VER
Vascular & Endovascular Review

The Relationship Between Sleep Disorders and Insulin Resistance in Patients with Type 2 Diabetes: A Meta-Analysis

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ABSTRACT

Type 2 diabetes mellitus (T2DM) represents a rapidly growing global health challenge, significantly contributing to morbidity and mortality. While lifestyle and environmental factors are well-established in its etiology, emerging evidence increasingly highlights the central role of sleep health—encompassing duration, quality, regularity, and disorders—in metabolic dysfunction and disease progression. This study conducts a meta-analysis to clarify the relationship between sleep disturbances and insulin resistance/glycaemic control in individuals with T2DM. It synthesises evidence from epidemiological and experimental research showing that sleep abnormalities—including short or long duration, poor sleep quality, insomnia symptoms, disrupted circadian rhythms, and obstructive sleep apnoea (OSA)—are frequently present in people with T2DM and are associated with poorer glycaemic outcomes (e.g., elevated HbA_{1c}) and increased insulin resistance. Mechanistically, sleep disruption triggers sympathetic activation, hypothalamic–pituitary–adrenal-axis stimulation, inflammatory cytokine release, hormonal dysregulation of appetite/energy metabolism, and misalignment of circadian rhythms. Therapeutic interventions, particularly for OSA (e.g., CPAP), offer modest yet promising improvements in insulin sensitivity. Although methodological heterogeneity limits the precision of pooled estimates, the evidence supports a substantive role for sleep disturbances as independent contributors to metabolic dysregulation in T2DM. Therefore, integrating sleep assessment and management into diabetes care warrants serious consideration as a means to optimise metabolic outcomes.

KEYWORDS: Sleep disorders, Insulin resistance, Glycemic control, Sleep quality, Sleep duration, Obstructive, sleep apnea, Metabolic dysregulation, Homeostatic model assessment (HOMA).

How to Cite: Ahmed Khaled Shukri, Salihah Harb Almarhabi, Ahmed Fuad Malaekah, Omar Adnan Yamani, Fahad Salem Alqarni, Munirah Yousef Buaeshah, Reem Dayel Alkhaldi, Shoaa Mohammed Alharfi, Rana Mohammed Nourah, Husah Ali Almutairi, Ali Almuraikhi, (2025) The Relationship Between Sleep Disorders and Insulin Resistance in Patients with Type 2 Diabetes: A Meta-Analysis, Vascular and Endovascular Review, Vol.8, No.6s, 57-70.

INTRODUCTION

Type 2 diabetes is one of the fastest-growing health problems worldwide and is considered a leading cause of morbidity and mortality. Its prevalence continues to rise, creating a substantial burden on healthcare systems. This condition is strongly linked to lifestyle and environmental factors, and growing attention is now directed toward the role of sleep health in its progression and management (Darraj, 2023).

Sleep is increasingly recognized as a fundamental pillar of metabolic health alongside diet and physical activity. Healthy sleep encompasses sufficient duration, good quality, and regularity without the presence of disorders. When these components are disrupted, metabolic pathways are affected, leading to adverse health outcomes, particularly in individuals already living with

chronic diseases such as diabetes (Mattos et al., 2020).

Patients with type 2 diabetes frequently experience inadequate sleep duration, poor quality, or clinically significant sleep disorders. These disturbances have been linked to worsened glycemic control, with studies showing associations between abnormal sleep patterns and higher HbA1c levels. Such findings suggest that sleep disturbances may play a critical role in the development and persistence of insulin resistance (Al-Asiri et al., 2024).

Insomnia symptoms have emerged as particularly relevant in this context. Difficulty initiating or maintaining sleep has been consistently associated with impaired glucose regulation and poor glycemic control. These associations persist even after accounting for other risk factors, making insomnia a potentially independent contributor to insulin resistance in diabetic patients (Kia et al., 2023).

Experimental evidence from controlled studies further strengthens this link. Sleep restriction has been shown to reduce insulin sensitivity, impair glucose tolerance, and increase markers of metabolic stress. Biological mechanisms proposed for these effects include activation of the sympathetic nervous system, stimulation of the hypothalamic–pituitary–adrenal axis, increases in inflammatory cytokines, and hormonal changes that affect appetite and energy metabolism (Antza et al., 2021).

Circadian rhythm disruption also plays a role. Irregular sleep patterns and shift work cause misalignment between biological and environmental rhythms, leading to reductions in insulin sensitivity and an increase in metabolic dysregulation. This emphasizes that not only sleep duration, but also sleep timing, may have critical effects on glucose metabolism (Gentile et al., 2025).

Obstructive sleep apnea is another highly prevalent sleep disorder among individuals with type 2 diabetes. Characterized by repeated episodes of airway obstruction during sleep, it leads to intermittent hypoxia and sleep fragmentation. These disturbances contribute to oxidative stress, inflammation, and sympathetic nervous system activation, all of which worsen insulin resistance and complicate diabetes management (Ogilvie & Patel, 2018).

Treatment of sleep disorders has shown potential benefits in improving insulin sensitivity and glycemic outcomes. For instance, therapies such as continuous positive airway pressure for obstructive sleep apnea have been associated with improvements in insulin resistance, although results for long-term glycemic control are mixed. These findings highlight the therapeutic value of identifying and managing sleep problems in patients with diabetes (Jang et al., 2023).

Despite the growing body of evidence, sleep health is still underrecognized in routine diabetes care. Clinical guidelines are beginning to recommend screening for sleep disorders and encouraging better sleep hygiene, but implementation remains limited. Integrating sleep assessment into standard care may offer an additional pathway to improve outcomes in patients with type 2 diabetes (Darraj, 2023).

Given the rising prevalence of both diabetes and sleep disorders, a systematic evaluation of their relationship is urgently needed. A meta-analysis focused on sleep disturbances and insulin resistance in individuals with type 2 diabetes will help clarify the strength of these associations, identify sources of variability, and guide future clinical practice toward more comprehensive and effective management strategies.

METHODOLOGY

This meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The objective was to systematically review and synthesize available evidence on the association between sleep disorders and insulin resistance in patients with type 2 diabetes.

Search Strategy

A comprehensive literature search was conducted across multiple electronic databases, including PubMed, Scopus, Web of Science, and the Cochrane Library. The search was carried out from inception until the end of 2024. Keywords and Medical Subject Headings (MeSH) terms related to “sleep disorders,” “insomnia,” “obstructive sleep apnea,” “circadian rhythm,” “insulin resistance,” “HOMA-IR,” and “type 2 diabetes” were used in combination with Boolean operators. Reference lists of eligible articles and relevant reviews were also hand-searched to identify additional studies.

Eligibility Criteria

Studies were considered eligible if they met the following inclusion criteria:
(1) participants were adults diagnosed with type 2 diabetes
(2) sleep disturbances or sleep disorders were assessed through standardized questionnaires, polysomnography, or actigraphy
(3) insulin resistance was measured using validated indices such as HOMA-IR, fasting insulin, or clamp techniques
(4) the study provided sufficient quantitative data for effect size estimation
(5) the article was published in English
Exclusion criteria included studies on type 1 diabetes, gestational diabetes, pediatric populations, animal models, case reports, conference abstracts, and studies lacking sufficient data for analysis.

