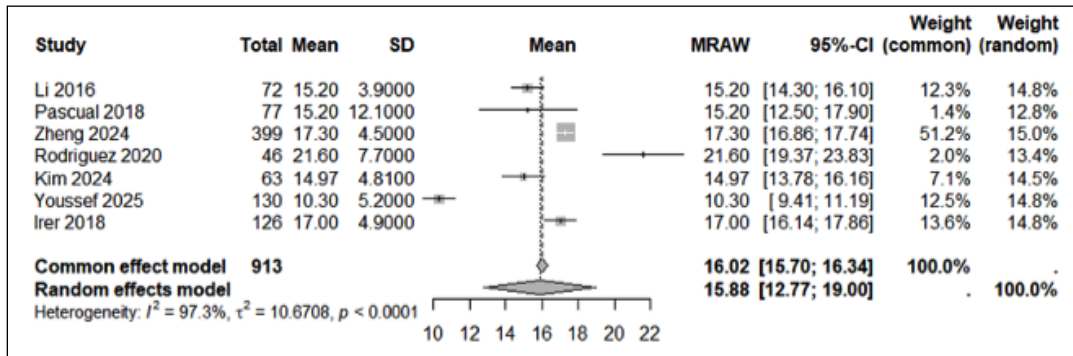


Figure 2.
Forest plot of the IIEF scores.

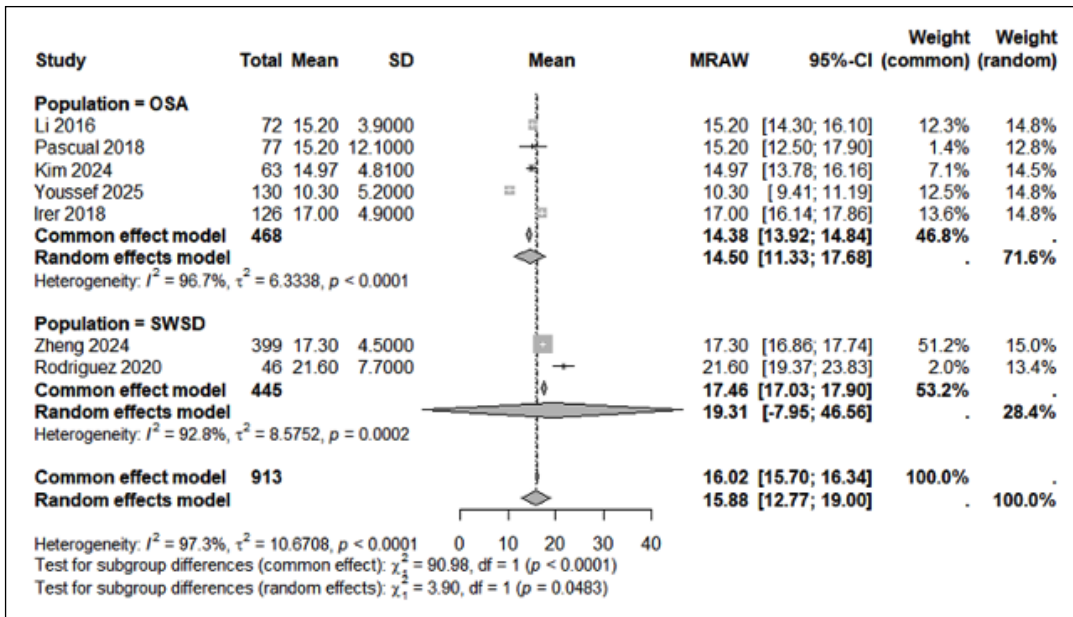


Figure 3.
Forest plot of subgrouping of IIEF score.

Testosterone (nmol/L) level

The meta-analysis of four studies with 325 patients reported a pooled testosterone level of 7.14 nmol/L (95% CI: 6.79-7.49) under the common effect model and 11.16 nmol/L (95% CI: 1.43-20.89) under the random effects model. The difference between the two models reflects the very high degree of heterogeneity ($Q = 197.59$, $df = 3$, $p < 0.0001$; $I^2 = 98.5\%$, 95% CI: 97.6-99.0%; $\tau^2 = 32.52$, 95% CI: 8.39->325.22; $\tau = 5.70$, 95% CI: 2.90->18.03). These values indicate that nearly all of the observed variability between studies is due to real differences rather than chance. The extremely wide confi-

dence interval of the random effects model highlights the instability of the pooled estimate and suggests substantial inconsistency between studies. This heterogeneity is likely driven by the inclusion of different populations (obstructive sleep apnea *versus* shift work sleep disorder) and the very large standard deviation reported in Rodriguez 2020 (20.45 ± 19.50 nmol/L) (Figure 4).

Subgrouping analysis of the testosterone (nmol/L) level

When stratified by population, patients with OSA (3 studies) had a pooled testosterone mean of 7.09 nmol/L (95% CI: 6.73-7.44; $I^2 = 98.9\%$) under the common

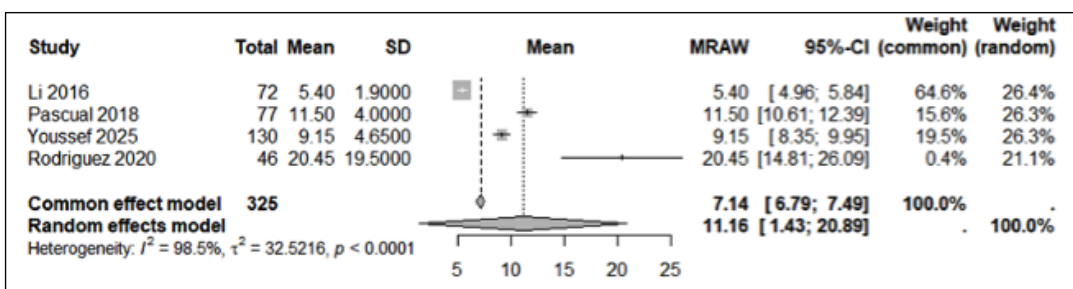


Figure 4.
Forest plot of testosterone (nmol/L) level.

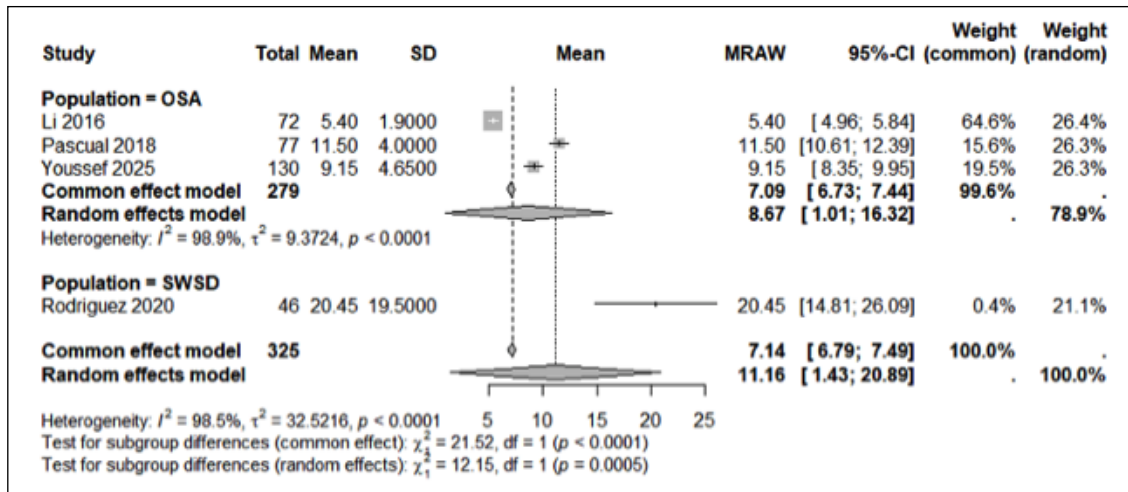


Figure 5.
Forest plot of
subgrouping of
testosterone
(nmol/L) level.