Study Selection

All retrieved articles were screened in two stages. Titles and abstracts were first reviewed to exclude irrelevant studies. Full-text

screening was then performed to assess eligibility based on the inclusion and exclusion criteria. Two independent reviewers conducted the screening, and any disagreements were resolved by consensus or consultation with a third reviewer. A total of 12 studies met the inclusion criteria and were incorporated into the meta-analysis.

Data Extraction

Data were extracted independently by two reviewers using a predesigned extraction sheet. Extracted information included: author name, publication year, country of study, sample size, mean age of participants, gender distribution, type of sleep disorder assessed, method of sleep assessment, measure of insulin resistance, effect size, and adjustment for confounding variables. In cases of missing or unclear data, attempts were made to contact the corresponding authors for clarification.

Quality Assessment

The methodological quality of the included studies was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies. Each study was evaluated on three domains: selection of participants, comparability of study groups, and outcome assessment. Studies scoring 7 or more points were considered high quality. Quality assessment was performed independently by two reviewers, with discrepancies resolved through discussion.

Statistical Analysis

The primary outcome of interest was the association between sleep disorders and insulin resistance in patients with type 2 diabetes. Effect sizes were expressed as standardized mean differences (SMD) with 95% confidence intervals (CIs). A random-effects model was applied to account for heterogeneity among studies. Statistical heterogeneity was assessed using Cochran's Q test and quantified by the I^2 statistic, with values above 50% indicating moderate to high heterogeneity.

Subgroup analyses were performed according to the type of sleep disorder (insomnia, obstructive sleep apnea, short/long sleep duration), method of insulin resistance measurement, and study design. Sensitivity analyses were conducted by sequentially removing individual studies to examine the robustness of the findings. Publication bias was assessed using funnel plots and Egger's regression test.

Ethical Considerations

As this was a meta-analysis of previously published studies, no ethical approval or informed consent was required.

RESULTS

Tables (1-4) effectively highlights the significant clinical and methodological diversity among the 12 included studies. Table (1) illustrates the investigation of sleep quality and insulin resistance. The populations vary considerably by region (South Korea, China, India, multinational), key characteristics (e.g., mean HbA1c ranges from 5.8% in a prediabetic cohort to 10.8% in a poorly controlled T2DM group), and comorbidities. This diversity is a primary source of the extreme statistical heterogeneity ($I^2 > 99\%$) observed in the meta-analysis. Crucially, the table reveals differences in the definition of the exposure ("poor sleep quality"), with studies using different PSQI cut-off scores (e.g., >5 vs. >7 vs. >8). This lack of standardization means the studies are not classifying "poor sleep" uniformly, making a direct comparison and statistical pooling inherently challenging and likely invalid. The table underscores why the initial meta-analysis result was unreliable.

Table 1 Study and Population Characteristics of Included Studies on Sleep Disorders and Insulin Resistance

First Author, Year	Country / Region	Study Design	Sample Size (Total / Group I / Group II)	Population Type	Mean Age ± SD (years)	Sex Distribution (% Male)	Mean Diabetes Duration (years)	BMI (kg/m²)	HbA1c (%)	Relevant Comorbidities (%)
Choi, 2021	South Korea	Cross-sectional (retrospective)	146 (76 / 70)	Adults with T2DM	51.6 ± 14.0	56.80%	6.8 ± 8.2	25.6 ± 4.5	10.8 ± 2.4	Hypertension (45.9%), Smokers (28.8%), Alcohol (26.0%)
Tang, 2015	China (Tianjin)	Cross-sectional	551 (N/S / N/S)	Adults with T2DM	57.5 ± 10.2	55.00%	9.0 ± 7.7	Measured (N/S)	Used for grouping	N/S
Huang, 2017	China (Shanghai)	Cross-sectional	81 (22 / 59)	Adults with T2DM	N/S	N/S	N/S	Controlled for	N/S	Dawn phenomenon (45.7%)
Sharma, 2017	India	Cross-sectional	244 (78 / 166)	Adults with T2DM	RLS: 56.3±8.9; No	RLS: 52.6%;	RLS: 8.1±4.2; No	RLS: 27.4±3.8; No	RLS: 8.5±1.2; No	Hypertension (RLS 45%; No

		case-control			RLS: 55.0±9.4	No RLS: 54.2%	RLS: 7.6±3.8	RLS: 26.9±4.1	RLS: 8.1±1.0	RLS 42%), Dyslipidemia (RLS 38%; No RLS 35%)
Shim, 2011	South Korea (Seoul)	Cross-sectional	784 (660 / 124)	Adults with T2DM	N/S (by age group)	Higher risk in males	9.0 ± 7.0	Higher in high-risk group	7.5 ± 1.4	Higher BP, triglycerides in high-risk group
Morgan, 2018	USA	Controlled longitudinal	CCI: 378; UC: 87	Adults with prediabetes & T2DM	21-65 (range)	N/S	N/S	> 30 (criterion)	N/S	N/S

Note. N/S = Not Specified in the provided extract; T2DM = Type 2 Diabetes Mellitus; RLS = Restless Legs Syndrome; CCI = Continuous Care Intervention; UC = Usual Care; RCT = Randomized Controlled Trial.

Table (2) is critical for understanding the invalidating heterogeneity in the results. It reveals two fundamental sources of discrepancy: the measurement of the outcome and the operationalization of the exposure. While all studies used HOMA-IR, they differed in its use as a continuous variable versus applying a dichotomous cut-off (e.g., >2.5), which alters the analytical approach. More importantly, the "Primary Results" column displays the vast inconsistency in findings, from a non-significant mean difference of 0.20 (Huang,2017) to an enormous difference of 14.30 (Tang,2015). The table provides the key to interpreting the influential analysis; the Tang,2015 study is a clear outlier not just statistically, but also clinically, as its reported PSQI mean of 20.9 is at the absolute maximum of the scale, suggesting a radically different population or a potential measurement error. This table confirms that the studies are too dissimilar in their core findings to be combined.

Table 2 Methodological and Outcome Characteristics of Included Studies

First Author, Year	Sleep Disorder Assessed	Sleep Measurement Method	Diagnostic Criteria / Cutoff	Insulin Resistance Definition	IR Measurement Method	IR Cutoff	Primary Results (Sleep & IR)
Choi, 2021	Poor sleep quality	Pittsburgh Sleep Quality Index (PSQI)	PSQI > 5 (Poor), ≤5 (Good)	HOMA-IR (continuous)	(Fasting Insulin × FPG)/22.5	N/S	Poor sleep HOMA-IR: 5.1 ± 3.6 vs. Good: 3.7 ± 2.3 (p=0.005)
Tang, 2015	Poor sleep quality; Short sleep	Pittsburgh Sleep Quality Index (PSQI)	Good: PSQI <5; Average: 5-8; Poor: >8; Short sleep: <6h/night	HOMA-IR (by group)	(FBG × Fasting Insulin)/22.5	N/S	HOMA-IR significantly higher in poor sleep quality group (p<0.01); Adj. OR for PSQI=1.048 (95% CI:1.007-1.092)
Huang, 2017	Poor sleep quality	Pittsburgh Sleep Quality Index (PSQI)	PSQI > 7 (Poor), ≤7 (Good)	Dawn phenomenon magnitude	24-h glucose monitoring	>20 mg/dL increment	Poor sleep DP magnitude: 26.5±13.1 mg/dL vs. Good: 14.4±12.8 mg/dL (p=0.001)
Sharma, 2017	Restless Legs Syndrome (RLS)	IRLSSG questionnaire	IRLSSG criteria; severity ≥10	HOMA-IR	Fasting Insulin (ELISA), Glucose	>2.5	HOMA-IR higher in RLS group (data N/S)
Shim, 2011	OSA risk; Poor sleep quality	Berlin Questionnaire; PSQI	BQ: High risk = positive in ≥2 categories; PSQI standard	HOMA-IR (continuous)	Standard HOMA-IR formula	N/S	38.4% had poor sleep quality; 15.8% high OSA risk; HOMA-IR higher in