effect model and 8.67 nmol/L (95% CI: 1.01-16.32; $\tau^2 = 9.37$) under the random effects model. By contrast, the single SWSD study reported a much higher mean of 20.45 nmol/L (95% CI: 14.81-26.09). The test for subgroup differences was statistically significant under both models ($Q = 21.52$, $p < 0.0001$ for the common effect model; $Q = 12.15$, $p = 0.0005$ for the random effects model), confirming that testosterone levels differed substantially between OSA and SWSD patients.

Overall, these findings suggest that patients with OSA generally exhibit reduced testosterone levels, averaging around 7-9 nmol/L, whereas the available evidence from SWSD indicates markedly higher levels (~20 nmol/L) (Figure 5).

FSH (IU/L) level

A meta-analysis of FSH (IU/L) levels across four studies, involving a total of 325 patients, yielded differing results between the models used. Under the common-effect model, the pooled mean FSH level was 3.75 IU/L (95% CI: 3.56-3.93), suggesting a relatively precise and consistent estimate if all studies were assumed to measure the same underlying effect. However, the random-effects model produced a higher mean of 4.94 IU/L, with a wide confidence interval ranging from -0.23 to 10.10, indicating substantial uncertainty and inconsistency across studies. Measures of heterogeneity confirmed extreme variability, with an I^2 value of 99.6% and a highly significant Q statistic ($Q = 690.98$, $p < 0.0001$). The high τ^2 value (10.45) further highlighted considerable between-study variance (Figure 6).

Subgrouping of FSH (IU/L) levels

Subgroup analysis provided further insight. In the common-effect model, patients with OSA had a pooled mean of 3.83 IU/L (95% CI: 3.64-4.02), while those with SWSD had a lower mean of 2.24 IU/L (95% CI: 1.42-3.06). The test for subgroup differences was significant ($Q = 13.74$, $p = 0.0002$), suggesting that population type (OSA vs. SWSD) contributes to the variability. However, heterogeneity within the OSA subgroup remained very high ($I^2 = 99.7\%$).

Using the random-effects model, OSA patients had a higher pooled mean (5.83 IU/L, 95% CI: -2.40 to 14.05), but with considerable imprecision due to wide confidence intervals. SWSD patients had a mean of 2.24 IU/L (95% CI: 1.42-3.06), which was more stable. The test for subgroup differences under the random-effects model was not statistically significant ($Q = 3.36$, $p = 0.067$).

Overall, these findings suggest that FSH levels differ between OSA and SWSD populations, with OSA patients exhibiting higher values (Figure 7).

LH (IU/L) level

The meta-analysis of LH (IU/L) levels based on four studies with 325 participants demonstrated variability across populations and studies. When pooled, the common-effect model estimated a mean LH level of 3.21 IU/L (95% CI: 3.02-3.40), while the random-effects model produced a slightly higher mean of 3.39 IU/L but with a much wider confidence interval (95% CI: 1.85-4.93), reflecting substantial between-study variation. Measures of heterogeneity confirmed extreme inconsistency, with an I^2 value

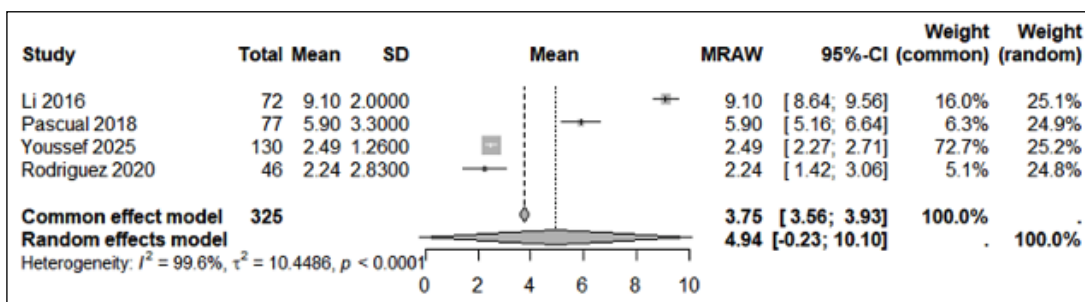


Figure 6.
Forest plot of FSH
(IU/L) level.

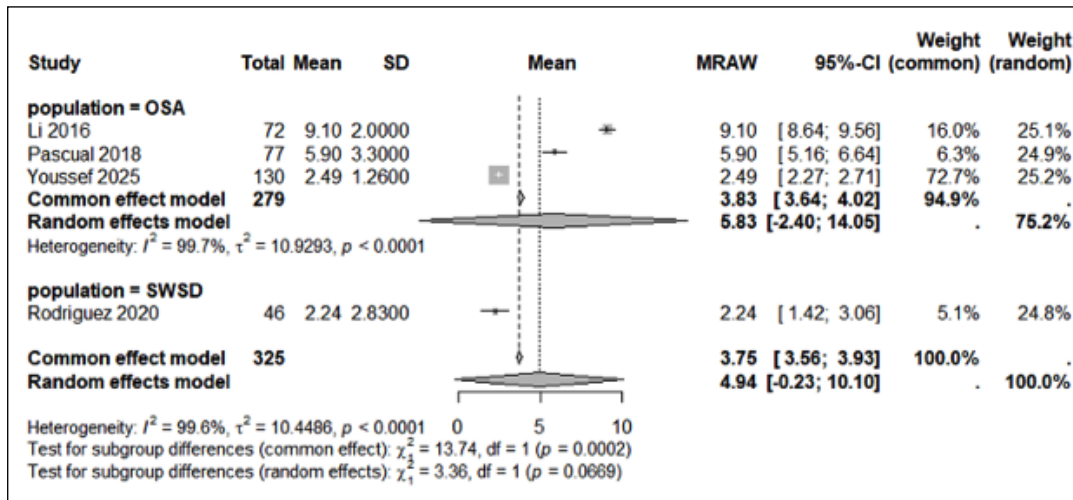


Figure 7.
Forest plot of
subgrouping of
FSH (IU/L) level.

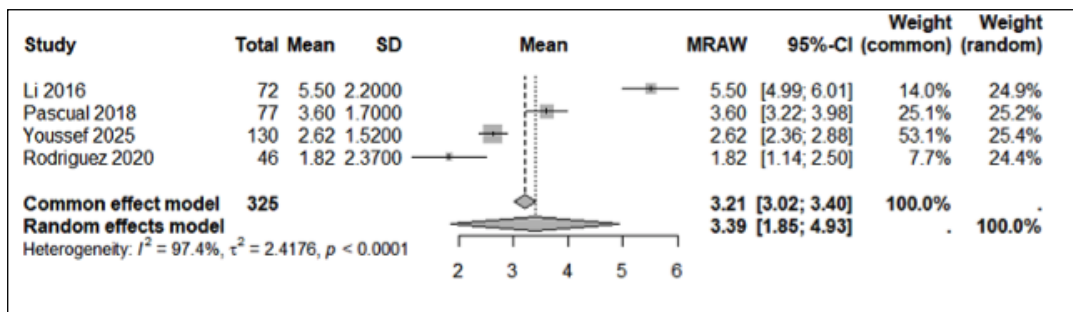


Figure 8.
Forest plot of LH
(IU/L) level.

of 97.4% and a highly significant Q statistic ($Q = 117.47$, $p < 0.0001$). Weighting also differed between models: under the common-effect model, the largest contribution came from Youssef (2025) due to its large sample size, whereas in the random-effects model, weights were distributed more evenly across studies (Figure 8).