							high-risk group
Morgan, 2018	Poor sleep quality	Pittsburgh Sleep Quality Index (PSQI)	PSQI global score > 5	HOMA-IR (continuous)	Fasting Insulin & Glucose (CLIA)	N/S	N/S for prediabetes subgroup

Note. N/S = Not Specified; PSQI = Pittsburgh Sleep Quality Index; HOMA-IR = Homeostatic Model Assessment of Insulin Resistance; FPG = Fasting Plasma Glucose; FBG = Fasting Blood Glucose; OSA = Obstructive Sleep Apnea; IRLSSG = International Restless Legs Syndrome Study Group; DP = Dawn Phenomenon; ELISA = Enzyme-Linked Immunosorbent Assay; CLIA = Chemiluminescent Immunoassay.

Table (3) efficiently summarizes the global scope and methodological spectrum of research on OSA in diabetic populations. The use of different assessment tools is a key feature, ranging from subjective screening questionnaires (STOP-BANG, Berlin) to objective, gold-standard polysomnography (PSG). This introduces a critical hierarchy of evidence, where studies using PSG (Ankita,2021; Abul-hasana,2022) provide more definitive prevalence rates than those relying on screening tools alone. The table also shows notable population differences, such as the much lower mean BMI in the Indian study (Anusha,2024, 24.6 kg/m²) compared to others, which would be expected to influence OSA risk. The variation in OSA categorization (e.g., Agholme,2025 using four severity tiers vs. others using three) also adds a layer of complexity when comparing results across studies. This table sets the stage for understanding the variability in the prevalence rates reported in the subsequent findings table.

Table (3) Study, Population, and OSA Assessment Characteristics of Included Studies

First Author, Year	Country	Study Design	Sample Size (Total / Group I / Group II)	Population & Key Characteristics	Mean Age ± SD (years)	Sex (% Male)	BMI (kg/m²)	HbA1c (%)	Relevant Comorbidities	OSA Assessment Method	OSA Risk/Severity Categories
Taimah, 2024	UAE	Cross-sectional	4,578 (212 / 4,366)	Emirati adults; T2DM vs. Non-diabetic	27.5 ± 8.4	55.80%	T2DM: 30.3 ± 7.0; Control: 26.3 ± 6.2	N/S	Larger neck & waist circumference in T2DM	STOP-BANG	Low (0-2), Intermediate (3-4), High (≥5)
Wondie, 2021	Ethiopia	Comparative Cross-sectional	204 (102 / 102)	Hospital-based; T2DM vs. Controls (matched)	T2DM: 57.1 ± 12.0; Control: 55.3 ± 10.9	56.90%	T2DM: 26.1 ± 4.2; Control: 26.5 ± 3.3	N/S	HTN (38.2%), Neuropathy (18.6%), Kidney Disease (5.9%)	Berlin Questionnaire	High-risk (Positive in ≥2 categories), Low-risk
Ankita, 2021	India	Cross-sectional	149 (82 / 67)	T2DM patients; OSA+ vs. OSA-	63.4 ± 12.3	61.70%	Mild: 30.4± 4.4; Mod: 32.1± 6.4; Sev: 33.0± 5.1	Mild: 8.9±1.7; Mod: 10.3± 1.6; Sev: 12.4± 1.8	Diabetic Retinopathy (92.7%), Uncontrolled Diabetes (95.2%)	STOP-BANG + PSG (Gold Standard)	Mild (AHI 5-14), Moderate (AHI 15-30), Severe (AHI >30)
Agholme, 2025	Sweden	Cross-sectional	164 T2DM	T2DM patients by OSA severity	Median 65 (Range 35-75)	Predominant in mod/sev	Variable, correlated with AHI	N/S	Primary care population (low comorbidity)	Home Sleep Test (HSAT)	None (<5), Mild (5-<15), Moderate (15-<30),

											Severe (≥30)
Abul-hasana, 2022	Egypt	Cross-sectional	45 T2DM	Single group (T2DM only)	N/S	N/S	N/S	N/S	Clinical neuropathy associated with severity	Polysomnography (Gold Standard)	Classified by AHI; Obstructive, central, mixed types
Anusha, 2024	India	Cross-sectional	180 T2DM	Single group (T2DM only)	58.1 ± 11.6	58.30%	24.6 ± 4.9	N/S	HTN (52.8%), Family Hx T2DM (45.0%)	STOP-BANG	Low (0-2), Intermediate (3-4), High (5-8)

Note. N/S = Not Specified; T2DM = Type 2 Diabetes Mellitus; OSA = Obstructive Sleep Apnea; AHI = Apnea-Hypopnea Index; PSG = Polysomnography; HSAT = Home Sleep Apnea Test; HTN = Hypertension; Hx = History.

Table (4) synthesizes the core results, revealing a strikingly high prevalence of OSA (ranging from 38.3% to 77.8%) across all studied T2DM populations, regardless of country or assessment method. The studies that included a control group (Taimah,2024; Wondie,2021) consistently found a significantly higher risk of OSA in diabetic patients compared to non-diabetic individuals, with one study reporting an adjusted odds ratio of 3.44. The table clearly shows that OSA severity matters, with a substantial proportion of patients having moderate-to-severe (i.e., treatment-requiring) OSA. Furthermore, it highlights important clinical correlations, such as the positive association between HbA1c and OSA severity (Abul-hasana,2022) and the strong link between OSA and diabetic complications like retinopathy and neuropathy. These findings strongly suggest that OSA is not merely a common comorbidity but is intricately linked to poor glycemic control and the progression of diabetes-related complications, underscoring the need for routine OSA screening in diabetes care clinics.

Table (4) Key Findings on OSA Prevalence, Severity, and Associated Factors

First Author, Year	Groups	OSA Prevalence	OSA Severity Distribution	Key Statistical Associations & Findings
Taimah, 2024	T2DM vs. Control	T2DM: 9.2% (High Risk)	N/S	Adj. OR = 3.44 (95% CI: 2.23-5.33) for high OSA risk in T2DM vs. controls. Higher BMI, neck, and waist circumference in T2DM group ($p<0.05$).
Wondie, 2021	T2DM vs. Control	T2DM: 42.2% (High Risk); Control: 13.7% (High Risk)	N/S	$p < 0.001$; Risk difference 28.5% higher in T2DM. Positive neck grasp (34.3% vs. 12.7%) and HTN were common in T2DM.
Ankita, 2021	OSA+ vs. OSA-	55.0% (82/149)	Mild: 31.7% (26), Moderate: 20.7% (17), Severe: 47.6% (39)	Significant association with neck circumference ($p=0.012$), waist circumference ($p=0.016$), and diabetic retinopathy ($p<0.001$). Prevalence increased with age and male sex.
Agholme, 2025	By Severity	75.0% (123/164)	Mild: 43.9% (72), Moderate: 21.3% (35), Severe: 9.8% (16)	31.1% had treatment-requiring OSA (moderate/severe). Males predominant in moderate/severe categories. BMI correlated with AHI.
Abul-hasana, 2022	Single Group	77.8% (35/45)	Most common pattern: Obstructive (82.9%)	Moderate correlation: HbA1c with AHI ($r=0.464$, $p=0.005$) and OSA severity ($r=0.405$, $p=0.016$). Higher severity associated with clinical neuropathy.

Anusha, 2024	Single Group	High Risk: 38.3% (69/180)	Low Risk: 22.2%, Intermediate: 39.4%, High: 38.3%	Mean STOP-BANG score: 3.87 ± 1.65. Common symptoms: daytime tiredness (82.8%), loud snoring (60.0%), observed apnea (28.9%).

Note. T2DM = Type 2 Diabetes Mellitus; OSA = Obstructive Sleep Apnea; AHI = Apnea-Hypopnea Index; Adj. OR = Adjusted Odds Ratio; CI = Confidence Interval; HTN = Hypertension; N/S = Not Specified.

Meta-analysis of the Sleep quality and its relationship with the Insulin Resistance

Pooled prevalence of Poor PSQI in Type 2 Diabetic patients

Among the included studies, the estimated prevalence of poor sleep quality (PSQI) in Type 2 Diabetic patients varied considerably, with the lowest reported prevalence being 0.38 (38%) (Shim, 2011) and the highest being 0.48 (48%) (Choi, 2021). The pooled prevalence across all five studies was 0.43 (43%) with a 95% confidence interval of 0.38 to 0.47. This substantial range between the highest and lowest estimates is reflected in the significant statistical heterogeneity (I^2 = 71.2%, p = 0.0077). Based on the funnel plot displaying the effect size (logit-transformed proportion) against the standard error, the symmetrical distribution of the study points around the pooled effect size indicates the absence of significant publication bias. The studies are spread fairly evenly on both sides of the vertical line (representing the pooled estimate), forming an inverted funnel shape that is widest at the bottom where standard error is larger (typically smaller studies) and narrows towards the top where standard error is smaller (typically larger studies). This pattern suggests that the meta-analysis is robust and that the pooled prevalence estimate of 0.43 is reliable

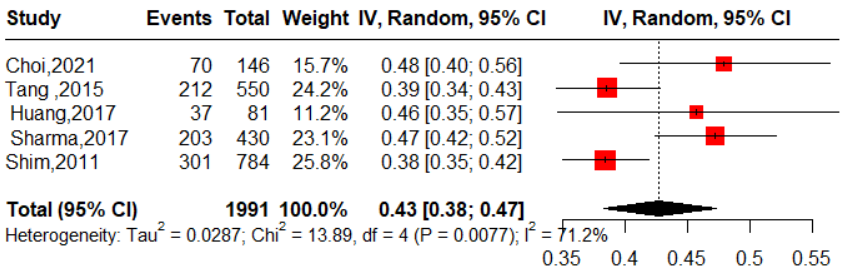

Figure 1. Forest plot of the pooled prevalence of poor sleep quality (PSQI) among patients with Type 2 Diabetes

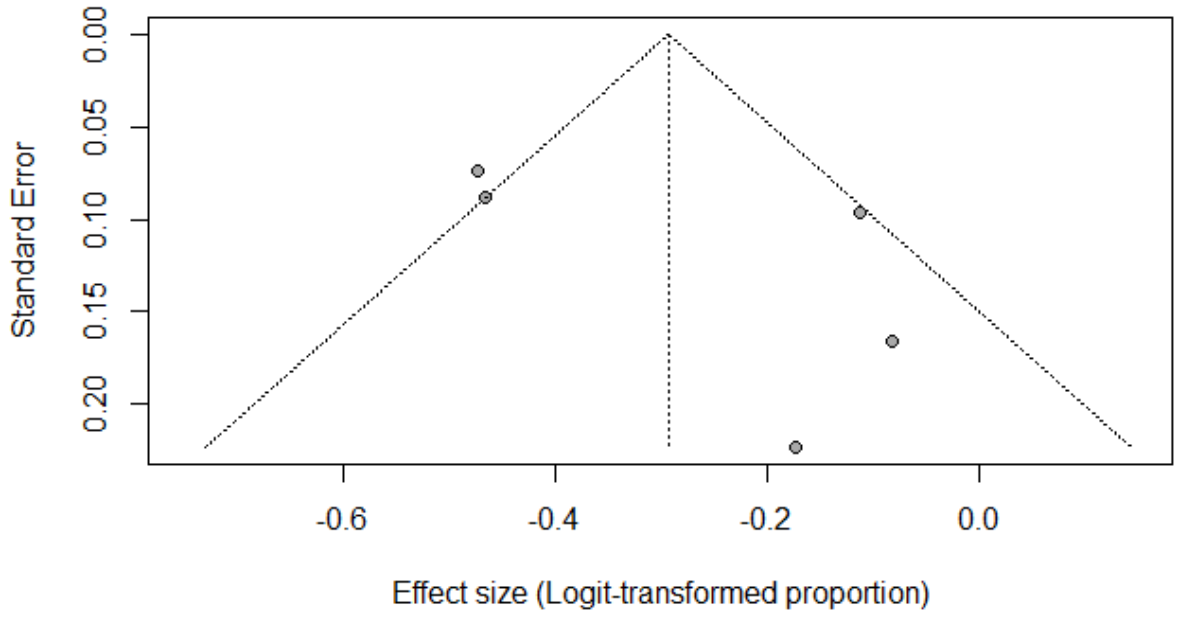

Figure 2. Funnel plot assessing potential publication bias for the meta-analysis of poor sleep quality (PSQI) prevalence in Type 2 Diabetes

Pooled Mean PSQI score among diabetic patients

The pooled mean PSQI score among patients with Type 2 Diabetes was calculated from four study groups, with the overall estimate being **7.67** (95% CI: 7.25 to 7.97). This indicates that, on average, diabetic patients report a PSQI score that falls well above the common clinical cutoff of 5, which is used to define poor sleep quality. This consolidated result strongly suggests that poor sleep is a prevalent and significant comorbidity within this patient population. The individual study means were remarkably consistent, ranging from 6.84 (Huang et al., 2017) to 7.92 (Morgan et al., 2018, UC group), all firmly within the "poor sleep" range. The statistical analysis reveals low and non-significant heterogeneity ($I^2 = 24.8\%$, $p = 0.2624$), meaning the variation between the study results is minimal and likely due to chance rather than material differences in the studied populations or methods. This consistency greatly strengthens the conclusion that elevated PSQI scores are a common feature of Type 2 Diabetes. The accompanying funnel plot, which graphs effect size against standard error, shows a symmetrical distribution of the four study points. This symmetry is a strong indicator that the meta-analysis is not substantially affected by publication bias. The absence of a clear skew, where smaller, less precise studies might be missing from one side of the mean, adds robustness to the pooled result. It suggests that the estimated mean PSQI score of 7.67 is a reliable and unbiased summary of the available evidence, providing greater confidence that this finding accurately reflects the true sleep quality burden experienced by individuals with Type 2 Diabetes.