Subgrouping of the LH (IU/L) level

Subgroup analyses further clarified these differences. For OSA patients (three studies), the pooled mean LH was 3.33 IU/L (95% CI: 3.13-3.52) under the common-effect model and 3.90 IU/L (95% CI: 0.26-7.53) under the random-effects model, with high heterogeneity remaining ($I^2 =$

98.0%). In contrast, the single SWSD study showed a mean of 1.82 IU/L (95% CI: 1.14-2.50), with no heterogeneity due to the single-study nature. Tests for subgroup differences were statistically significant in both models (common-effect $Q = 17.12$, $p < 0.0001$; random-effects $Q = 5.16$, $p = 0.023$), confirming that LH levels were significantly lower in the SWSD population compared to the OSA population.

Overall, these findings indicate that patients with sleep-related disorders have reduced LH levels, averaging approximately 3 IU/L; however, OSA patients generally exhibit higher LH levels compared with SWSD patients (Figure 9).

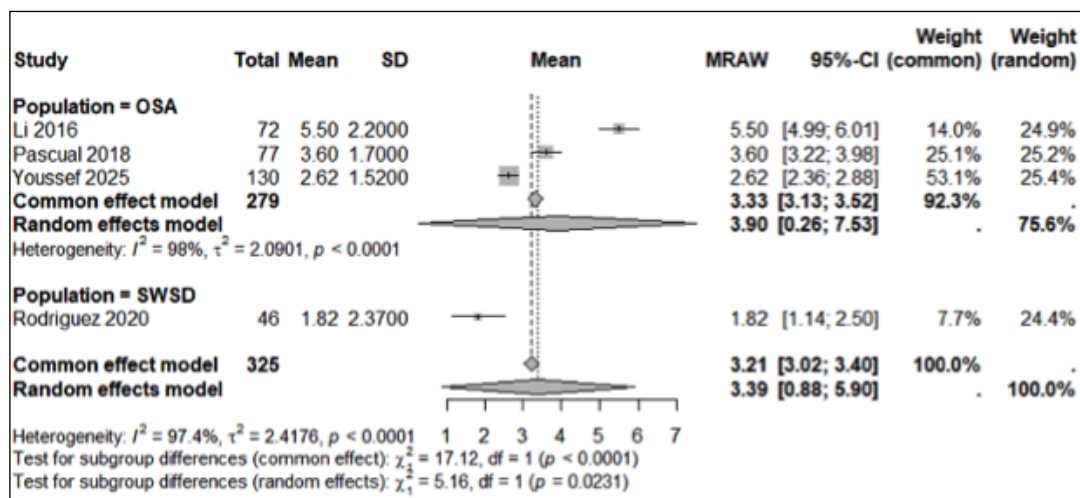


Figure 9.
Forest plot of
subgrouping of LH
(IU/L) level.

The IIEF scores according to severity of OSA

A meta-analysis of three studies (Irer, 2018; Youssef, 2025; Li, 2016) compared erectile function in patients with mild obstructive sleep apnea (OSA) to that of healthy controls. The pooled SMD was -0.65 (95% CI: -0.98 to -0.32, $p < 0.001$), indicating a moderate and statistically significant reduction in IIEF scores among patients with mild OSA. Heterogeneity across studies was $I^2 = 28\%$, $p = 0.24$, indicating low-to-moderate heterogeneity that was not statistically significant. These findings demonstrate that even mild OSA is associated with impaired erectile function compared with controls (Figure 10).

A meta-analysis of three studies with a total of 246 participants compared 114 with moderate OSA and 132 controls. The common-effect model demonstrated a sig-

A meta-analysis of three studies with 313 participants compared 181 patients with severe OSA and 132 controls. The common-effect model demonstrated a statistically significant and substantial reduction in erectile function among patients with severe OSA compared with controls (SMD = -2.05, 95% CI -2.38 to -1.72, $p < 0.0001$). However, when accounting for between-study variability, the random-effects model yielded a much larger negative effect (SMD = -5.29), but with very wide confidence intervals (95% CI: -19.16 to 8.59) and no statistical significance ($p = 0.243$). Measures of heterogeneity indicated extreme inconsistency across studies ($I^2 = 98.8\%$, $\tau^2 = 30.73$, $Q = 165.46$, $p < 0.0001$), suggesting that almost all variability in effect sizes was due to differences between studies rather than chance (Figure 12).