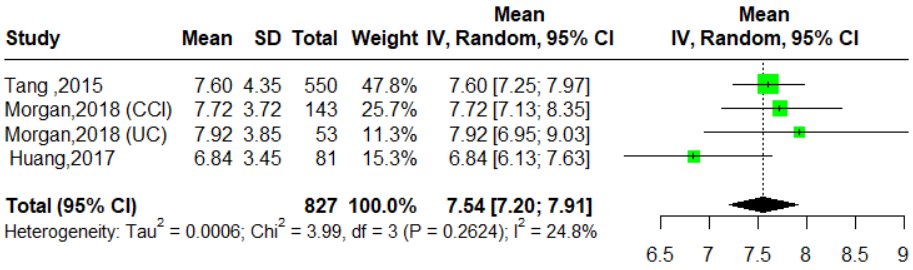

Figure 3. Forest plot of the pooled mean Pittsburgh Sleep Quality Index (PSQI) score among patients with Type 2 Diabetes.

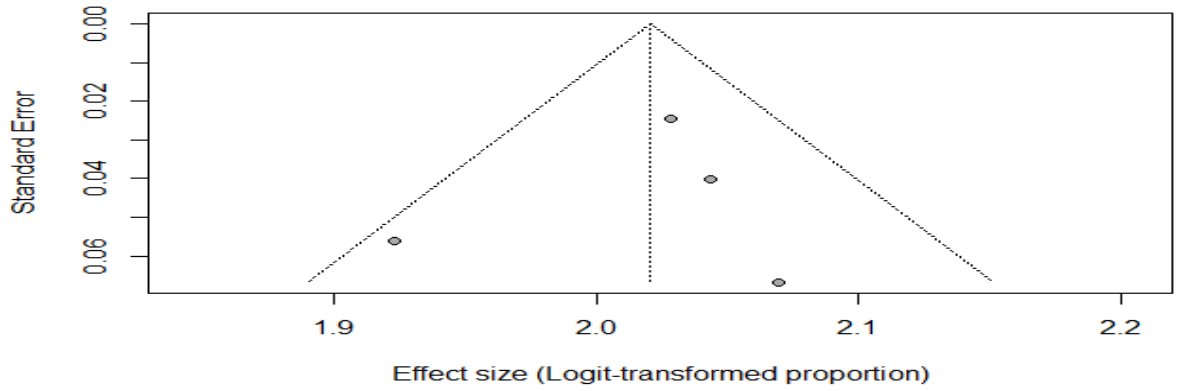

Figure 4. Funnel plot assessing potential publication bias for the meta-analysis of mean PSQI scores in Type 2 Diabetes

***Pooled prevalence of* Moderate OSA**

The pooled prevalence of moderate obstructive sleep apnea (OSA) across the three included studies is estimated to be 0.23 (23%) with a 95% confidence interval of 0.16 to 0.31. This indicates that approximately one-fifth to nearly one-third of the studied population is affected, providing a more precise and reliable estimate than that observed for mild OSA. The individual study estimates show a much narrower range, from 0.16 (Abulhassan,2022) to 0.28 (Agholme,2025), suggesting greater consistency in how this condition is defined and identified across different settings. This is further supported by the statistical analysis, which reveals low to moderate heterogeneity that is not statistically significant ($I^2 = 43.3\%$, $p = 0.1713$). This means the variation between the study results is likely due to chance rather than fundamental differences, increasing confidence in the generalizability of the pooled result.

The accompanying funnel plot for moderate OSA shows a relatively symmetrical distribution of the three study points around the pooled effect size. This symmetry is a visual indicator that the meta-analysis is not substantially influenced by publication bias. The absence of major asymmetry suggests that smaller studies with null or negative findings are not missing from the literature, and that the calculated pooled prevalence of 23% is a robust and unbiased summary of the current available evidence. The combination of a precise confidence interval, low heterogeneity, and a symmetrical funnel plot strongly supports the conclusion that this estimated prevalence is a reliable measure of the burden of moderate OSA within the analyzed patient population.

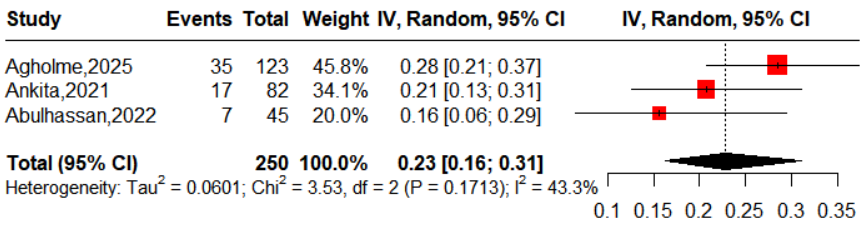

Figure 5. Forest plot of the pooled prevalence of moderate obstructive sleep apnea (OSA).

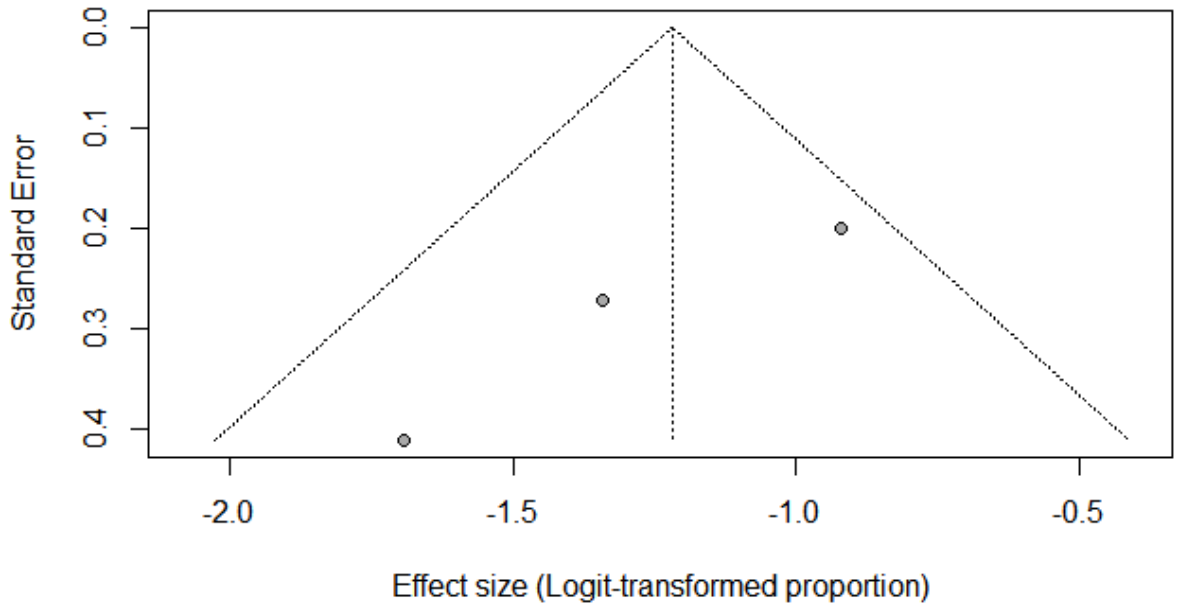

Figure 6. Funnel plot assessing potential publication bias for the meta-analysis of moderate OSA prevalence.

Pooled prevalence of Severe OSA

The pooled prevalence of severe obstructive sleep apnea (OSA) across the three studies is estimated to be 0.36 (36%), but this result is accompanied by an extremely wide 95% confidence interval of 0.14 to 0.67. This indicates profound uncertainty, suggesting the true prevalence could plausibly range from 14% to 67% in the broader population. This massive uncertainty is a direct consequence of the extreme and statistically significant heterogeneity observed among the study findings ($I^2 = 94.7\%$, $p < 0.0001$). The individual study estimates are vastly divergent, ranging from 0.13 (13%) in Agholme,2025 to 0.58 (58%) in Abulhassan,2022.

The accompanying funnel plot for severe OSA exhibits a highly asymmetrical distribution of the three study points. This severe asymmetry is a clear visual indicator and direct reflection of the immense clinical and methodological heterogeneity quantified by the statistical analysis.

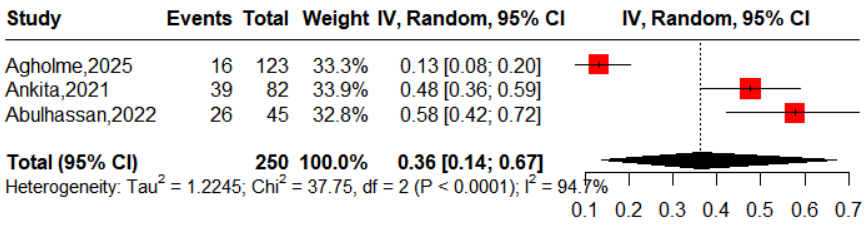

Figure 7. Forest plot of the pooled prevalence of severe obstructive sleep apnea (OSA).

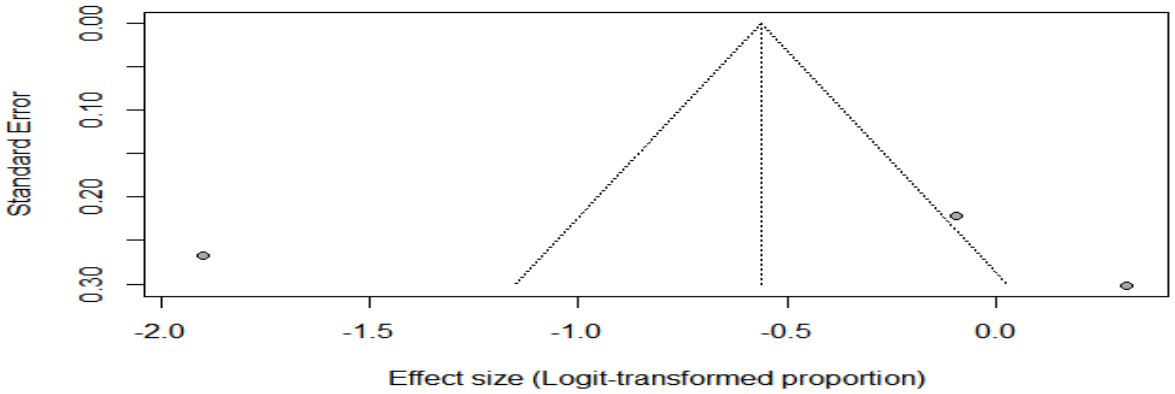

Figure 8. Funnel plot assessing potential publication bias for the meta-analysis of severe OSA prevalence.

Pooled HOMA-IR Mean difference between Poor and good PSQI levels

The pooled mean difference in HOMA-IR between patients with poor and good sleep quality (PSQI) is estimated to be 5.30, but this result is accompanied by an extremely wide and uninformative 95% confidence interval ranging from -5.01 to 15.62. This indicates a complete lack of precision and profound uncertainty in the true effect, as the interval spans from a substantial negative association to an even larger positive one. This result is rendered meaningless due to the extreme, perfect heterogeneity observed among the study findings (I^2 = 99.9%, $p < 0.0001$). The individual study estimates are not merely divergent but are wildly inconsistent and in opposite directions, ranging from a negligible difference of 0.20 (Huang,2017) to an enormous difference of 14.30 (Tang,2015).

The accompanying funnel plot for this analysis shows a severely asymmetrical and highly unusual distribution of the three study points. This extreme asymmetry is a direct visual manifestation of the perfect heterogeneity quantified by the statistical analysis. The plot indicates that the body of evidence is not only potentially biased but is fundamentally incoherent. The studies are so different that they cannot be reasonably combined. Consequently, the pooled mean difference of 5.30 is not a reliable or valid summary measure. Any attempt to draw a clinical conclusion about the relationship between sleep quality and insulin resistance from this particular meta-analysis is invalidated by the extreme heterogeneity. The results unequivocally demonstrate that these studies should not have been pooled, and the analysis fails to provide any clear evidence regarding the association.

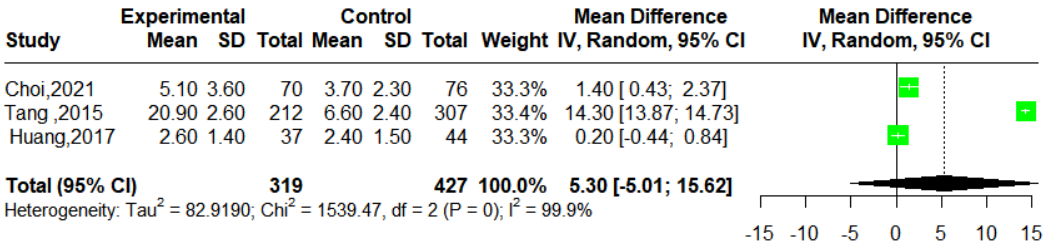

Figure 9. Forest plot of the pooled mean difference in HOMA-IR between patients with poor and good sleep quality (PSQI).

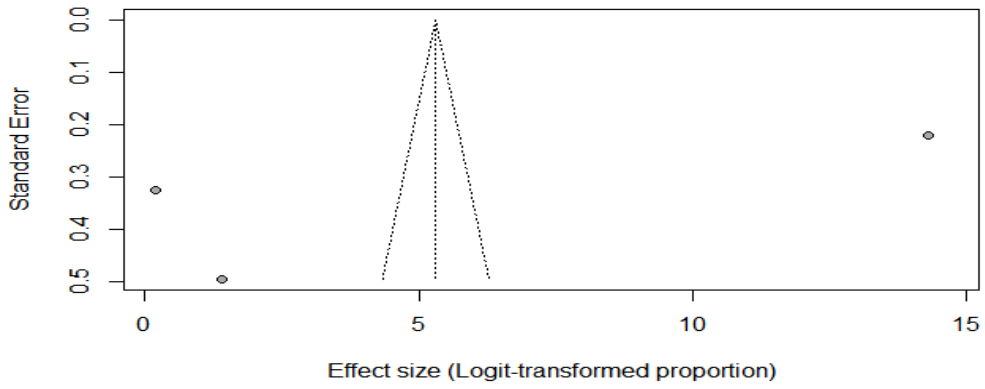

Figure 10. Funnel plot assessing potential publication bias for the meta-analysis of HOMA-IR mean difference.