Figure 10.

Forest plot of the IIEF scores of patients with mild OSA.

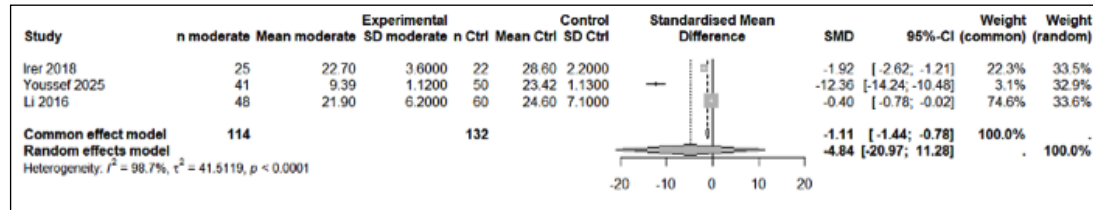
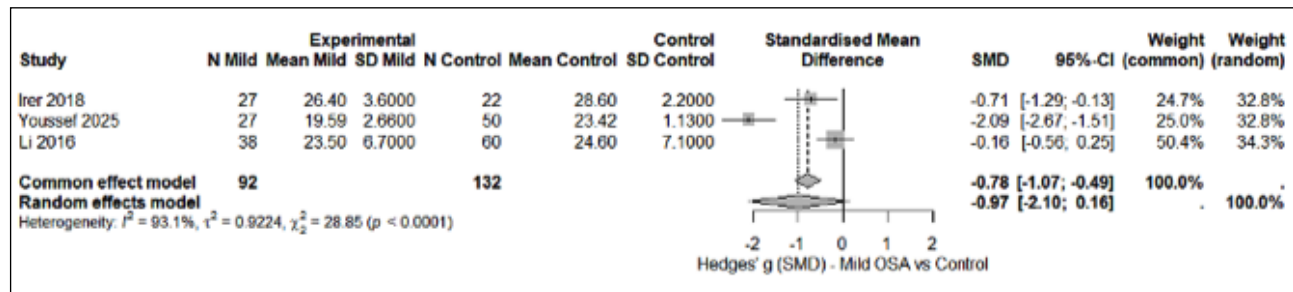


Figure 11.

Forest plot of the IIEF scores of patients with moderate OSA.

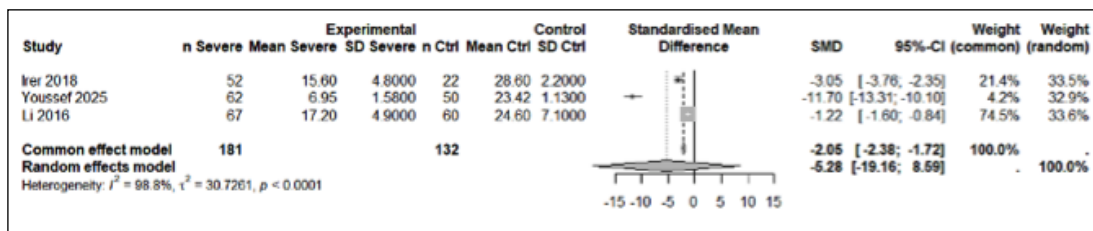


Figure 12.

Forest plot of the IIEF scores of patients with severe OSA.

nificant reduction in erectile function among moderate OSA patients compared with controls (SMD = -1.11, 95% CI -1.44 to -0.78, $p < 0.0001$). In contrast, the random-effects model, which accounts for variability across studies, estimated a much larger effect (SMD = -4.84) but with very wide confidence intervals (95% CI -20.97 to 11.28) and no statistical significance ($p = 0.326$). Measures of heterogeneity indicated extreme inconsistency among the included studies ($I^2 = 98.7\%$, $\tau^2 = 41.51$, $Q = 155.87$, $p < 0.0001$), suggesting that most of the variability was due to true differences across studies rather than chance (Figure 11).

DISCUSSION

This meta-analysis synthesized data from several studies to evaluate the impact of sleep-related disorders, particularly OSA and SWSD, on male reproductive hormones and erectile function. Overall, patients with these disorders exhibited significantly reduced IIEF scores, indicating impaired sexual performance. Subgroup analyses revealed that erectile function was notably lower in OSA compared to SWSD patients, suggesting a stronger association between OSA severity and sexual dysfunction. Regarding hormonal profiles, OSA patients demonstrated substantially lower testosterone levels, averaging around

7-9 nmol/L, while SWSD patients showed markedly higher concentrations. Similarly, FSH and LH levels varied between the two groups, with OSA patients tending to have higher values, reflecting potential compensatory activation of the gonadal axis.

When stratified by OSA severity, even mild cases were associated with significantly reduced IIEF scores compared to healthy controls, and the degree of erectile dysfunction increased with disease severity. However, the analyses were characterized by substantial heterogeneity, largely attributable to variations in study design, population characteristics, and measurement scales. Despite this variability, the overall findings consistently highlight a link between sleep disorders, particularly OSA, and impaired erectile and hormonal function, underscoring the importance of early recognition and management of these conditions.

A meta-analysis reported that individuals with sleep disorders had a markedly higher likelihood of experiencing *sexual dysfunction* (SD). The pooled estimates showed a relative risk of 1.97 (95% CI: 1.46-2.67, $p < .001$; $I^2 = 95.0\%$) in longitudinal studies and an odds ratio (OR) of 2.05 (95% CI: 1.76-2.39, $p < .001$; $I^2 = 91.4\%$) in cross-sectional studies. Furthermore, poor sleep quality increased the risk of SD by approximately 1.5 times (OR = 1.49; 95% CI: 1.31-1.71, $p < .001$; $I^2 = 73.4\%$), and short sleep duration was also significantly associated with SD (OR = 1.14; 95% CI: 1.06-1.22, $p < .001$; $I^2 = 0.0\%$). Overall, these results highlight that disturbances in both sleep quality and duration considerably heighten the risk of sexual dysfunction, underscoring the need for clinicians to assess and address sleep health in patients presenting with SD (24).

Furthermore, evidence suggests that disturbances in sleep architecture and intermittent hypoxia linked to COMISA can aggravate ED. The condition affects multiple physiological pathways, including hormonal imbalance, endothelial dysfunction, autonomic dysregulation, and heightened inflammation and oxidative stress. Together, these mechanisms compromise vascular integrity, lower testosterone production, disrupt neural pathways involved in erection, and ultimately intensify the severity of ED (6).

Another meta-analysis involving 1,275 participants from nine studies, five assessing ED and four evaluating *female sexual dysfunction* (FSD), found that OSA was significantly associated with a higher risk of both ED (pooled RR = 1.82, 95% CI: 1.12-2.97) and FSD (pooled RR = 2.00, 95% CI: 1.29-3.08). Sensitivity analyses confirmed the stability of these findings, and no evidence of publication bias was detected. Overall, the results from observational studies indicate that individuals with OSA are more likely to experience sexual dysfunction, although substantial heterogeneity was present. Further research is needed to better elucidate the mechanisms linking OSA to increased risk of sexual dysfunction (25).