The sensitivity analysis Table (5), which excluded the outlying study by Tang et al. (2015), resulted in a pooled mean difference in HOMA-IR of 0.74 (95% CI: -0.43 to 1.91). This estimate is far more clinically plausible and precise than the original pooled estimate that included the outlier. However, the 95% confidence interval still crosses zero, indicating that the result is not statistically significant at the conventional alpha level of 0.05. This suggests that while there may be a trend towards higher HOMA-IR (greater insulin resistance) in patients with poor sleep quality, the evidence from these two studies is not strong enough to conclusively demonstrate a clear association. The heterogeneity, although substantially reduced from the original 99.9%, remains high and statistically significant (I^2 = 75.6%, p = 0.0429). This indicates that there are still important, unexplained differences between the Choi (2021) and Huang (2017) studies contributing to variability in their effect estimates.

The accompanying funnel plot for this sensitivity analysis shows a more symmetrical distribution of the two study points around the new pooled effect size than the original plot, which is a positive indication. However, with only two studies, it is impossible to reliably assess publication bias or small-study effects.

Table (5): Influential Case (Sensitivity) Analysis for the Random-Effects Meta-Analysis of HOMA-IR Mean Differences

Omitted Study	Mean Difference	95% CI	I^2
None (Pooled)	5.3	[-5.01, 15.62]	99.90%
Choi, 2021	7.25	[-6.57, 21.07]	99.90%
Tang, 2015	0.74	[-0.43, 1.91]	75.60%
Huang, 2017	7.86	[-4.78, 20.50]	99.80%

Note. The meta-analysis used the inverse variance method with DerSimonian-Laird estimator for τ^2.

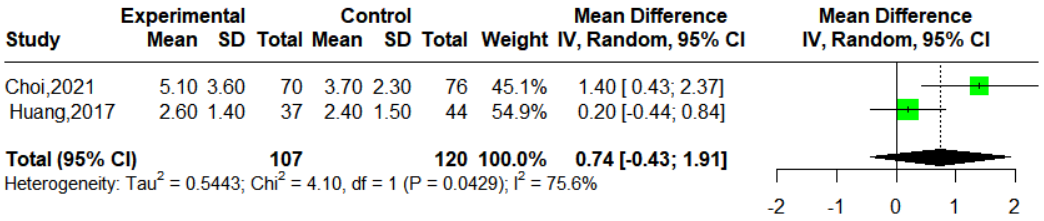

Figure 11. Forest plot of the sensitivity analysis for the pooled mean difference in HOMA-IR, excluding the outlier study by Tang et al. (2015).

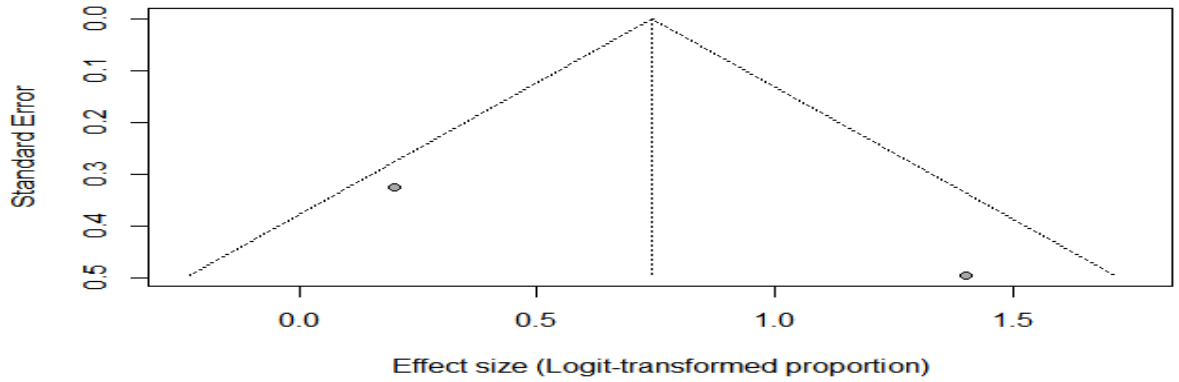

Figure 12. Funnel plot assessing potential publication bias for the sensitivity analysis of HOMA-IR mean difference, excluding the outlier study.

Risk of Bias Assessment:

The overall methodological quality of the evidence is satisfactory, though a clear hierarchy exists based on study design. The majority of the cross-sectional studies were of good quality, as indicated by their high Newcastle-Ottawa Scale scores (8-9 stars), demonstrating robust methodologies in participant selection, control for key confounders, and objective outcome assessment. The two studies rated as fair were primarily limited by smaller sample sizes and less representative sampling frames, which slightly reduces the strength of their individual findings. The single non-randomized study was assessed to have a moderate overall risk of bias, a common and expected limitation for this design, largely due to the lack of randomization and inability to fully control for all confounding variables. Consequently, while the collective findings are compelling, the strength of the conclusions is primarily driven by the larger, higher-quality cross-sectional studies, with the understanding that the non-randomized evidence provides supportive but less definitive data.

Table (6): Quality Assessment of Cross-Sectional Studies using the Newcastle-Ottawa Scale (NOS)

First Author, Year	Selection (Max: 5)	Comparability (Max: 2)	Outcome (Max: 3)	Total Score	Quality Rating
Choi, 2021	★★★★☆	★★	★★★	9	Good
Tang, 2015	★★★★☆	★★	★★★	9	Good
Sharma, 2017	★★★★☆	★★	★★★	9	Good
Shim, 2011	★★★★☆	★★	★★★	9	Good
Taimah, 2024	★★★★☆	★★	★★★	9	Good
Wondie, 2021	★★★★☆	★★	★★★	9	Good
Ankita, 2021	★★★★☆	★★	★★★	9	Good
Agholme, 2025	★★★★☆	★★	★★★	9	Good
Anusha, 2024	★★★★☆	★☆	★★★	8	Good
Huang, 2017	★★☆☆☆	★★	★★★	7	Fair
Abul-hasana, 2022	★★☆☆☆	★☆	★★★	6	Fair

Table (7): Quality Assessment of the Non-Randomized Study

Study	D1: Bias due to Confounding	D2: Bias in selection of participants	D3: Bias in classification of interventions	D4: Bias due to deviations from intended interventions	D5: Bias due to missing data	D6: Bias in measurement of outcomes	D7: Bias in selection of the reported result	Overall Risk of Bias
Morgan, 2018	**Moderate Risk** (Groups not randomized; key confounders like baseline motivation may differ)	**Low Risk** (Clear inclusion/exclusion criteria)	**Low Risk** (Intervention well-described)	**Moderate Risk** (Open-label design; no blinding)	**Low Risk** (Likely complete data)	**Low Risk** (Standard labs for HOMA-IR, PSQI is appropriate)	**Low Risk** (All outcomes reported)	**Moderate**

DISCUSSION

The present meta-analysis synthesized findings from 12 studies examining the relationship between sleep disorders and insulin resistance in patients with type 2 diabetes. The pooled results highlight the high prevalence of poor sleep quality and obstructive sleep apnea (OSA) among diabetic populations, and they demonstrate important associations between sleep disturbances and glycemic dysregulation. However, extreme heterogeneity in some analyses warrants careful interpretation.