Moreover, a meta-analysis aimed to clarify the relationship between sleep disorders and male reproductive health while exploring potential underlying mechanisms. The pooled results revealed that sleep disturbances were significantly associated with decreased total sperm count (MD = -27.91, 95% CI: -37.82 to -18.01, $p < 0.001$),

lower sperm concentration (MD = -5.16, 95% CI: -9.67 to -0.65, $p = 0.02$), reduced progressive motility (MD = -2.94, 95% CI: -5.28 to -0.59, $p = 0.01$), and decreased normal morphology (MD = -0.52, 95% CI: -0.80 to -0.24, $p < 0.001$). No significant associations were found between sleep disorders and semen volume or reproductive hormone levels. Further bioinformatics analysis indicated that key circadian rhythm-related genes (PER1, PER2, CRY2, NR1D1, and NPAS2) were downregulated in patients with non-obstructive azoospermia. Overall, the evidence suggests that sleep disorders negatively affect male reproductive function, likely through disruption of the circadian rhythm; however, due to the limited quality and quantity of studies, these findings warrant confirmation through larger, high-quality research (26). Findings also illustrate that OSA causes an increase in the FSH/LH levels and a decrease in the testosterone levels, which also contributes to the ED. This is aligned with observational cross-sectional study results that pointed to the significant impact of OSA on ED and hormonal profiles, particularly testosterone levels (15). A retrospective cohort study included men who had insomnia, sleep apnea, and circadian rhythm sleep disorders which were independently assessed to determine the association with ED and testosterone deficiency. It was found that testosterone deficiency was more likely to be found in men diagnosed with sleep apnea (27). Additionally, a prospective cohort study suggests that OSA in men is associated with dysfunction in the hypothalamic-pituitary-gonadal axis, characterized by high levels of serum LH (28). Overall, these findings highlight the potential reproductive consequences of untreated OSA and underscore the importance of early screening and intervention (29).

All other studies consistently align with our findings, demonstrating that sleep disorders adversely affect male sexual function through multiple physiological and psychological pathways. These effects are thought to result from disrupted hormonal regulation with reduced testosterone production. Collectively, the growing body of evidence highlights that sleep disturbances, regardless of their type or severity, play a significant role in diminishing overall male sexual performance and reproductive health.

These findings are further supported by recent evidence demonstrating a significant association between sleep quality impairment and male sexual dysfunction, reinforcing the association between sleep disturbances and reduced sexual performance (30).

Limitations

Despite its strengths, this analysis has several limitations that must be acknowledged. The high degree of heterogeneity among the included studies, stemming from variations in study design, diagnostic criteria, sample sizes, and measurement tools, limits the generalizability of the pooled estimates. Additionally, most included studies were observational in nature, which restricts the ability to infer causality between sleep disorders and sexual or hormonal dysfunction. Differences in population characteristics such as age, BMI, comorbidities, and medication may also have introduced residual confounding. Furthermore, the absence of sufficient SWDS studies reporting stan-

dardized IIEF outcomes limited subgroup-level meta-analysis for this condition, reduces the precision of subgroup comparisons and limits statistical power.

Recommendations for future research

Future studies should aim to address these limitations by employing standardized diagnostic criteria for both sleep disorders and sexual dysfunction, as well as uniform measurement scales for hormonal assays and erectile function. Large-scale, prospective cohort or interventional studies are needed to clarify causal relationships and to determine whether treating sleep disorders, such as with continuous positive airway pressure (CPAP) in OSA, leads to measurable improvements in erectile and hormonal outcomes. Additionally, future research should investigate molecular and physiological mechanisms, such as oxidative stress, endothelial dysfunction, and circadian rhythm disruption, to better understand the underlying biological pathways linking sleep disturbances to reproductive dysfunction.

CONCLUSIONS

The findings of this meta-analysis provide strong evidence that sleep-related disorders, particularly OSA, are significantly associated with impaired EF and altered reproductive hormone levels in men. The observed reduction in IIEF scores and testosterone levels underscores the clinical importance of recognizing and managing sleep disturbances as part of male sexual health assessment.

DECLARATIONS

Ethical approval and consent for participate: The Prospero registration number was given to the protocol of this systematic review [CRD420251154081].

Consent for publication: Not applicable.

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