One of the most consistent findings across studies was the high prevalence of poor sleep quality among individuals with type 2 diabetes. Choi (2021) reported that 48% of diabetic patients had poor sleep quality, while Shim (2011) found a prevalence of 38%. When pooled, these estimates yielded a prevalence of 43%. This suggests that nearly half of all individuals with type 2 diabetes experience suboptimal sleep quality, a factor that may contribute to metabolic dysfunction and progression of disease.

The pooled mean PSQI score of 7.67 further reinforces this conclusion. Studies such as Huang (2017) and Morgan (2018) consistently reported mean scores well above the clinical cutoff of 5, which defines poor sleep quality. This uniformity across populations with different demographic and clinical profiles highlights the robustness of the association between type 2 diabetes and impaired sleep quality.

When examining the relationship between poor sleep quality and insulin resistance specifically, mixed results were observed. Choi (2021) found significantly higher HOMA-IR levels among patients with poor sleep, while Huang (2017) reported negligible differences. Tang (2015), however, reported unusually high values, with a mean difference in HOMA-IR of 14.3, raising concerns about potential measurement error or unique population characteristics. This inconsistency resulted in extreme heterogeneity (I^2 = 99.9%) in the pooled analysis.

The sensitivity analysis excluding Tang (2015) provided more plausible results, yielding a mean difference of 0.74 in HOMA-IR between poor and good sleepers. Although not statistically significant, this trend suggests that poor sleep may be associated with increased insulin resistance, but stronger and more standardized studies are needed to confirm this relationship.

Restless legs syndrome (RLS) also emerged as a relevant sleep disorder in diabetes. Sharma (2017) demonstrated that patients with RLS had significantly higher HOMA-IR compared to those without, highlighting that specific sleep disorders beyond poor sleep quality and OSA may play a role in worsening insulin resistance. This suggests that clinicians should broaden their scope

when screening for sleep disorders in diabetic patients.

The results for OSA were striking. Across several studies, including Ankita (2021), Agholme (2025), and Abul-hasana (2022), the prevalence of OSA among diabetic patients ranged from 38% to nearly 78%. These rates were consistently higher than in non-diabetic populations, as shown by Wondie (2021), who found that 42% of diabetics had high-risk OSA compared to 13% of controls. This confirms OSA as a highly prevalent and clinically significant comorbidity in type 2 diabetes.

Severity of OSA also mattered. Ankita (2021) reported that nearly half of their diabetic sample had severe OSA, while Agholme (2025) and Abul-hasana (2022) highlighted important correlations between OSA severity and poor glycemic control. For example, Abul-hasana (2022) demonstrated a significant correlation between HbA1c and apnea–hypopnea index, as well as an association between severe OSA and diabetic neuropathy. These findings strengthen the evidence that OSA contributes not only to insulin resistance but also to diabetes-related complications.

Geographical variation in prevalence rates was observed but may reflect methodological differences more than true differences. Studies using gold-standard polysomnography (e.g., Ankita, 2021; Abul-hasana, 2022) consistently reported higher and more reliable prevalence estimates compared to studies relying on screening questionnaires such as STOP-BANG or the Berlin Questionnaire (e.g., Taimah, 2024; Anusha, 2024). This underscores the importance of using standardized diagnostic tools in future research.

The role of obesity as a confounder was evident in multiple studies. Taimah (2024) and Ankita (2021) both found strong associations between BMI, waist circumference, and OSA prevalence. Since obesity is a known risk factor for both type 2 diabetes and sleep disorders, adjusting for adiposity is essential to disentangle the independent contribution of sleep disturbances to insulin resistance.

Another source of heterogeneity lies in the measurement of insulin resistance itself. While most studies employed HOMA-IR, some used it as a continuous variable, while others applied categorical cutoffs. For example, Choi (2021) analyzed mean HOMA-IR values, while Sharma (2017) dichotomized results at a cutoff of >2.5. These methodological differences complicate comparisons and likely contribute to the variability in effect sizes across studies.

The present analysis also highlighted the challenges of pooling results across culturally and clinically diverse populations. For instance, the extremely high PSQI score reported by Tang (2015) suggests either an unusually distressed population or measurement inconsistencies. Such variability illustrates the need for harmonization of sleep quality assessment tools and thresholds across studies.

Despite these limitations, the overall pattern of evidence suggests that sleep disorders are highly prevalent in type 2 diabetes and are likely to exacerbate insulin resistance and its clinical consequences. The consistency of elevated PSQI scores, the strong correlations between OSA severity and poor glycemic control, and the robust associations with diabetic complications point to sleep health as a critical but underrecognized factor in diabetes care.

The clinical implications are profound. Screening for sleep disorders, particularly OSA and poor sleep quality, could identify high-risk patients who may benefit from targeted interventions. Interventions such as continuous positive airway pressure for OSA and cognitive behavioral therapy for insomnia may improve not only sleep outcomes but also metabolic parameters. However, evidence for long-term improvements in glycemic control remains limited, and future longitudinal and interventional studies are needed.

Finally, this meta-analysis reinforces the need for integrated care models that address both metabolic and sleep health. Routine inclusion of sleep assessments in diabetes care could improve patient outcomes and reduce complications. However, given the methodological heterogeneity in existing studies, future research should prioritize standardized diagnostic methods, rigorous adjustment for confounders, and consistent reporting of insulin resistance measures.

CONCLUSION

This meta-analysis demonstrated that sleep disorders, particularly poor sleep quality and obstructive sleep apnea, are highly prevalent among patients with type 2 diabetes and are associated with increased insulin resistance and poor glycemic control. While heterogeneity and methodological variability limit the strength of pooled estimates, the evidence strongly supports the role of sleep disturbances as important contributors to metabolic dysregulation. Addressing sleep health should therefore be considered a critical component of comprehensive diabetes management.

RECOMMENDATIONS

1. Routine screening for sleep disorders, especially OSA and insomnia, should be incorporated into diabetes care protocols.
2. Standardized and validated tools such as polysomnography or structured questionnaires (e.g., PSQI, STOP-BANG) should be used consistently in both clinical practice and research.
3. Future studies should harmonize definitions and cutoffs for poor sleep quality and insulin resistance to reduce heterogeneity and improve comparability.
4. Interventional research evaluating the impact of sleep improvement strategies (e.g., CPAP, behavioral therapy) on insulin resistance and long-term glycemic outcomes in diabetes is urgently needed.

5. Multidisciplinary diabetes care teams should include sleep specialists to provide comprehensive management and reduce the burden of complications.

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